Personalized medicine in early stage lung cancer

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Goals

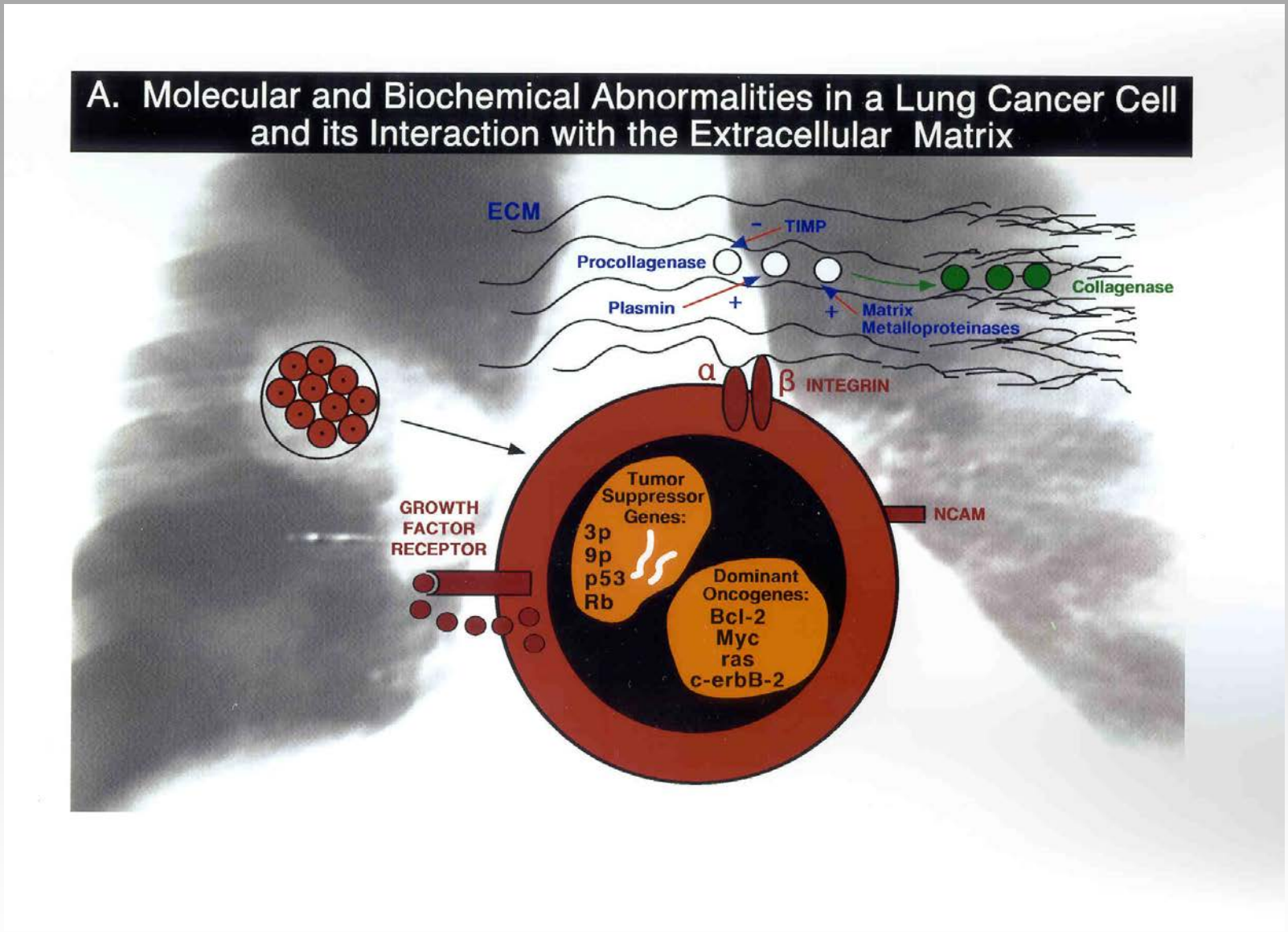
- Describe Molecular Advances in Lung Cancer
- Describe Molecularly Targeted Therapies—EGFR and ALK
- Detail the Early Stage Disease and Potential for Novel Therapies

NSCLC: Treatment and Outcome by Stage

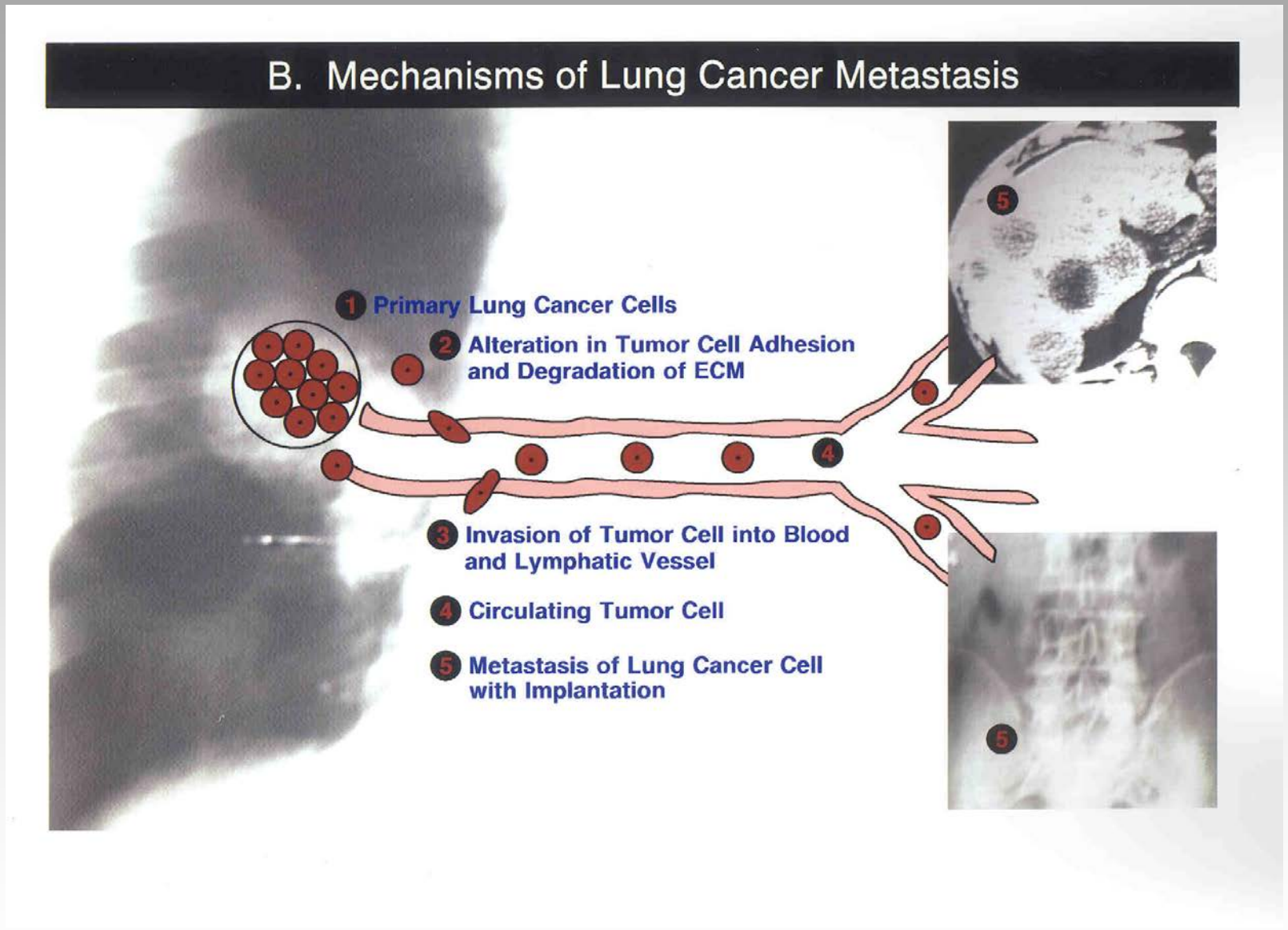
Pathologic Stage	Treatment	5-Year Survival, %*
I	Surgery/Chemo	60-70
II	Surgery/Chemo	30-50
IIIA	Surgery/ Multimodality Regimen	10-30
IIIB	Chemotherapy/ Radiation	5
IV	Chemotherapy	<1

***Overall 5-year survival is 18%.**

1. Mountain CF. *Semin Surg Oncol*. 2000;18:106-115.
2. National Cancer Institute. *SEER Cancer Statistics Review 1973-1999*.



Salgia & Skarin, 1996



Salgia & Skarin, 1996

Targeted Therapy in Oncology

- **Goals**
  - **Identify anti-tumor agents that target tumor-specific molecules, thus sparing normal cells**
    - **Increased specificity leads to decreased toxicity**
  - **Identify ideal drug target**
    - **Drives tumor growth**
    - **Turns on key mechanisms of cancer progression**
    - **Reversible by inhibition**
    - **Dispensable in normal cells**
    - **Target is measurable in tumor tissue used for diagnosis**

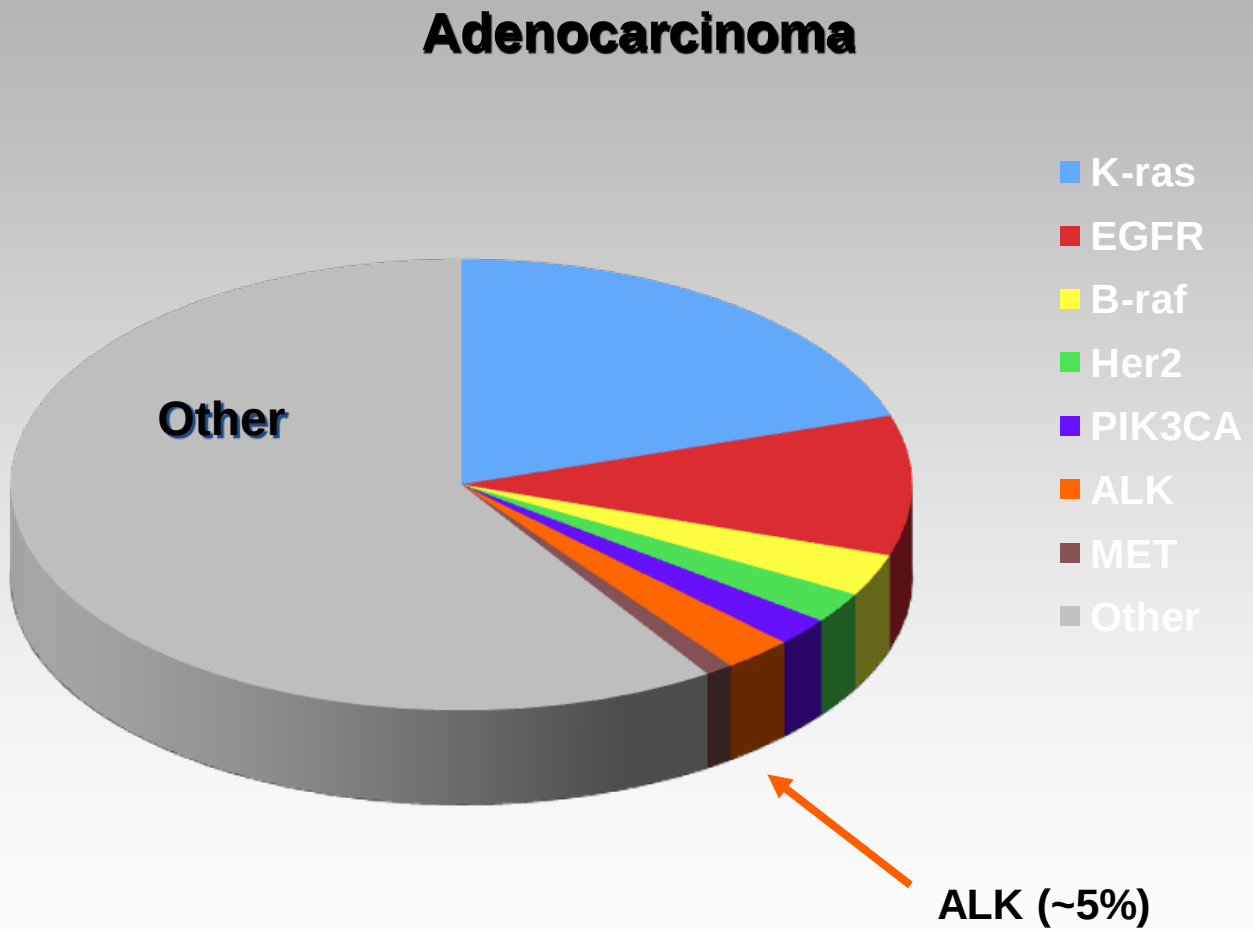
Table 1. Molecular Abnormalities in Lung Cancer

Molecular Abnormality	SCLC (%)	NSCLC (%)	Agent(s) Targeting Aberrant Pathway
Incidence	25	75	
Ras mutation	< 1	30	Farnesyltransferase inhibitors, antisense oligonucleotides, *raf* inhibitors, MEK inhibitors, 17-AAG
Myc amplification	30	10	?
EGFR expression	NR	40-80	EGFR tyrosine kinase inhibitors; EGFR antibodies
c-*erb*B-2 overexpression	10	30	Trastuzumab, EGFRTK inhibitors, 17AAG
c-kit/SCFR coexpression	70	15	STI-157
Bcl-2 expression	95	35	PS-341
p53 mutation	75-100	50	PS-341, flavopiridol
RB deletion (protein)	90	20	CCI-779, flavopiridol
p16 inactivation	< 1	70	CCI-779
COX-2 expression	NR	70	COX-2 inhibitors
3p deletion	90	50	?
VEGF expression	> 100-fold variation		Monoclonal antibodies, RTK inhibitors, FTI
Matrix metalloproteinase (gelatinase)	50	65	Matrix metalloproteinase inhibitors
Neuropeptides	90	NR	Antibodies

Abbreviations: EGFR, epidermal growth factor receptors; SCFR, stem-cell factor receptor; RB, retinoblastoma gene; VEGF, vascular endothelial growth factor; MEK, mitogen-activated protein kinase (MAPK) kinase; RTK, receptor tyrosine kinase; FTI, farnesyl transferase inhibitors; COX, cyclo-oxygenase.

Dy & Adjei, JCO 2002
Salgia & Skarin, JCO 1999

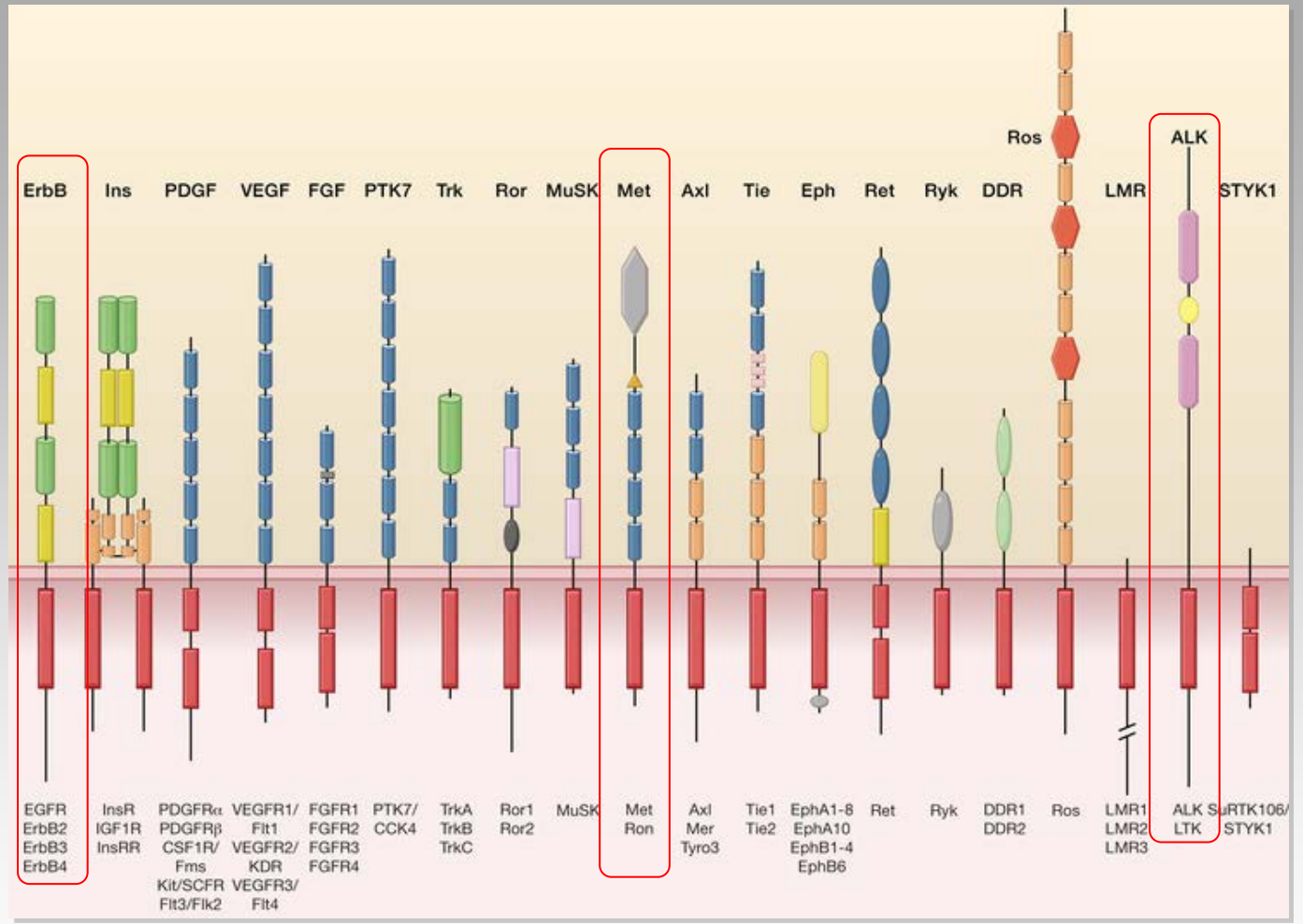
Potential Oncogenic "Drivers" in Non-small Cell Lung Cancer (NSCLC)

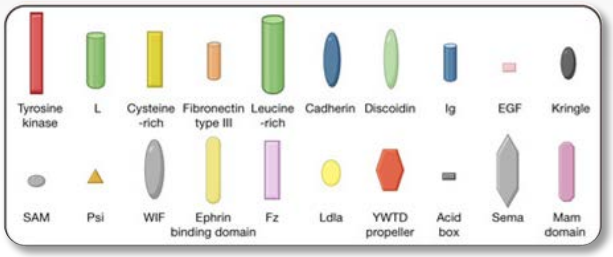


ALK = anaplastic lymphoma kinase; EGFR = epidermal growth factor receptor; Her2 = human epidermal growth factor receptor 2; PIK3CA = phosphoinositide-3-kinase, catalytic, alpha polypeptide

Massachusetts General Hospital, data on file.
[AT Shaw, personal communication]

Human Receptor Tyrosine Kinases





Lemmon & Schlessinger. Cell 141:1117-34 (2010)

EGFR

Diverse Antitumor Effects of EGFR Inhibition

Proliferation

Invasion

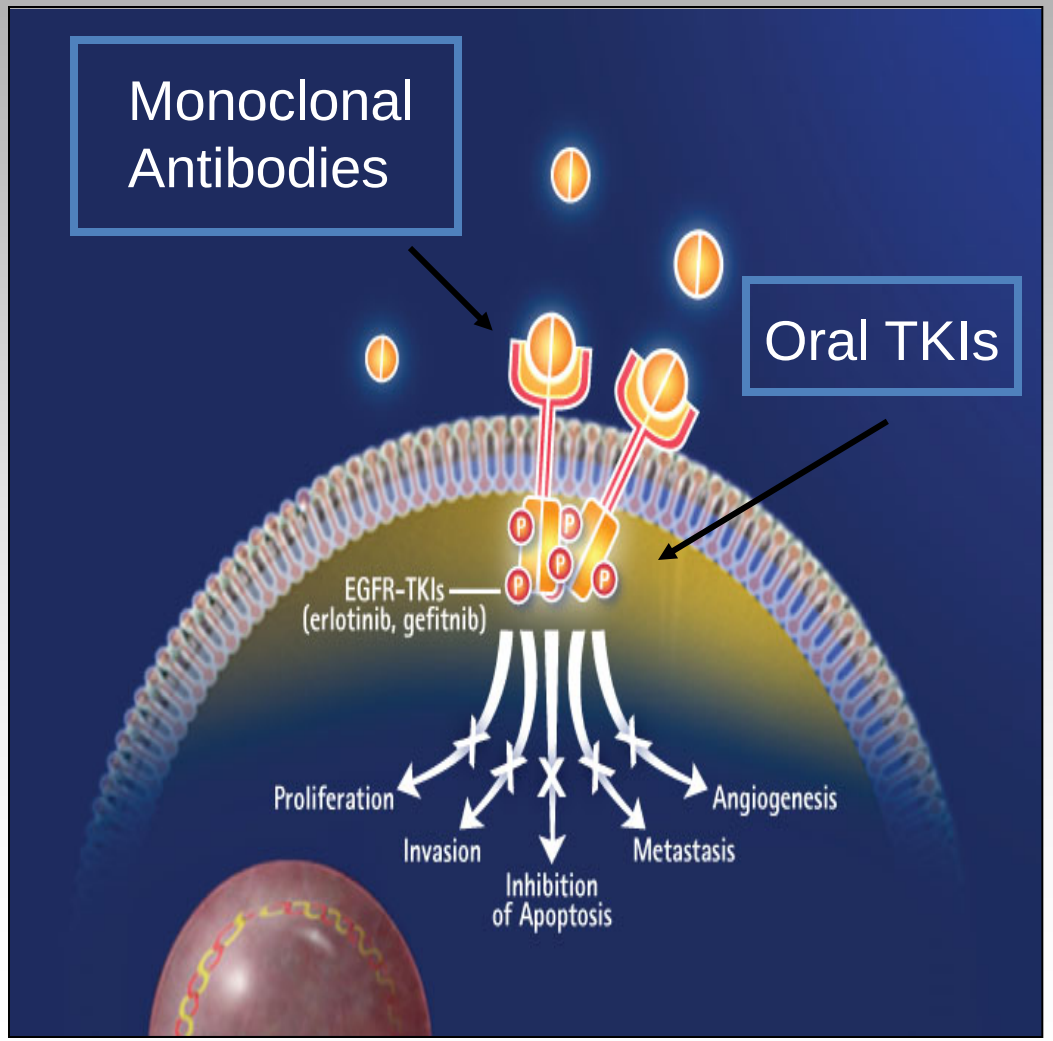
Inhibition of apoptosis

Metastasis

Angiogenesis

Frequently expressed and over-expressed in lung cancer

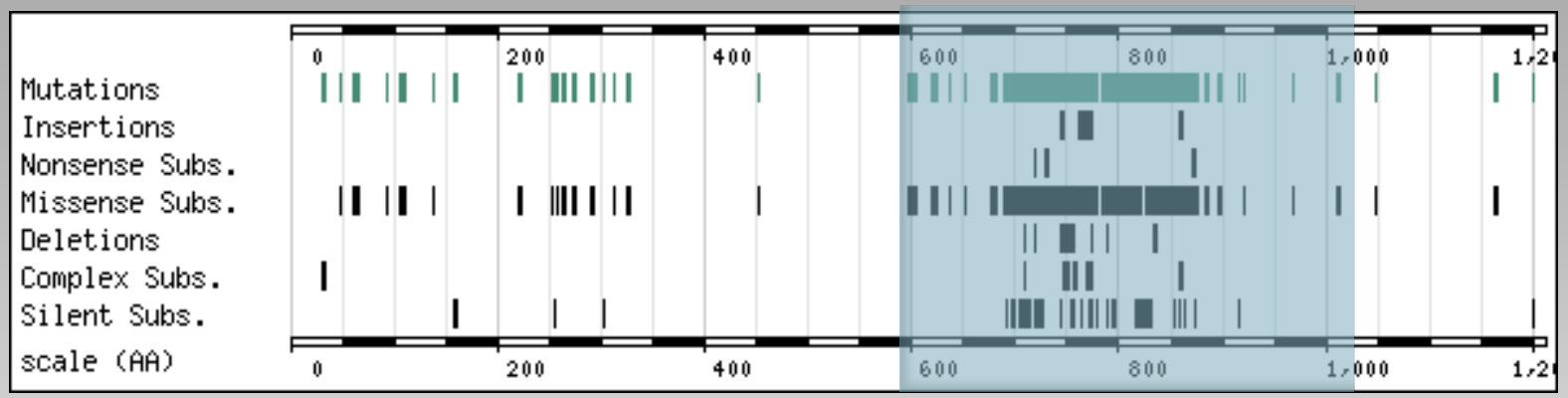
Associated with poor prognosis

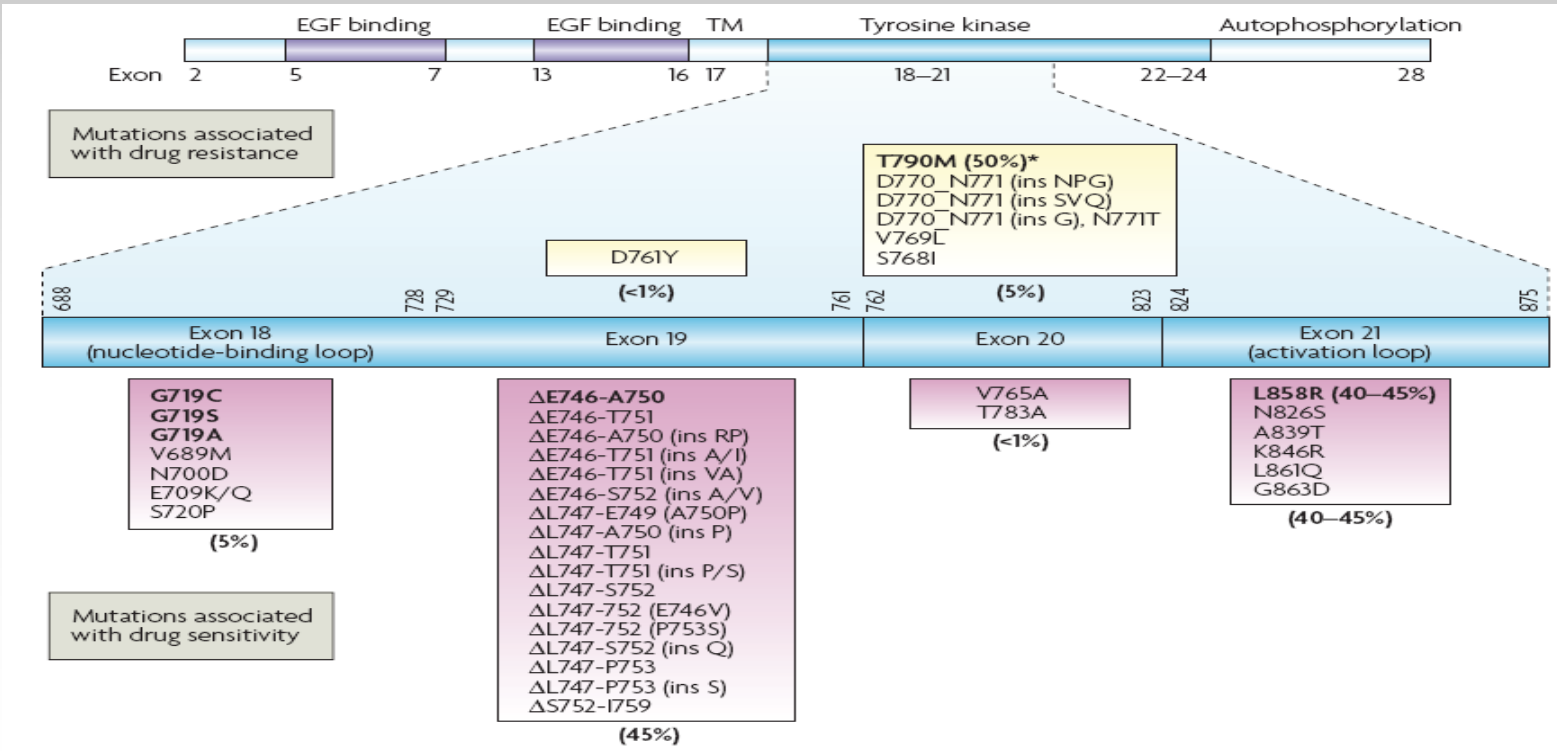


Characteristics of NSCLC patients Who Respond to EGFR TKIs

- Females
- Adenocarcinoma
- Asian
- Non-smokers

EGFR mutations





Sharma et al Nature Rev Cancer 7, 169, 2007

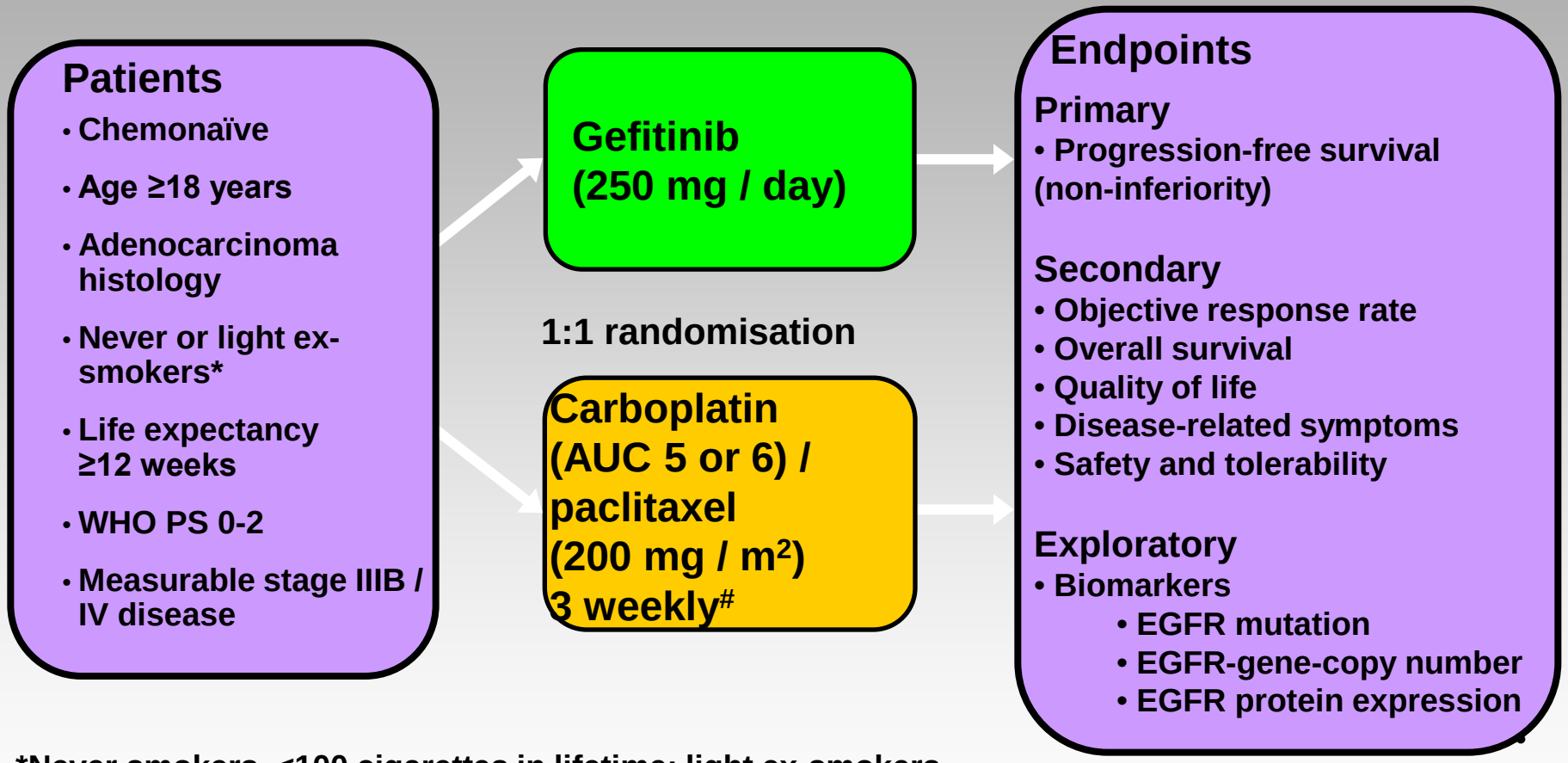
First-Line Treatment with Single-Agent EGFR Inhibitors in *Selected* Patient Populations

Clinically Selected Patients

IPASS
First Signal

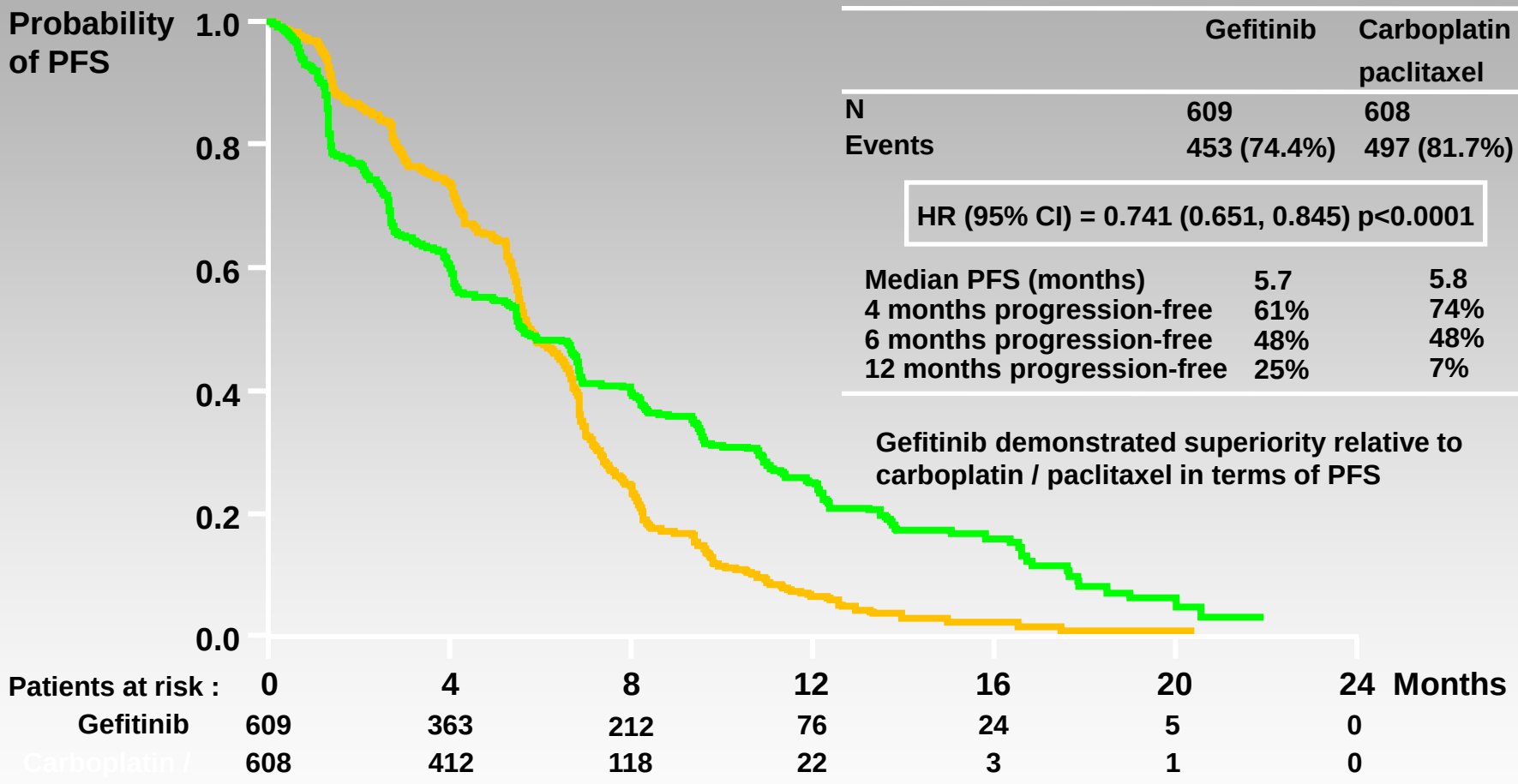
IPASS Study design

Conducted in China, Hong Kong, Indonesia, Japan, Malaysia, Philippines, Singapore, Taiwan and Thailand



*Never smokers, <100 cigarettes in lifetime; light ex-smokers, stopped ≥15 years ago and smoked ≤10 pack years; #limited to a maximum of 6 cycles
Carboplatin / paclitaxel was offered to gefitinib patients upon progression
WHO, World Health Organization; PS, performance status; AUC, area under curve; EGFR, epidermal growth factor receptor

Progression-free survival in ITT population



	Gefitinib	Carboplatin / paclitaxel
N	609	608
Events	453 (74.4%)	497 (81.7%)

HR (95% CI) = 0.741 (0.651, 0.845) p<0.0001

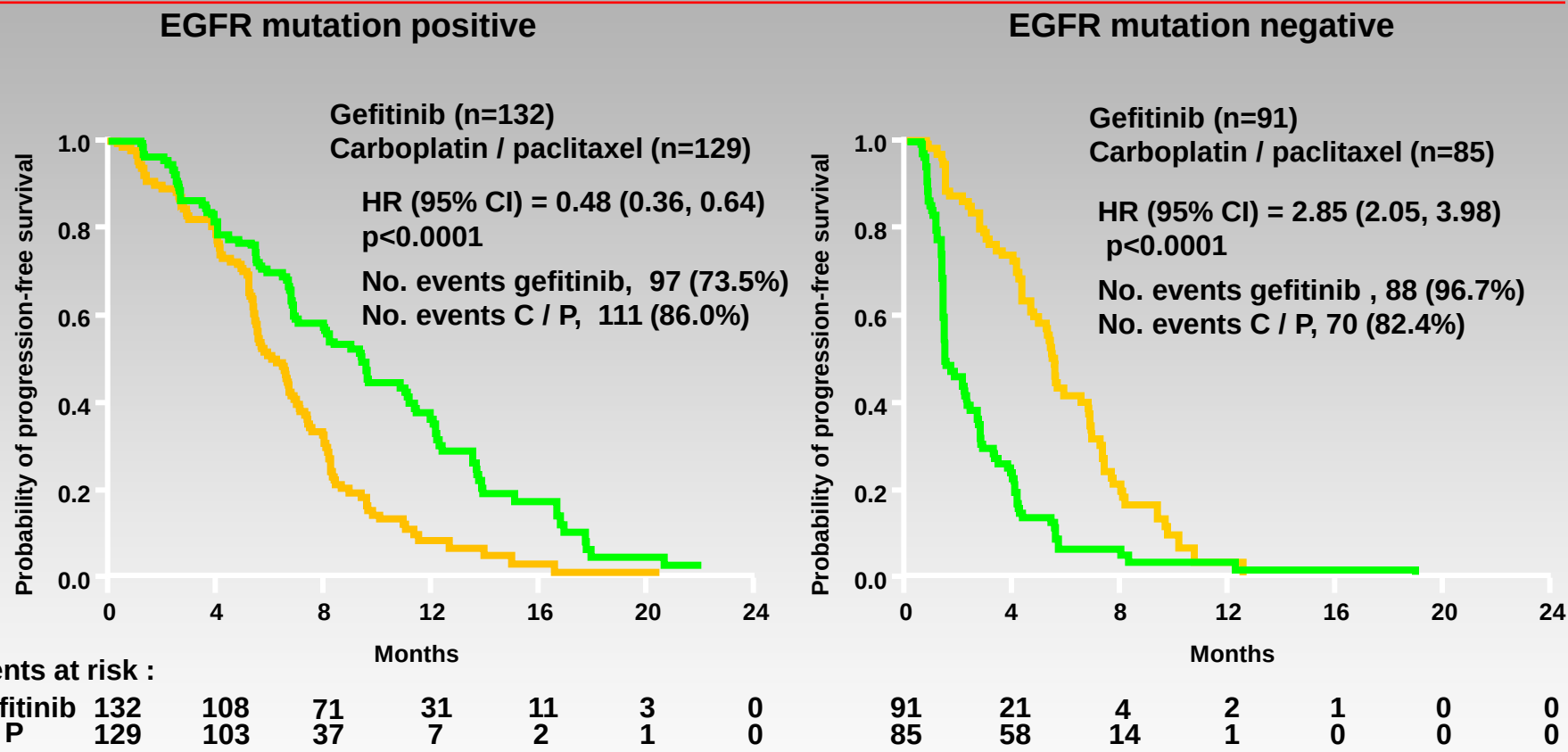
Median PFS (months)	5.7	5.8
4 months progression-free	61%	74%
6 months progression-free	48%	48%
12 months progression-free	25%	7%

Gefitinib demonstrated superiority relative to carboplatin / paclitaxel in terms of PFS

Primary Cox analysis with covariates

Mok et al 2008

IPass Progression-free survival in EGFR mutation positive and negative patients



Patients at risk :

Gefitinib	132	108	71	31	11	3	0	91	21	4	2	1	0	0
C / P	129	103	37	7	2	1	0	85	58	14	1	0	0	0

Treatment by subgroup interaction test, p<0.0001

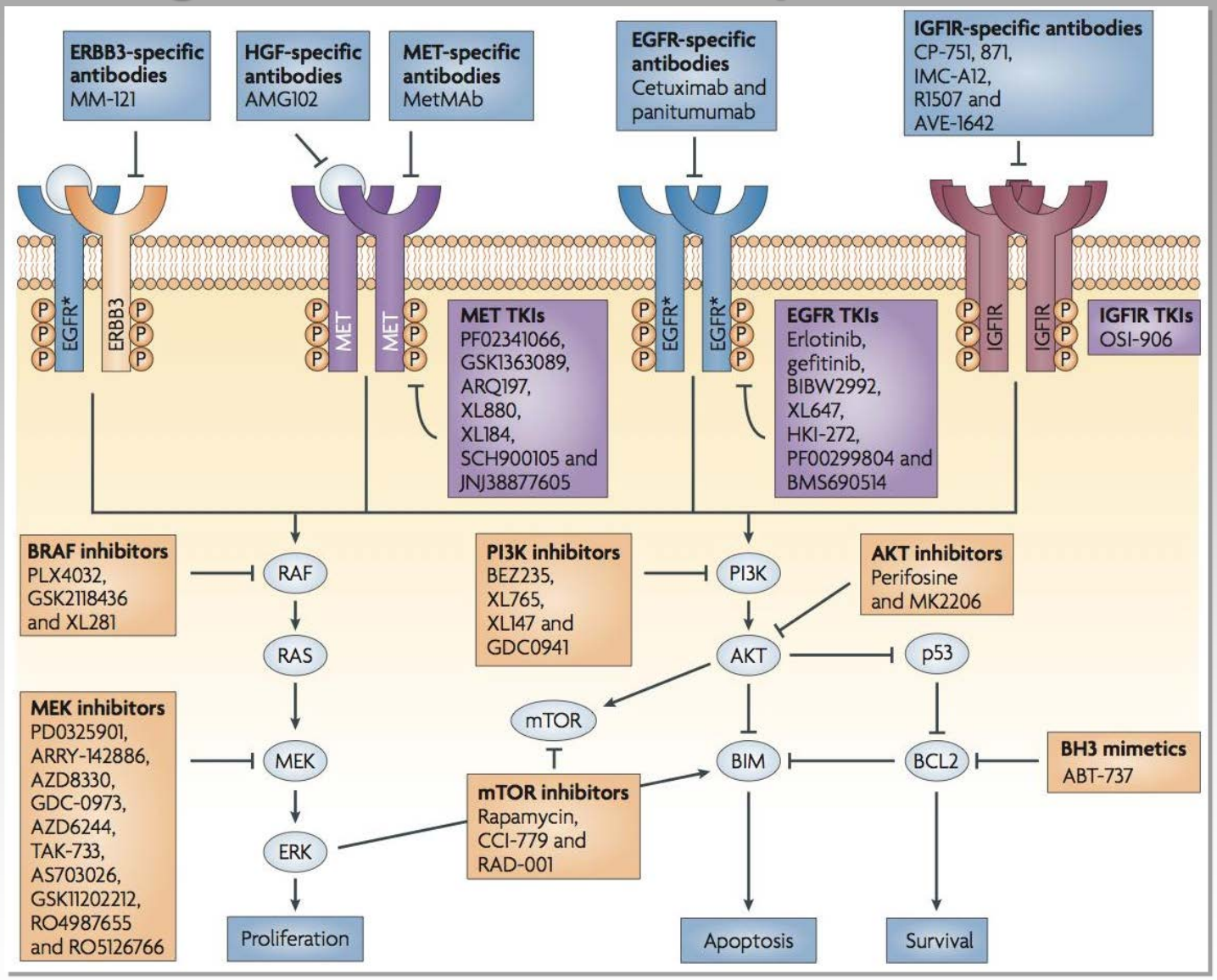
Cox analysis with covariates
HR <1 indicates a lower risk of progression on gefitinib

Mok et al 2008

Summary--EGFR

- EGFR TKI therapy is the treatment of choice for patients whose tumours have *EGFR* sensitizing mutations
- Selection should *not* be made on clinical grounds
- Even in Asian populations, 40% of highly selected patients have WT *EGFR*
- EGFR inhibitors should not be selected as “kinder and gentler” treatment for the elderly or infirm
- Patients with K-RAS mutations do not respond as well to EGFR inhibition
- There are several mechanisms of EGFR inhibitor resistance that are currently under investigation (T790M with HSP90 inhibition; compensatory signaling pathways such as c-MET)

Strategies to Inhibit RTK-dependent NSCLC



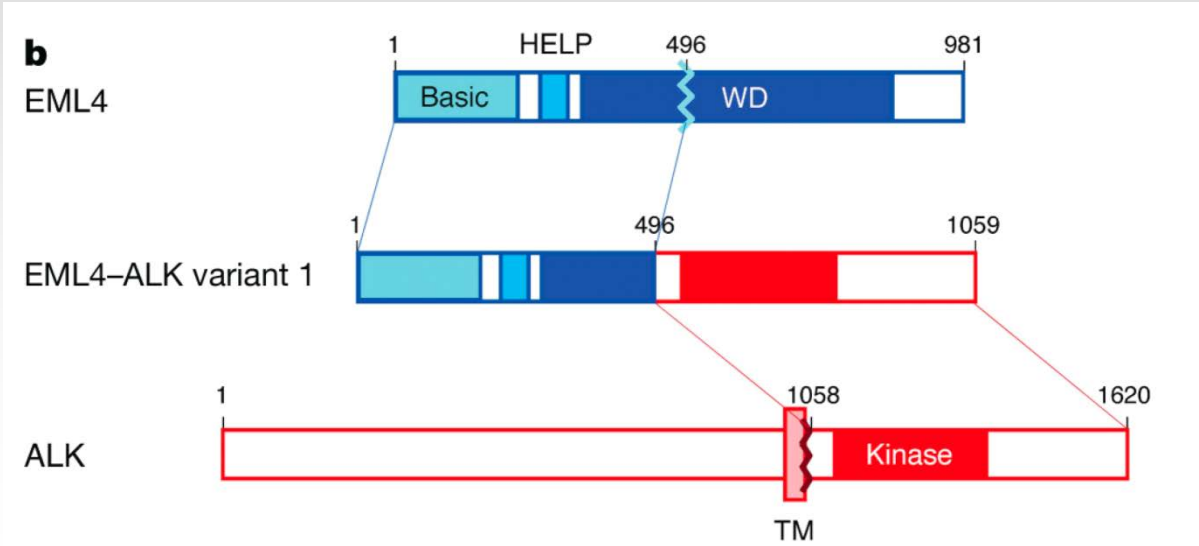
Pao & Chmielecki Nature Rev. 16:5371-73 (2010)

ALK

2007: EML4-ALK Rearrangements described as transforming event in NSCLC

Identification of the transforming *EML4–ALK* fusion gene in non-small-cell lung cancer

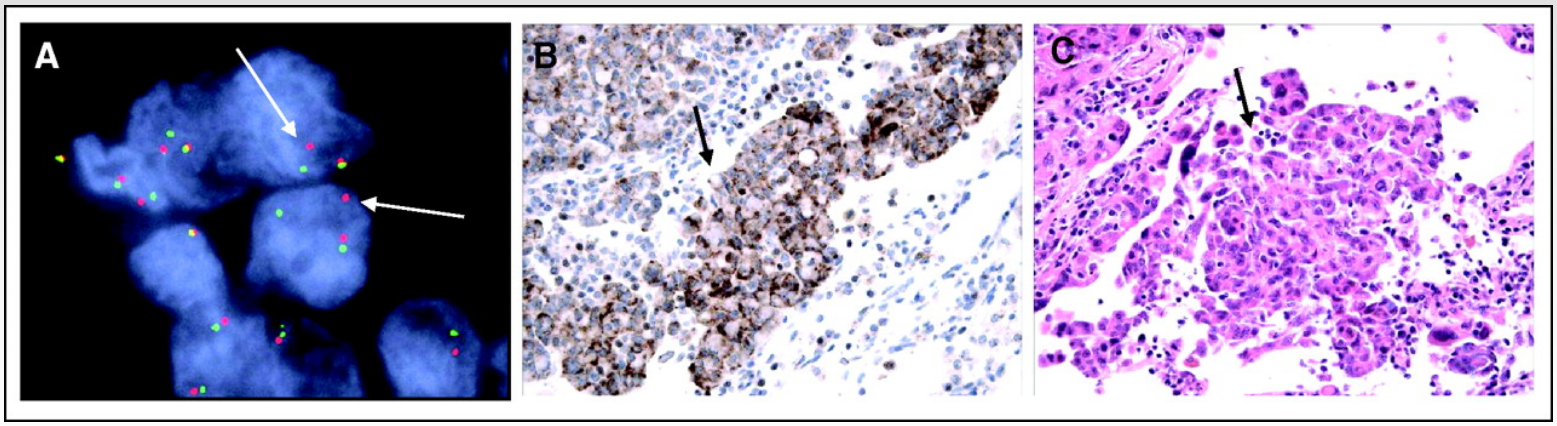
Manabu Soda[1,2], Young Lim Choi[1], Munehiro Enomoto[1,2], Shuji Takada[1], Yoshihiro Yamashita[1], Shunpei Ishikawa[5], Shin-ichiro Fujiwara[1], Hideki Watanabe[1], Kentaro Kurashina[1], Hisashi Hatanaka[1], Masashi Bando[2], Shoji Ohno[2], Yuichi Ishikawa[6], Hiroyuki Aburatani[5,7], Toshiro Niki[3], Yasunori Sohara[4], Yukihiko Sugiyama[2] & Hiroyuki Mano[1,7]



Soda et al., Nature 448: 561-566, 2007

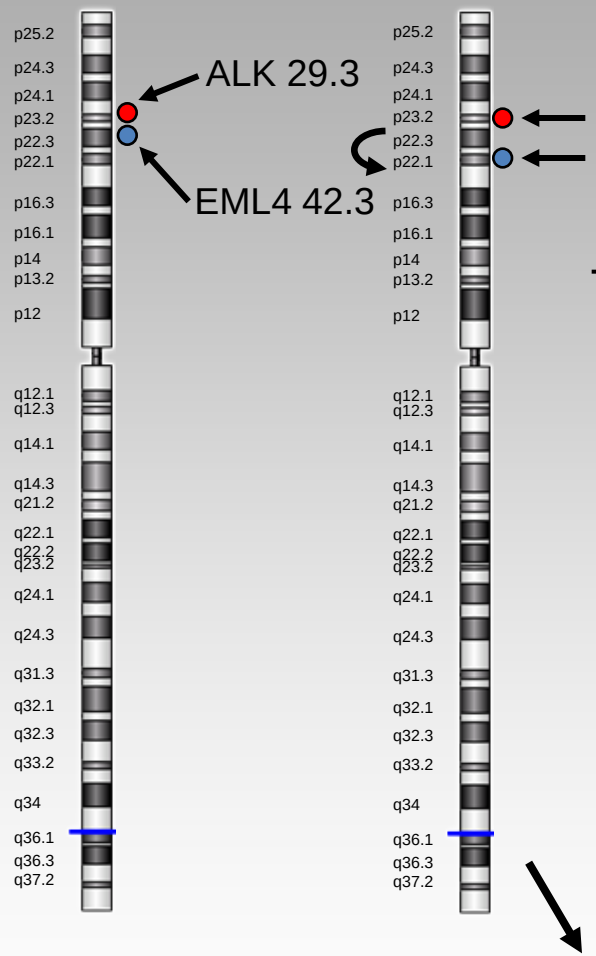
EML4-ALK

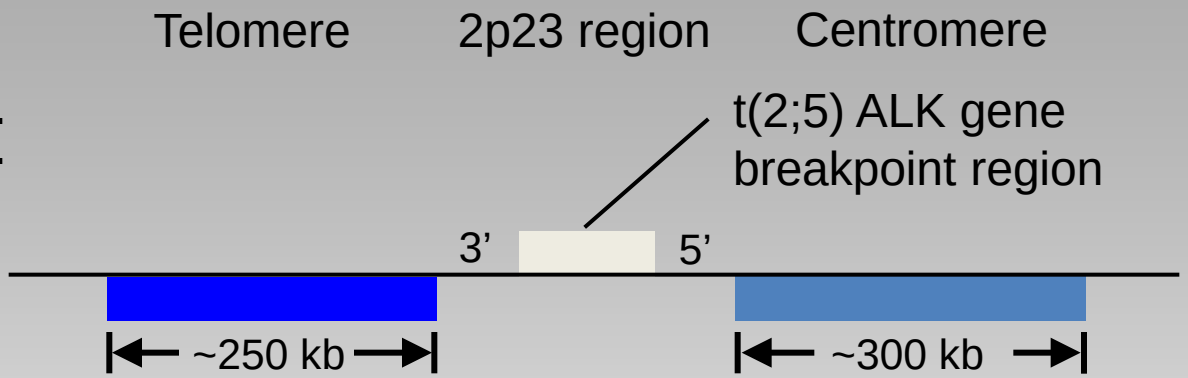
- Echinoderm microtubule-associated protein-like 4 (*EML4*) becomes fused with the anaplastic lymphoma kinase (*ALK*)
  - Inversion within chromosome 2p
- First identified in 2007 from a resected lung adenocarcinoma specimen



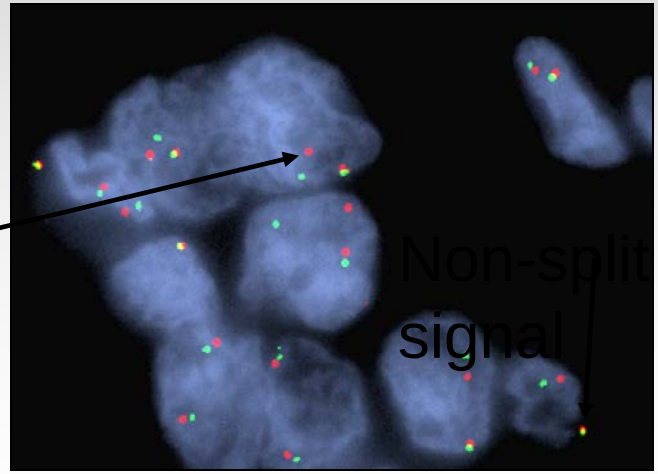
Soda, M., et al.. Nature, 2007. **448**(7153): p. 561-6.
Shaw, A. T. et al. J Clin Oncol; 27:4247-4253 2009

FISH Assay for *ALK* Rearrangement*





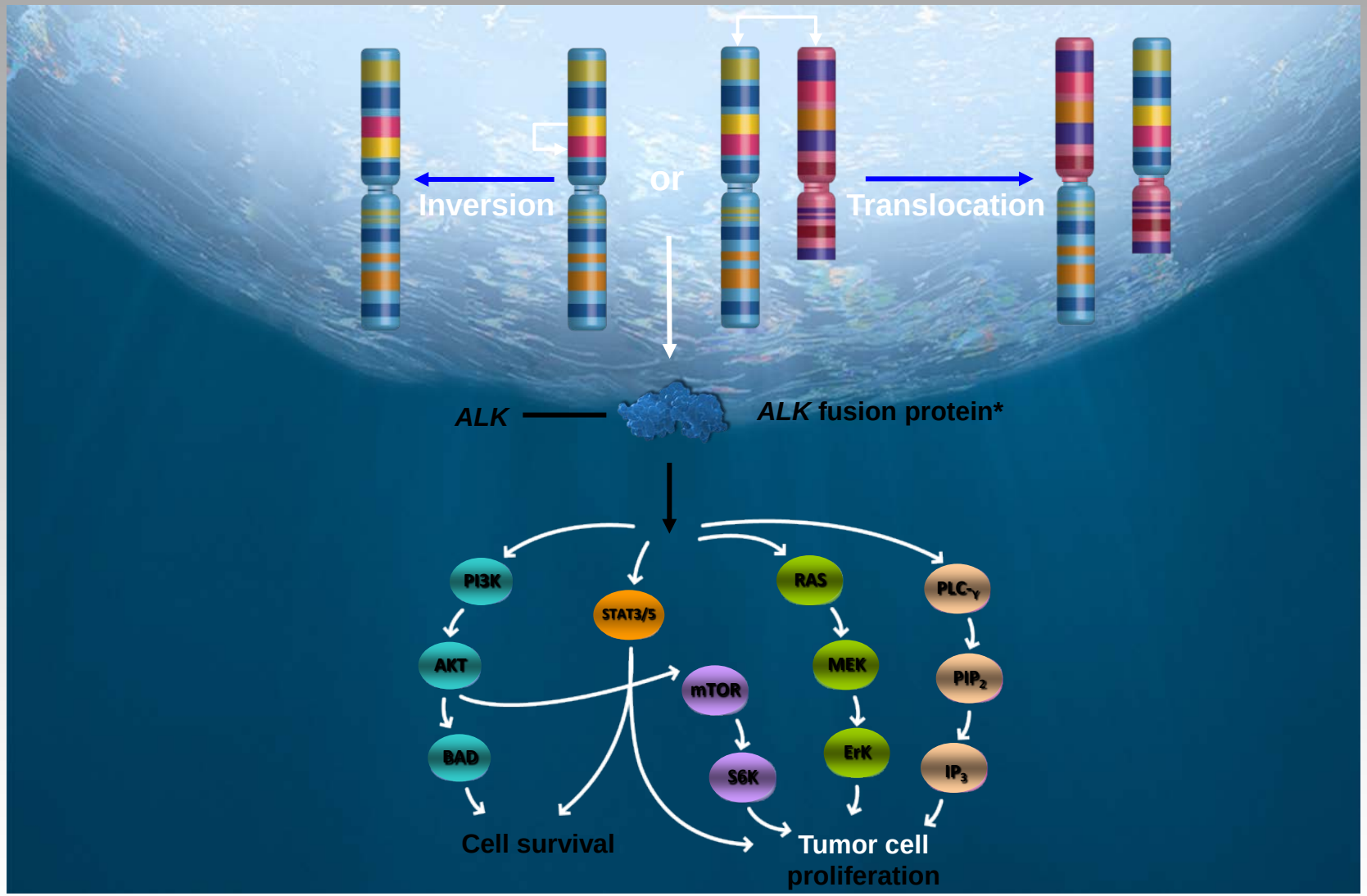
Break-apart FISH assay for *ALK*-fusion genes[1]



ALK break-apart FISH assay Courtesy John Iafrate Massachusetts General Hospital]

[1]Shaw AT et al. J Clin Oncol 2009;27:4247–4253

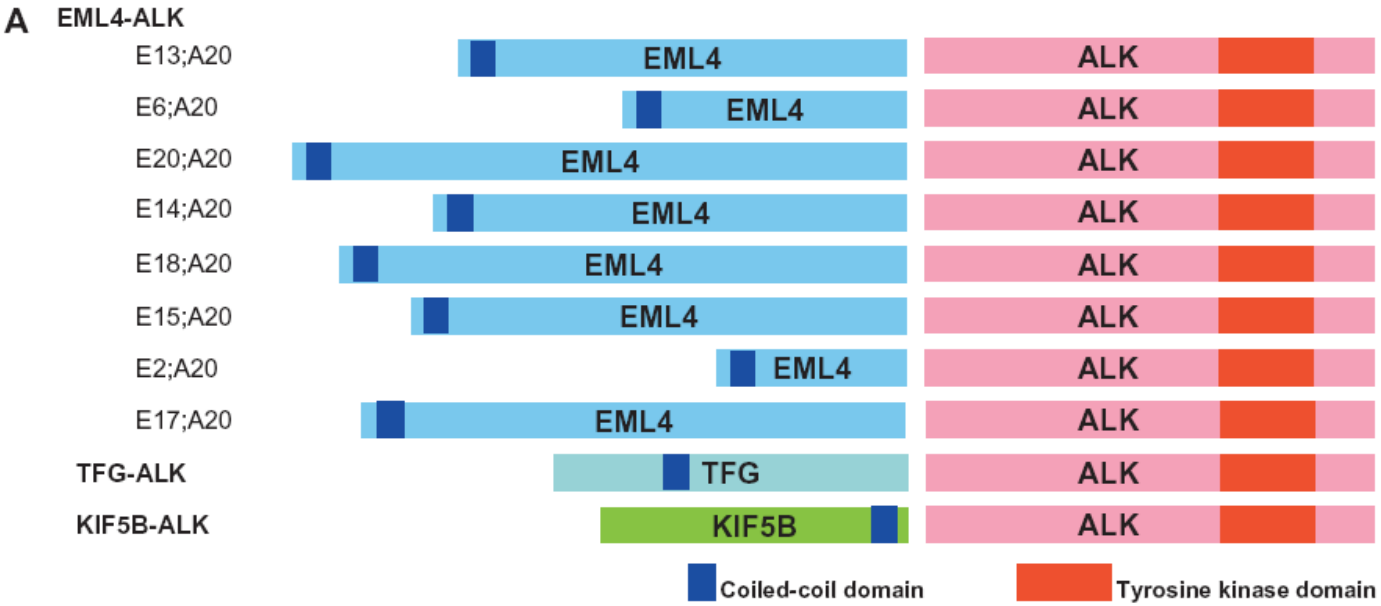
ALK Pathway

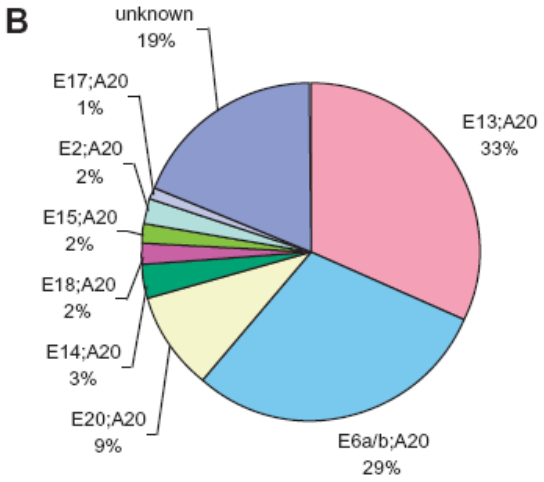


*Subcellular localization of the ALK fusion gene, while likely to occur in the cytoplasm, is not confirmed.[1,2]

1. Inamura K et al. J Thorac Oncol 2008;3:13–17
2. Soda M et al. Proc Natl Acad Sci U S A 2008;105:19893–19897
Figure based on: Chiarle R et al. Nat Rev Cancer 2008;8(1):11–23
Mossé YP et al. Clin Cancer Res 2009;15(18):5609–5614; and Data on file. Pfizer Inc.

EML4-ALK Variants





EML4-ALK Variants

E13;A20	E13;A20 (variant 1), E13;ins69 A20
E6;A20	E6a/b;A20 (variant 3a/b)
E20;A20	E20;A20 (variant 2), E20;ins18A20
E14;A20	E14;ins11del49A20(variant 4'), E14;del12A20 (variant 7)
E18;A20	E18;A20 (variant 5')
E15;A20	E15 del19;del20A20 (variant 4)
E2;A20	E2;A20 &E2;ins117A20 (variant 5a/b)
E17;A20	E17;ins68A20

NSCLC Cell lines

H3122 and DFCI032 contain E13;A20. H2228 contain E6;A20

Crizotinib Selectivity Profile

Upstate 102 kinase

Kinase	% Inhibition
Met(h)	94
Tie2(h)	103
TrkA(h)	102
ALK(h)	100
TrkB(h)	100
Abl(T315I)(h)	98
Yes(h)	96
Lck(h)	95
Rse(h) [SKY]	94
Axl(h)	93
Fes(h)	93
Lyn(h)	93
Arg(m)	91
Ros(h)	90
CDK2/cyclinE(h)	87
Fms(h)	84
EphB4(h)	80
Bmx(h)	79
EphB2(h)	77
Fgr(h)	73
Fyn(h)	68
IR(h)	64
CDK7/cyclinH/MAT1(h)	58
cSRC(h)	58
IGF-1R(h)	56
Aurora-A(h)	54
Syk(h)	52
FGFR3(h)	50
PKCμ(h)	50
BTK(h)	35
CDK1/cyclinB(h)	25
p70S6K(h)	24
PRK2(h)	22
PAR-1Bα(h)	21
PKBß(h)	21
Ret(h)	21
GSK3ß(h)	18
Flt3(h)	17
MAPK1(h)	17
ZAP-70(h)	17
Abl(h)	16
c-RAF(h)	16
PKD2(h)	15
ROCK-II(h)	14
Rsk3(h)	14
GSK3α(h)	11
CDK5/p35(h)	10
PDGFRα(h)	10
Rsk1(h)	7
SGK(h)	6
CHK1(h)	5
ErbB4(h)	5
Rsk2(h)	5
JNK1α1(h)	4
PKBα(h)	4
Blk(m)	3
CDK3/cyclinE(h)	3
PKCι(h)	3
PKCθ(h)	3
CDK2/cyclinA(h)	2
PAK2(h)	2
PKCßI(h)	2
Pim-1(h)	1
PKCη(h)	1
SAPK4(h)	1
CaMKII(r)	0
MKK7ß(h)	0
CaMKIV(h)	-1
CHK2(h)	-1
CK2(h)	-1
JNK2α2(h)	-1
MKK6(h)	-1
CK1δ(h)	-2
PKCα(h)	-2
MAPK2(h)	-3
MEK1(h)	-3
PKCδ(h)	-3
PKCε(h)	-3
Plk3(h)	-3
PKCßII(h)	-5
MSK1(h)	-6
PDGFRß(h)	-6
PKCζ(h)	-6
SAPK3(h)	-6
MAPKAP-K2(h)	-7
PKA(h)	-7
AMPK(r)	-9
CDK6/cyclinD3(h)	-9
CSK(h)	-9
SAPK2a(h)	-9
JNK3(h)	-10
PKBγ(h)	-10
IKKα(h)	-11
NEK2(h)	-11

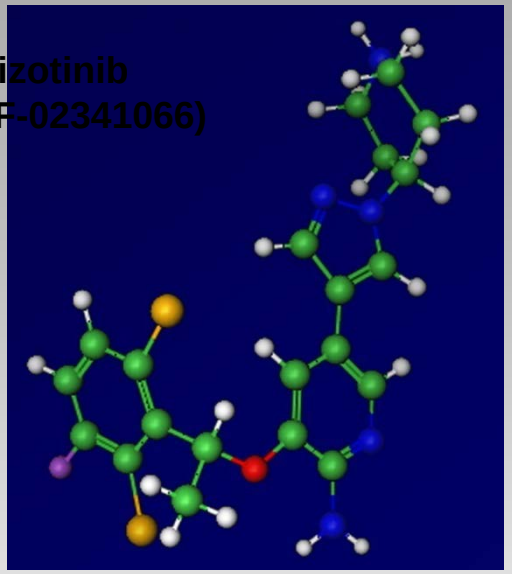
13 kinase "hits" <100X selective for c-MET

Cellular selectivity on 10 of 13 relevant hits

Kinase	IC_{50} (nM) mean*	Selectivity ratio
c-MET	8	–
ALK	20	2X
RON	298	34X
	189	22X
Axl	294	34X
	322	37X
Tie-2	448	52X
Trk A	580	67X
Trk B	399	46X
Abl	1,159	166X
IRK	2,887	334X
Lck	2,741	283X
Sky	>10,000	>1,000X
VEGFR2	>10,000	>1,000X
PDGFRβ	>10,000	>1,000X

*The cellular kinase activities were measured using ELISA capture method

Crizotinib (PF-02341066)



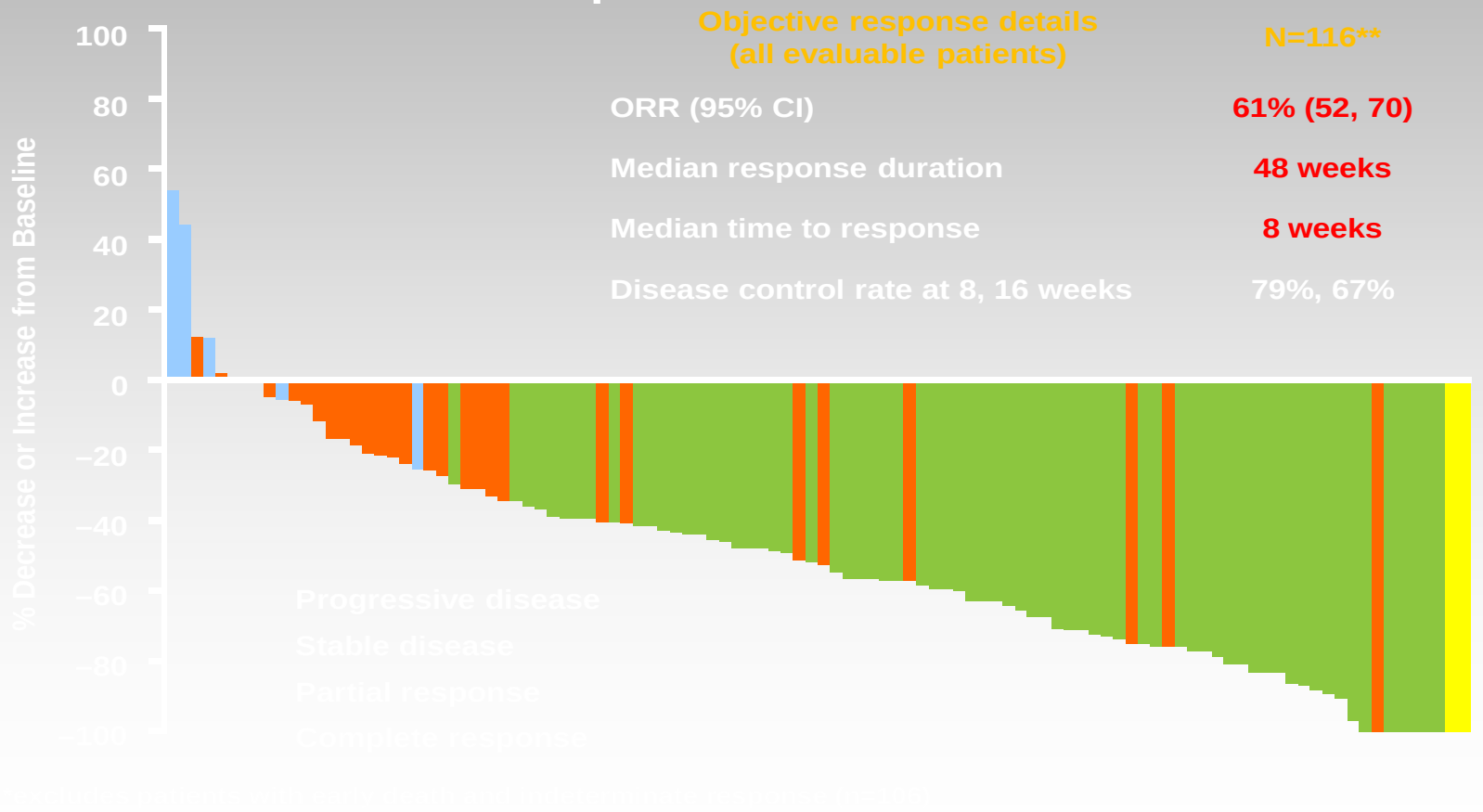
Selectivity findings

- **Crizotinib – ALK and c-MET inhibition at clinically relevant dose levels**
- **Crizotinib – <u>low</u> probability of pharmacologically relevant inhibition of any other kinase at clinically relevant dose levels**

Pfizer Inc. Data on file

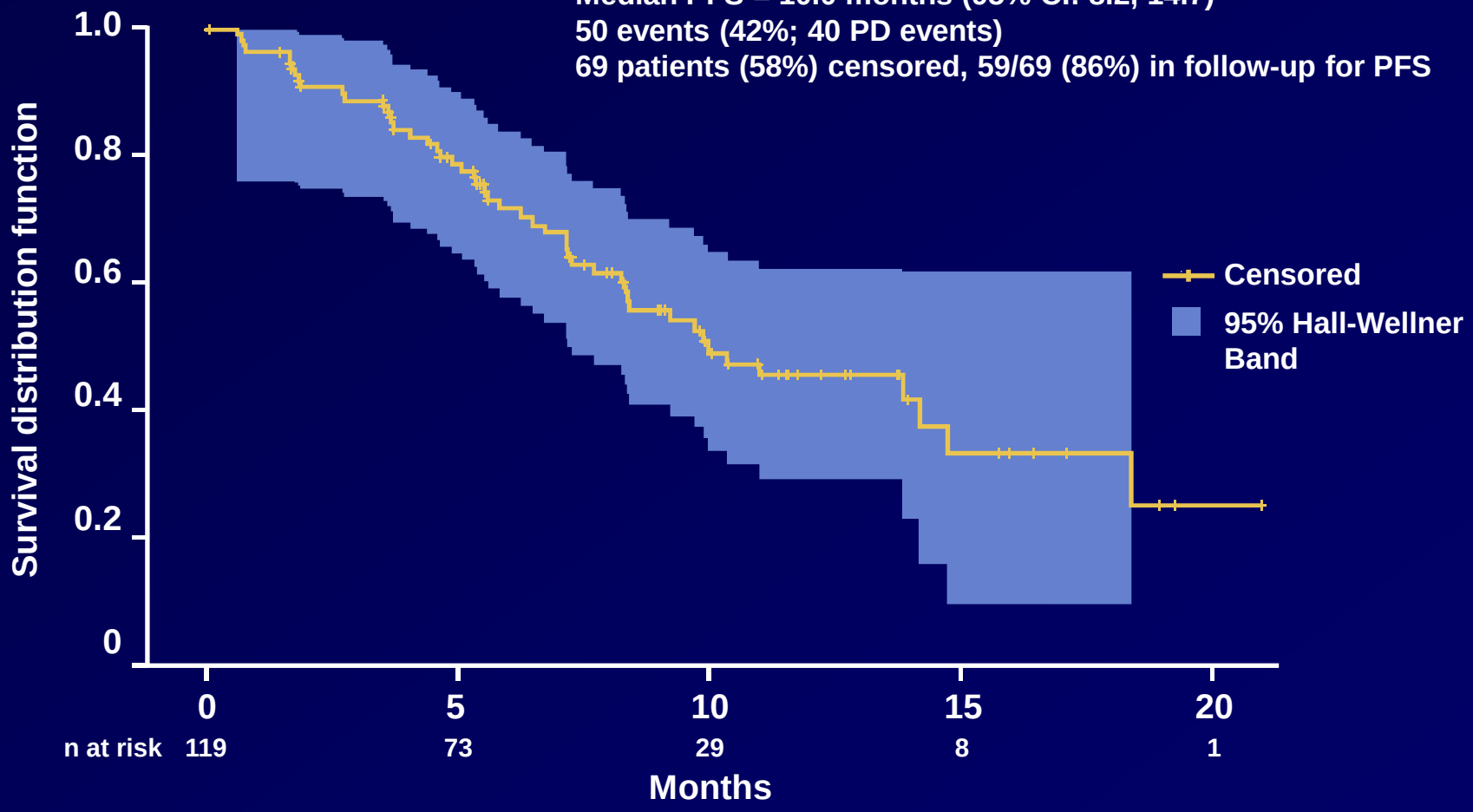
Tumor Responses to Crizotinib

Best Percent Change from Baseline in Target Lesions*



Camidge, et al, Lancet Oncology, 2011; Kwak, et al, NEJM, 2011

Progression-Free Survival (N=119)



Camidge, et al, 2011; Kwak, et al, 2011

Crizotinib--UofC

- Phase I: 74 consented, 27 enrolled
- Phase II (ALK): 52 screened, 12 enrolled
- Phase III (ALK): 25 screened, 3 enrolled
- Future Goals (ALK):
  - New inhibitors (Ariad, Cephalon, Astellas)
  - Relevance of HSP90 inhibitors (Synta, Daiichi)
  - Work with CALGB for analysis of tumor tissues, as well in early stage disease
  - Determine the relevance in maintenance

WebApp Therapy Finder—Lung Cancer (www.collabrx.com)

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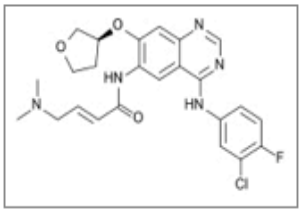
Breaking News:
afatinib may be effective in patients resistant to first line therapy



Other Drugs in the News

Provide Lung Cancer Information

Lung Cancer Stage
- Early (stages 0, I and II)
- Stage III
- Stage IV (Advanced Stage)

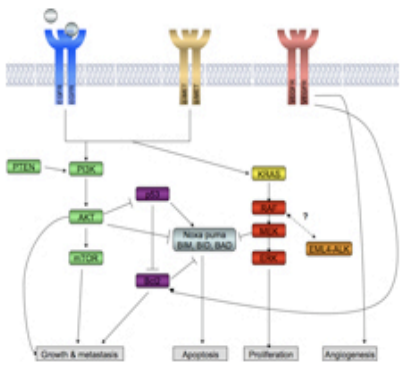
Histological Information
- Adenocarcinoma
- Adenosquamous Carcinoma
- Large Cell Carcinoma
- Squamous Cell Carcinoma
- NOS/Other
- Small Cell Lung Cancer

Metastatic Sites
- Lymph Nodes
- Liver
- Adrenal Glands
- Brain/CNS
- Bone
- Other

Mutation Information
- EGFR: Unknown
- kRAS: Unknown
- EML4-ALK: Unknown
- VeriStrat: Unknown

clear form | SEARCH

Learn the Science Behind This Tool



Cancer Commons, ASCO

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Stage Stage IV **Hist** Adenocarcinoma **Mets** Adrenal Glands, Bone **EGFR**– **EML4**+ **KRAS**–

EML4-ALK mutation: Consider Xalkori

You have indicated that your cancer has the EML4-ALK abnormality.

You belong to a group of patients who may benefit from medications such as Xalkori and ganetespib (read more). The EML4-ALK 'translocation' promotes cancer growth and survival. Hence your cancer may be effectively treated with medications that block EML4-ALK. See below for more information about relevant drugs and clinical trials.

Top drugs to consider:

Xalkori: (also called crizotinib) belongs to a class of drugs called ALK inhibitors. It was approved by the FDA on August 26th 2011. Once a drug gains approval your doctor can prescribe it to you for treatment of your cancer. This drug is exciting because it reduced tumor size in most patients during clinical testing (read more).

ganetespib: (also called STA-9090) belongs to a class of drugs called Hsp90 inhibitors. This drug is not yet approved by the FDA. It is being tested in clinical trials for a variety of cancers. In one case study from a patient with the EML4-ALK translocation who was resistant to Xalkori, ganetespib showed "signifiicant tumor shrinkage" after three weeks treatment. Over all, preliminary results have been positive, especially in non-small cell lung cancer patients who do *not* have mutations in EGFR and KRAS genes. You can try out this drug by enrolling in one of the clinical trials listed below.

Summary

Key gene	Potentially Relevant Drug Classes
ALK, EML4	ALK inhibitors, Hsp90 inhibitors

Trials | Drugs | Literature

Clinical Trials

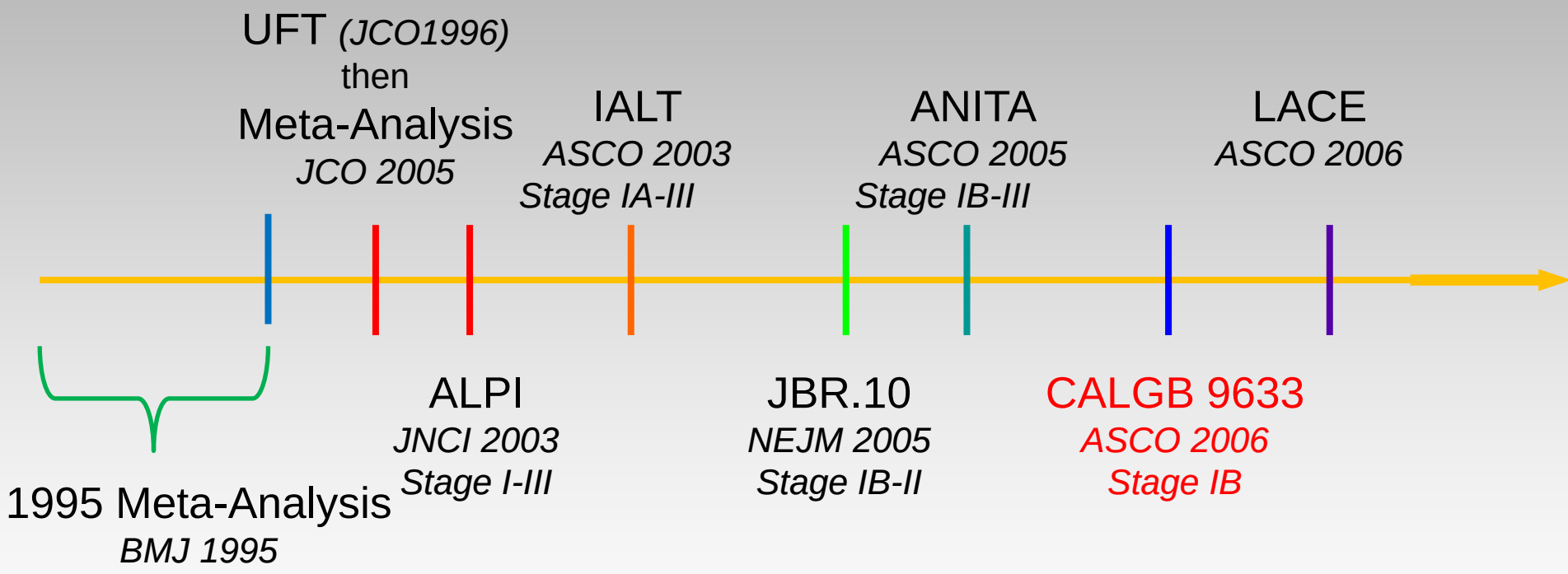
Details of your condition will determine eligibility for any specific trial. To select clinical trials for which you may be qualified, click here. Also, ask your doctor for more information.

SELECT CLINICAL TRIALS

Trial	Drug	Status	Phase
IPI-504 in NSCLC Patients With ALK Translocations [NCT01228435]	IPI-504	Recruiting	Phase 2
A Study Of Combined C- MET Inhibitor And PAN-HER Inhibitor (PF-02341066 And PF-00299804) In Patients With Non- Small Cell Lung Cancer [NCT01121575]	Xalkori, PF-00299804	Recruiting	Phase 1
Erlotinib Is Being Studied With Or Without An Investigational Drug, PF-02341066, In Patients With Lung Cancer [NCT00965731]	Tarceva, Xalkori	Recruiting	Phase 1 and 2
Study of Ganetespib (STA-9090) + Docetaxel in Advanced Non Small Cell Lung Cancer [NCT01348126]	Ganetespib, Docetaxel	Recruiting	Phase 2, Phase 3
A Study of the HSP90 Inhibitor, STA-9090 in Subjects With Stage IIIB or IV Non-Small Cell Lung Cancer (NSCLC) [NCT01031225]	STA-9090	Recruiting	Phase 2
A Study of AUY922 in Non-small-cell Lung Cancer Patients Who Have Received Previous Two Lines of Chemotherapy [NCT01124864]	AUY922	Recruiting	Phase 2
A Dose Finding Study With Oral LDK378 in Patients With Tumors Characterized by Genetic Abnormalities in Anaplastic Lymphoma Kinase (ALK) [NCT01283516]	LDK378	Recruiting	Phase 1
Study of STA-9090, Administered Twice-Weekly in Patients With Solid Tumors [NCT00688116]	Ganetespib	Recruiting	Phase I
A Dose Escalation Study of STA-9090 and Docetaxel in Patients With Solid Tumors [NCT01183364]	Ganetespib	Recruiting	Phase 1
A First In Patient, Study Of Investigational Drug PF-03446962 In Patients With Advanced Solid Tumors [NCT00557856]	PF-03446962	Recruiting	Phase 1
A Study of DS-2248, in Subjects With Advanced Solid Tumors [NCT01288430]	DS-2248	Recruiting	Phase 1
Study of an Investigational Drug, ASP3026, in Patients With			

Early Stage Disease

Timeline Major Adjuvant Systemic Therapy Trials



Timeline Major Adjuvant Systemic Therapy Trials

Molecular markers in early stage: **Frequency of mutations**

Gene	Alteration	Frequency in NSCLC
AKT1	Mutation	1%
ALK	Rearrangement	3-7%
BRAF	Mutation	1-3%
DDR2	Mutation	4%
EGFR	Mutation	10-35%
FGFR1	Amplification	20%
HER2	Mutation	2-4%
KRAS	Mutation	15-25%
MEK1	Mutation	1%
MET	Amplification	2-4%
NRAS	Mutation	1%
PIK3CA	Rearrangement	1-3%
PTEN	Mutation	4-8%
ROS1	Mutation	1%

EGFR and K-Ras in early stage lung cancer

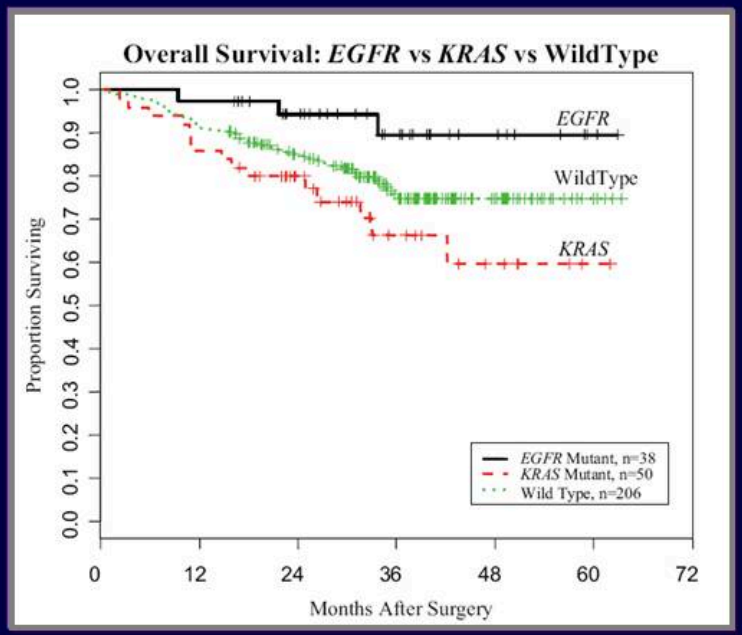
TABLE 1. Clinical Characteristics of 296 Patients Whose Tumors Were Genotyped

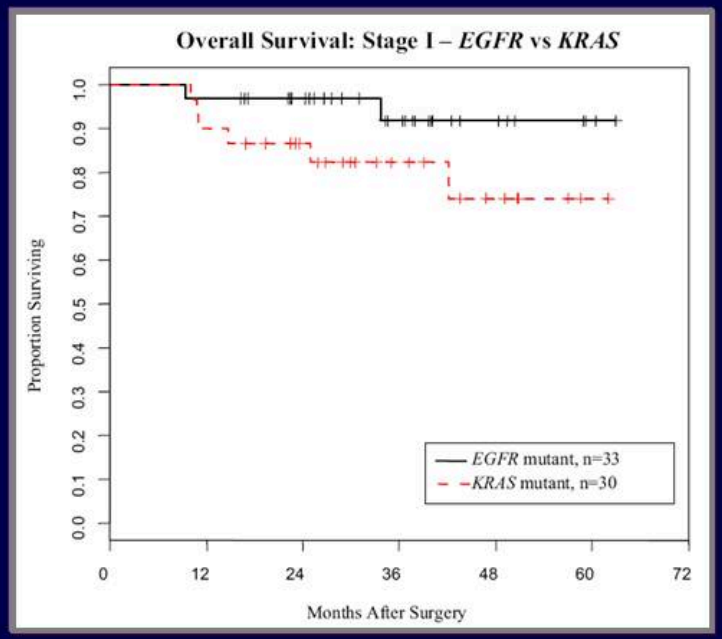
	EGFR Mutation $N = 40$	*KRAS* Mutation $N = 50$	*EGFR/KRAS* Wild-type $N = 206$	p
Age	70 (35–86)	70 (42–86)	68 (39–89)	
Gender				0.581
Men	14 (35%)	23 (46%)	83 (40%)	
Women	26 (65%)	27 (54%)	123 (60%)	
Stage				0.064
IA	21 (53%)	16 (32%)	103 (50%)	
IB	14 (35%)	14 (28%)	48 (23%)	
II	1 (2%)	7 (14%)	21 (10%)	
III	4 (10%)	13 (26%)	34 (17%)	
Cigarette smoking				<0.001
Never	18 (47%)	4 (8%)	14 (5%)	
Former	20 (53%)	37 (74%)	154 (83%)	
Current	0	9 (18%)	38 (12%)	
Chemotherapy				0.405
None	25 (62%)	26 (52%)	128 (62%)	
Adjuvant/ neoadjuvant	15 (38%)	24 (48%)	78 (38%)	
Mutation				
	L858R: 19 Exon 19 del: 19 Exon 20 ins: 1 Exon 21 (H835L): 1	G12C: 22 G12V: 14 G12A: 7 G12D: 6 G13C: 1	N/A	

Marks et al JTO, 2008

EGFR and K-Ras in early stage lung cancer

Survival with/without EGFR and KRAS Mutations Among US Patients with Resected NSCLC

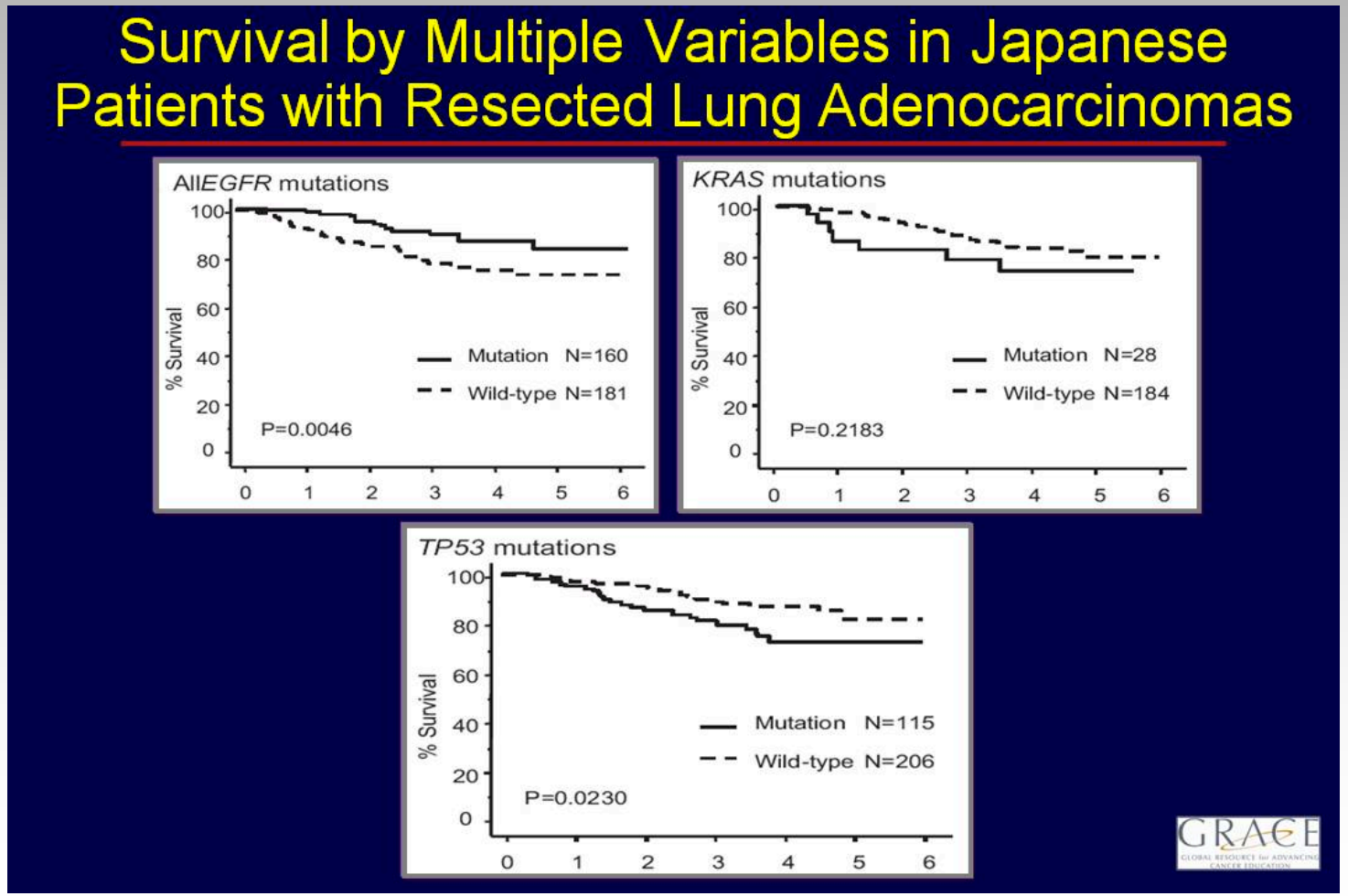




Marks, J Thorac Oncol 2008

GRACE
GLOBAL RESOURCE for ADVANCING CANCER EDUCATION

EGFR and K-Ras in early stage lung cancer



Takayuki, et al JTO, 2009

MOLECULARLY TARGETED AGENTS IN ADJUVANT SETTING

A Phase III Randomized, Double-Blind, Placebo-Controlled Trial of the Epidermal Growth Factor Receptor Inhibitor, Gefitinib in Completely Resected Stage IB-IIIA Non Small Cell Lung Cancer

NCIC CTG BR.19

G.D. Goss, MD

NCIC CTG BR.19

Trial Schema

Accrual: 503 (Closed)[1]

Eligibility

Resected IB, II and IIIA NSCLC ECOG PS 0-2

R

Gefitinib (n = 251) daily x 2 years

Placebo (n = 252) daily x 2 years

[1] In April 2005, accrual was closed early due to the inferiority of gefitinib arm.

Patients were stratified by stage, histology, post-operative radiation, sex and adjuvant chemotherapy.

Goss GD et al. *Proc ASCO* 2010;Abstract LBA7005.

Overall Survival and Disease-Free Survival

	Gefitinib (n = 251)	Placebo (n = 252)	Hazard Ratio	*p*-value
Median overall survival (OS)	5.1 years	Not reached	1.23	0.136
Median disease-free survival (DFS)	4.2 years	Not reached	1.22	0.152

Multivariate analysis

- **Age ≥65 years and tumor size ≥4 cm (p = 0.0003) were significantly associated with shorter survival.**
- **Gefitinib remained not significant, but there was a trend suggesting it may be harmful (p = 0.097).**

Goss GD et al. *Proc ASCO* 2010;Abstract LBA7005.

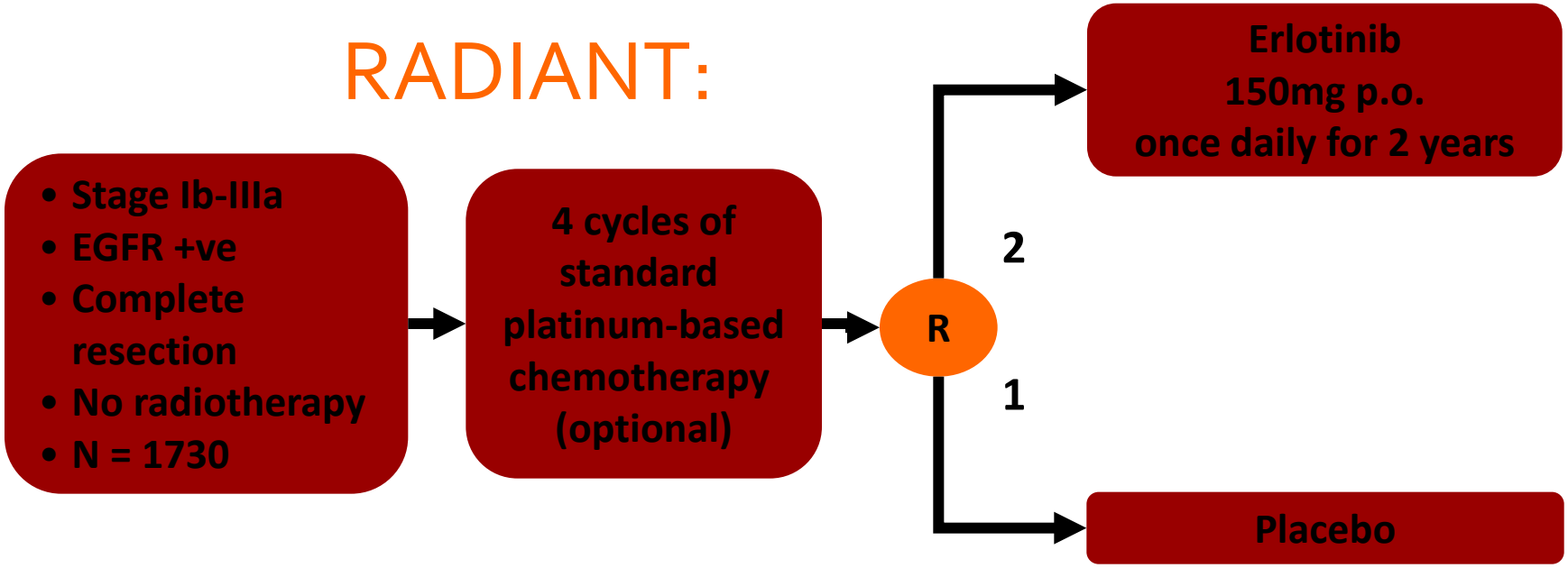
Conclusion: **NCIC CTG BR.19**

- Gefitinib was well tolerated.
- Gefitinib did not improve DFS and OS in patients with completely resected early stage NSCLC in this underpowered study.
- KRAS mutation status, EGFR by FISH or EGFR sensitizing mutation status were neither prognostic nor predictive of survival in exploratory analysis.
- A targeted agent that improves OS in NSCLC in the adjuvant setting has yet to be demonstrated.
- Currently, the treatment of choice for patients in good performance is chemotherapy.
- The results of the RADIANT trial of adjuvant erlotinib are awaited (NCT00373425).

Goss GD et al. *Proc ASCO* 2010;Abstract LBA7005; www.clinicaltrials.gov.

MOLECULARLY TARGETED AGENTS IN ADJUVANT SETTING

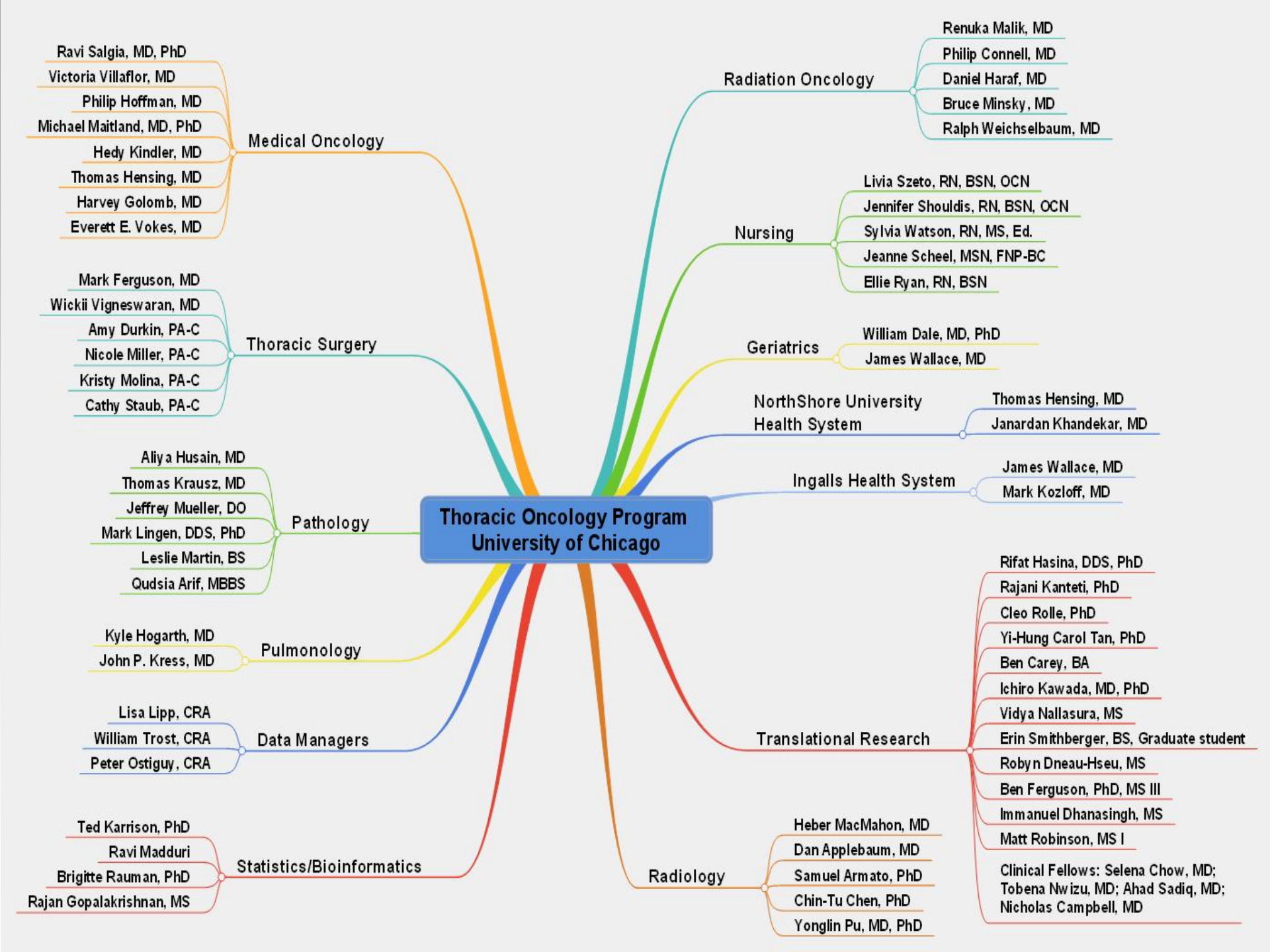
RADIANT:



- Primary endpoint = disease-free survival (all patients, IHC+ve and/or FISH+ve)
- Co-primary = DFS in FISH+ve (US); TBC in Europe
- Secondary endpoints: OS, safety, biomarkers
- Status: 1st patient entered 09/2006, 1. interim 1Q11, 2. interim 2Q12, final analysis 3Q13

Summary for Promising Targets in Lung Cancer

- ALK targeting has come to clinical fruition, with recent FDA approval of crizotinib
- EGFR is approved therapy for second line, and if mutated for first line
- Early stage molecular characteristics are beginning to be defined
- It will be important to arrive at a number of targets based on biology of lung cancer, especially in the early stage setting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