16th CONGRESS Lung ON CANCER

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GECP lung cancer research

Fusions in lung cancer

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DISCLOSURES

Personal financial interests

- **Consulation Honoraria**: Amgen, AstraZeneca, Bayer, BMS, Boehringer Ingelheim, F. Hoffmann-La Roche, Janssen, Lilly, MSD, Pfizer, Sanofi, Takeda, Pfizer
- **Speaker Honoraria**: Amgen, AstraZeneca, Bayer, BMS, Boehringer Ingelheim, F. Hoffmann-La Roche, Janssen, MSD, Novartis, Pfizer, Takeda, Merck, Amgen, Pfizer

Institutional financial interests

- **Clinical Trials:** Amgen, AstraZeneca, Bayer, Boehringer Ingelheim, BMS, DaiichiSankyo, F. Hoffmann-La Roche, GSK, Janssen, Lilly, Merck, Mirati Therapeutics, MSD, Novartis, Amgen, Pfizer
- **Research Grant**: BMS, F. Merck, Pfizer

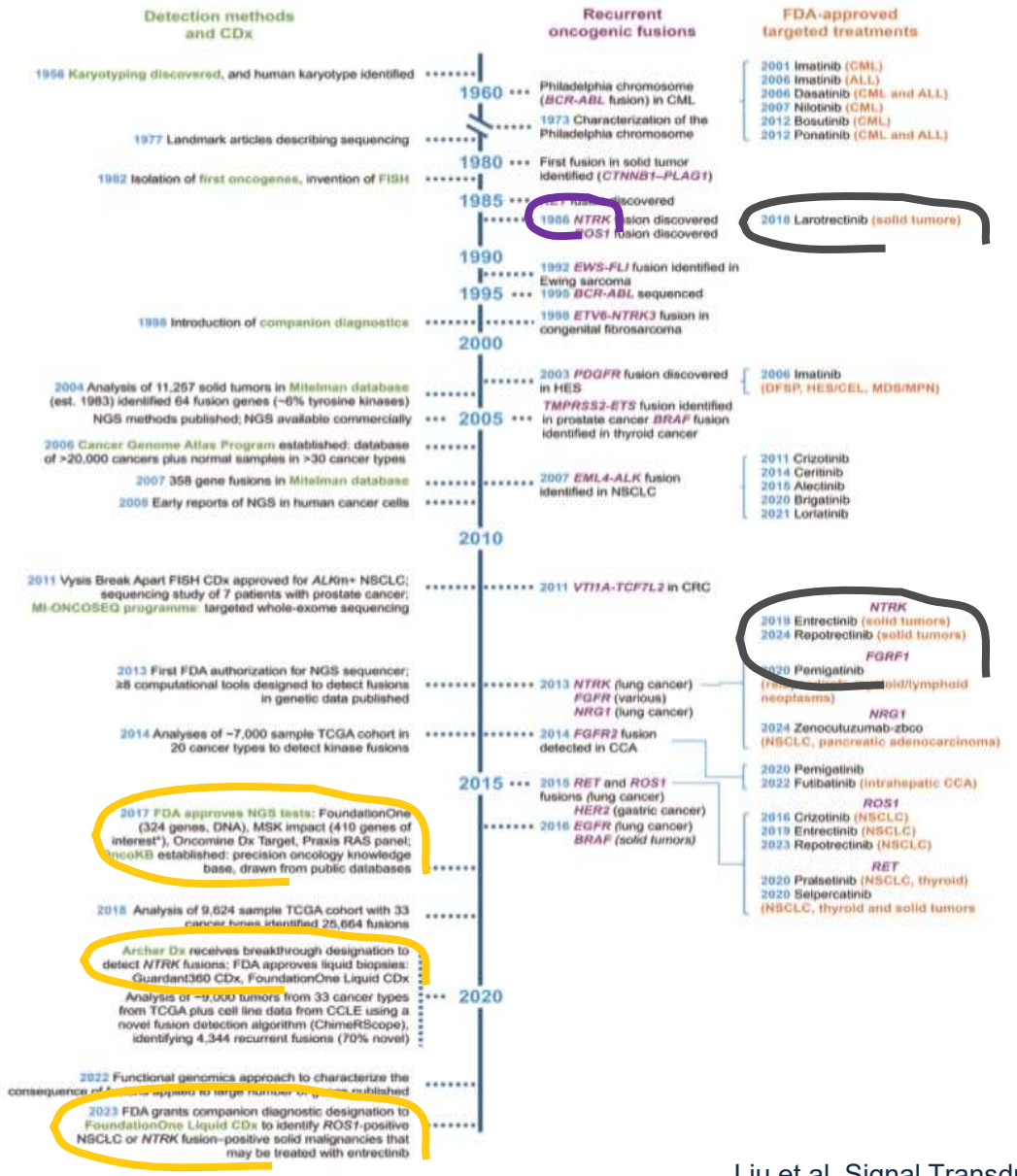
ONCOGENIC FUSIONS…RARE

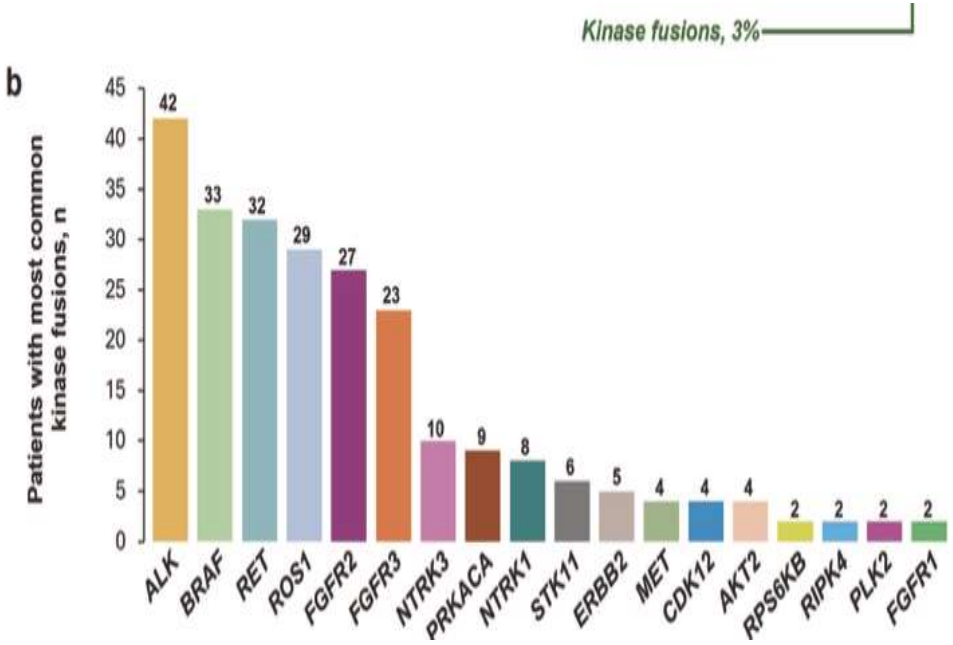
SPAIN… the corner or Europe…A CORUÑA..the corner of Spain…FINISTERRAE…the end of the world

We gather today to explore what is often considered rare, remote, and elusive disease…much like the ancient land of Finisterrae (named by the Roman), once believed to be the end of the known world

The journey to the edge is never easy, but it is always worthwhile

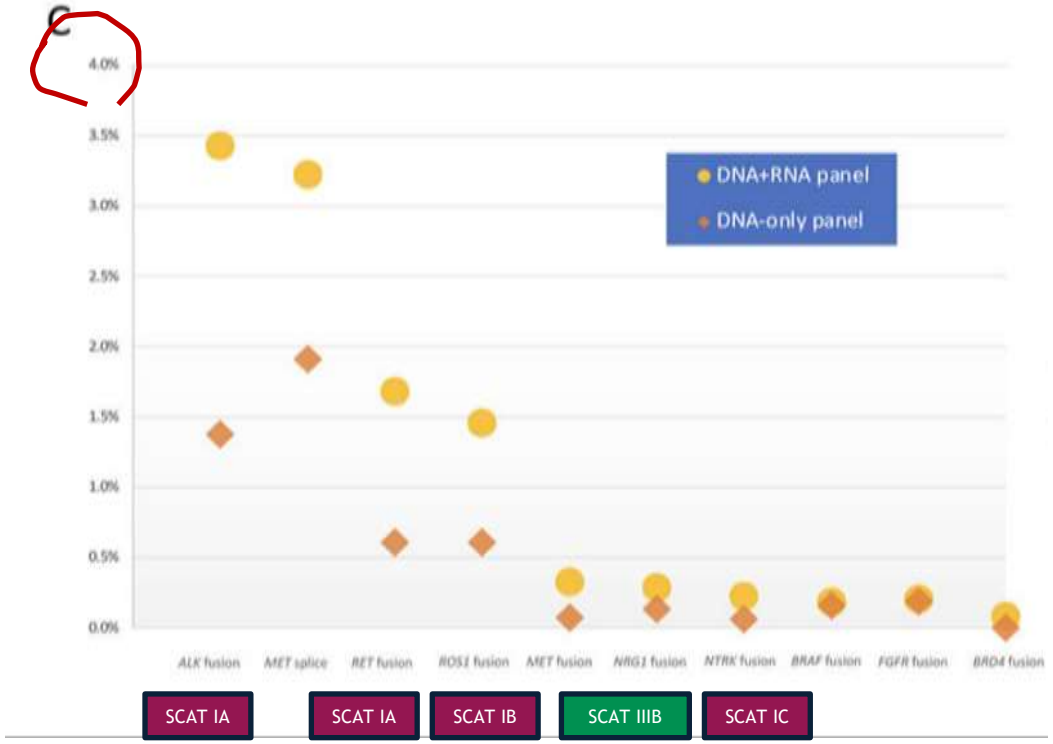
HISTORY AND MILESTONE EVENTS IN PROTEIN FUSIONS



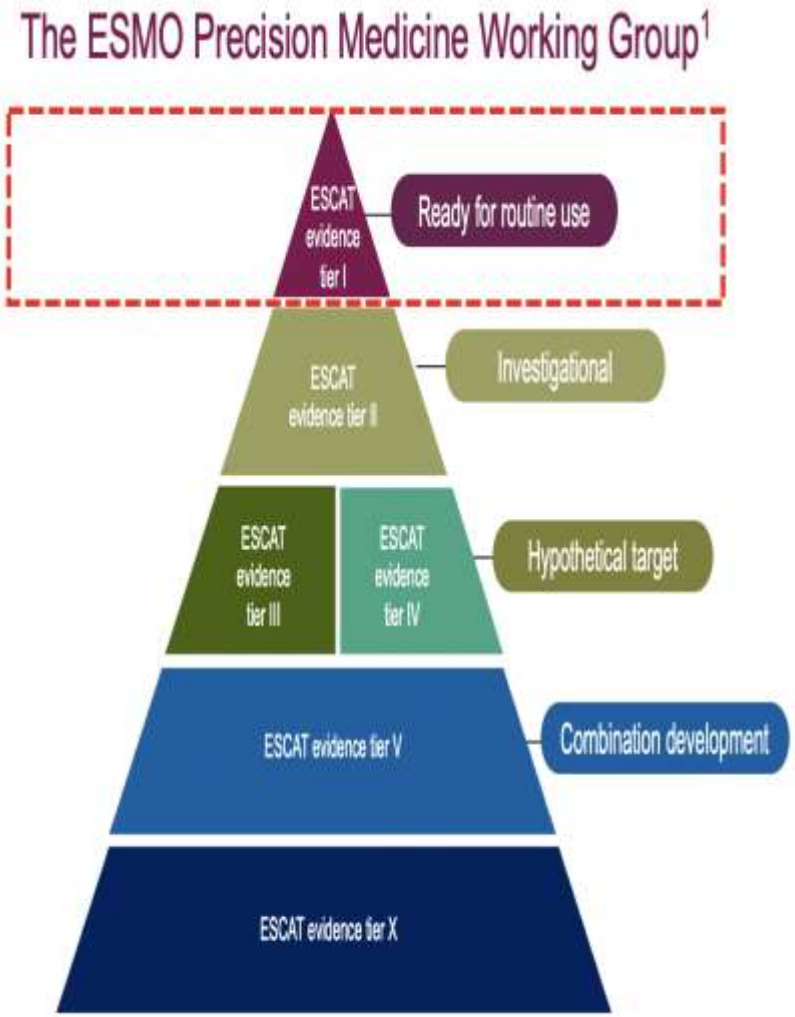


Liu et al. Signal Transduct Targeted Ther 2025

New treatment paradigm in NSCLC 2025



RNA-based NGS is preferred for identifying an expanding range of fusion genes Whichever testing modality is used, it is mandatory that adequate internal validation and quality control measures are in place and that laboratories participate in, and perform adequately in, external quality assurance schemes for each biomarker test	III, B III, A
Detection of the *ALK* translocation by FISH remains a standard, but IHC with high-performance ALK antibodies and validated assays may be used for screening and have been accepted as an equivalent alternative to FISH for ALK testing	III, A
Testing for *ROS1* rearrangements should be carried out Detection of the *ROS1* translocation by FISH remains a standard; IHC may be used as a screening approach	II, A N.A.
Testing for *NTRK* rearrangements should be carried out. Screening for *NTRK* rearrangements may use IHC or NGS, with appropriate testing follow-up to validate a positive result	II, A

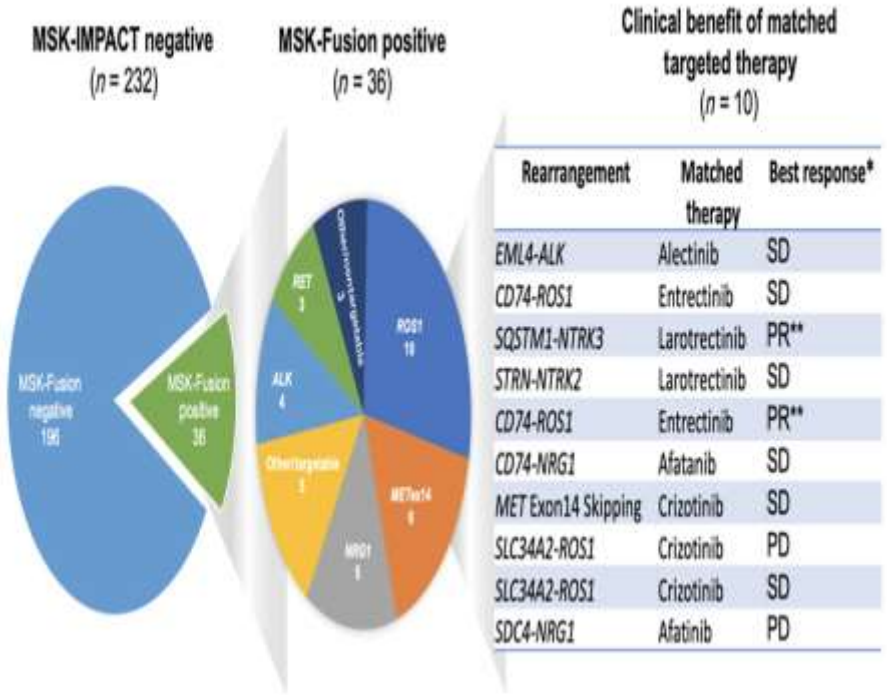


Gene	Alteration	ESCAT
ESCAT TIER EVIDENCE I		
EGFR	Common mutations (Del19, L858R)	IA
	Acquired T790M exon 20	IA
	Uncommon (G719X exon 18, L861Q exon 21, S768I exon 20)	IB
ALK	Fusions (mutations as mechanism of resistance)	IA
MET	Mutations ex 14 skipping	IB
BRAF^{V600}	Mutations	IB
ROS1	Fusions (mutations as mechanism of resistance)	IB
NTRK	Fusions	IC
RET	Fusions	IA
ESCAT TIER EVIDENCE II-III		
KRAS^{G12C}	Mutations	IIB
EGFR	Exon 20 insertion	IIB
ERBB2	Hotspot mutations and Amplifications	IIB
MET	Focal amplifications (acquired resistance on EGFR TKI)	IIB
BRCA 1/2	Mutations	IIIA
PIK3CA	Hotspot mutations	IIIA
NRG1	Fusions	IIIB

Harada et al, NRCO 2023; Mosele et al, Ann Oncol 2020; Hendriks et al, ESMO Living Guidelines 2025

FUSIONS ARE BEST DETECTED WITH RNA SEQ

RNA –seq may rescue 14% fusions: 92% actionable



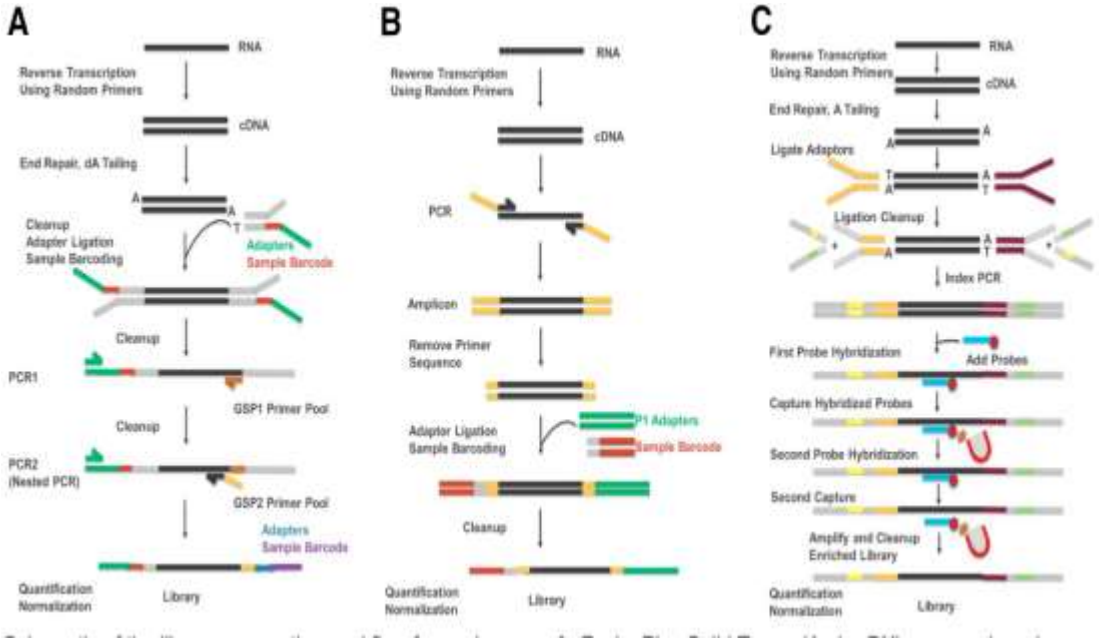
Clinical benefit of matched targeted therapy
(*n* = 10)

Rearrangement	Matched therapy	Best response*
EML4-ALK	Alectinib	SD
CD74-ROS1	Entrectinib	SD
SQSTM1-NTRK3	Larotrectinib	PR**
STRN-NTRK2	Larotrectinib	SD
CD74-ROS1	Entrectinib	PR**
CD74-NRG1	Afatanib	SD
MET Exon14 Skipping	Crizotinib	SD
SLC34A2-ROS1	Crizotinib	PD
SLC34A2-ROS1	Crizotinib	SD
SDC4-NRG1	Afatinib	PD

FusionPlex Solid Tumor (ArcherDX)

Oncomine Comprehensive Assay v3 (Thermo Fisher Scientific)

TruSight Oncology 500 (Illumina)



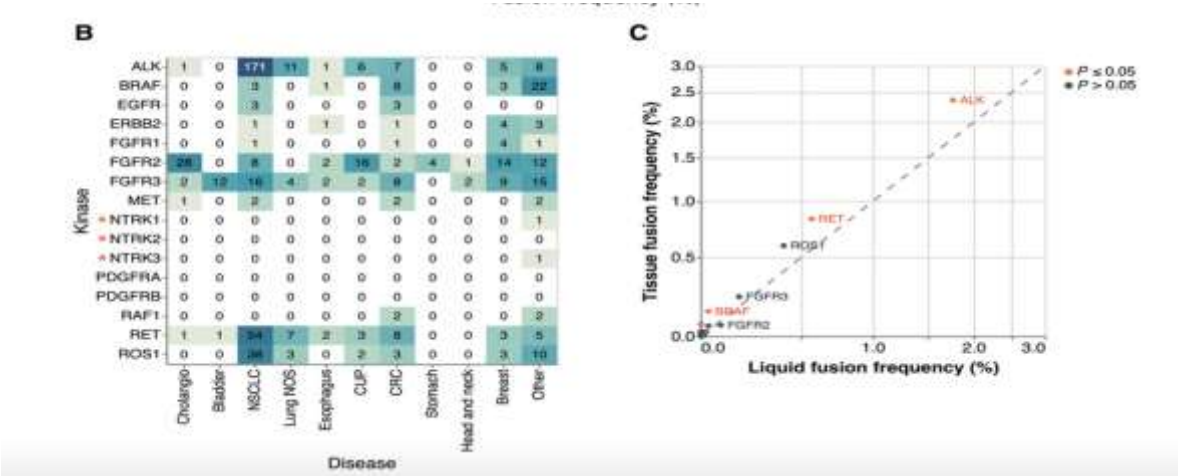
Anchored multiplex PCR, amplicon-based multiplex PCR, and hybrid capture based enrichment method: Highly concordant sensititvity and high-level performance in specificty

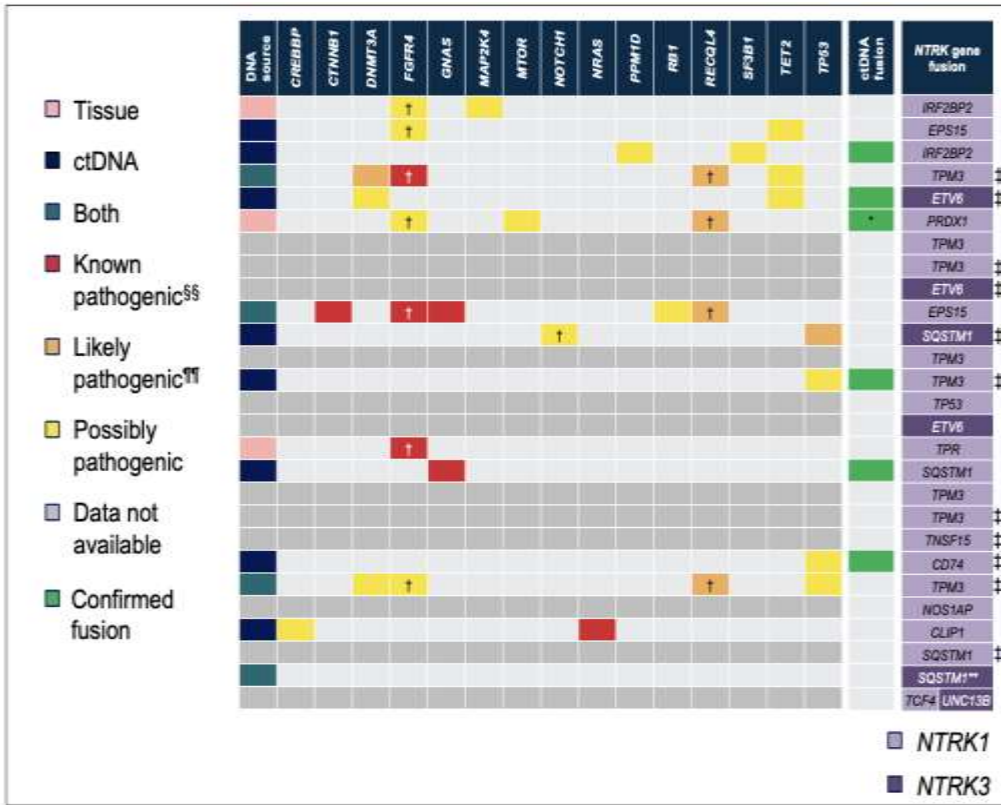
Benayed et al. Clin Cancer Res 2019; Park et al. J Mol Diagn 2021

The Pan-Tumor Landscape of Targetable Kinase Fusions in Circulating Tumor DNA

Table 1. Clinico-genomic characteristics of the overall cohort and of select cancer types.

	All ctDNA cases	NSCLC	Breast	CRC	CUP	Cholangiocarcinoma	Other cancer types
Total cases	36,916	10,754	5,148	2,742	1,918	901	15,453
Median age (y)	68	70	64	63	69	67	69
Gender (M:F)	50%:50%	47%:53%	0.84%:99%	57%:43%	50%:50%	50%:50%	66%:34%
Median ctDNA fraction	1.6%	1.5%	2.2%	3.3%	1.6%	0.96%	1.5%
Cases with ctDNA fraction > 0 (%)	32,492 (88%)	9,604 (89%)	4,617 (90%)	2,471 (90%)	1,667 (87%)	767 (85%)	13,366 (86%)
Cases with ≥1 GA (%)	30,348 (93%)	8,907 (93%)	4,413 (96%)	2,388 (97%)	1,569 (94%)	702 (92%)	12,369 (93%)
Avg GAs/case	3.3	3.0	3.8	4.2	3.1	2.9	3.2
Fusion-positive cases	571 (1.8%)	296 (3.1%)	44 (0.95%)	37 (1.5%)	29 (1.7%)	32 (4.2%)	133 (1.0%)





Targetable kinase fusions are identified in ctDNA across cancer types. In pairs with tissue-identified fusions, fusion detection in ctDNA is reliable with elevated ctDNA fraction.

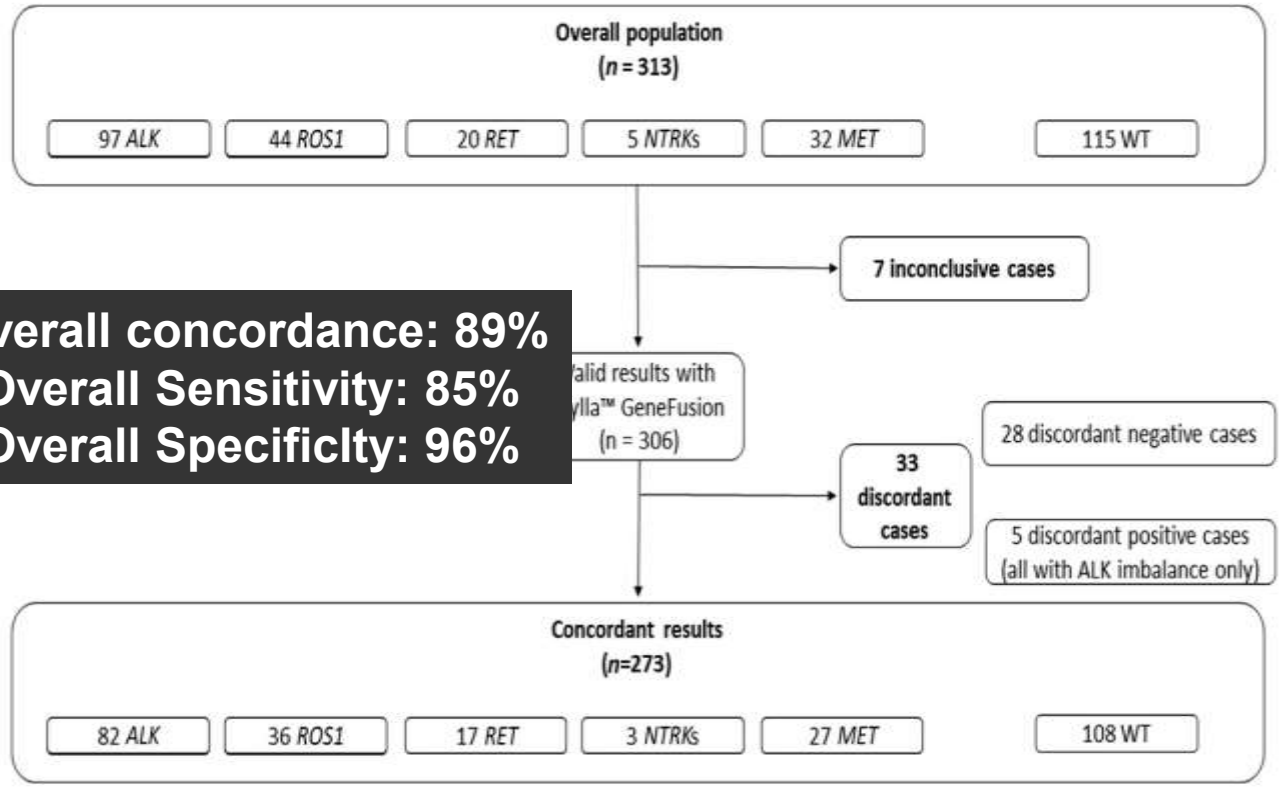
ctDNA data were available for 14 patients out of 27. ctDNA analysis detected *NTRK* gene fusions in 6 of the 14 patients (43%) at the start of treatment.

Lee J, et al. Clin Cancer Res 2022; Drilon et al, ELCC 2024

Idylla GeneFusion in NSCLC

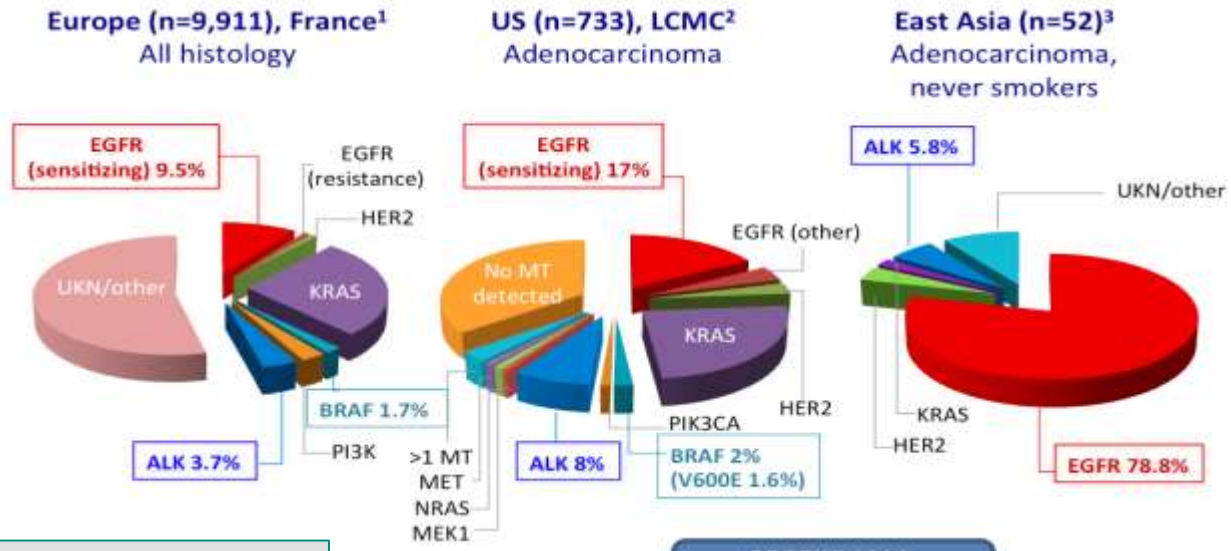
- Retrospective study evaluating the Idylla GeneFusion prototype, an automated test, with a short turnaround time (3 hours) to detect fusions (ALK, ROS1, RET, and NTRK1/2/3 genes and MET exon 14 skipping)
- 313 tissue samples from NSCLC lung cancer patients, previously identified with reference methods (RNA-based NGS/ FISH/ qPCR).

Overall concordance: 89%
Overall Sensitivity: 85%
Overall Specificlty: 96%

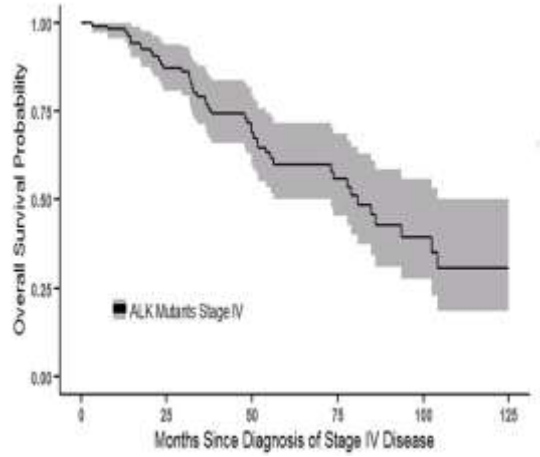


Depoilly et al. J Mol Diagn 2022

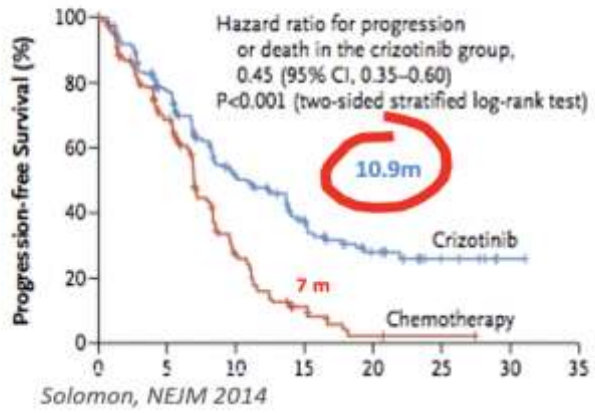
ALK
How the history begins…

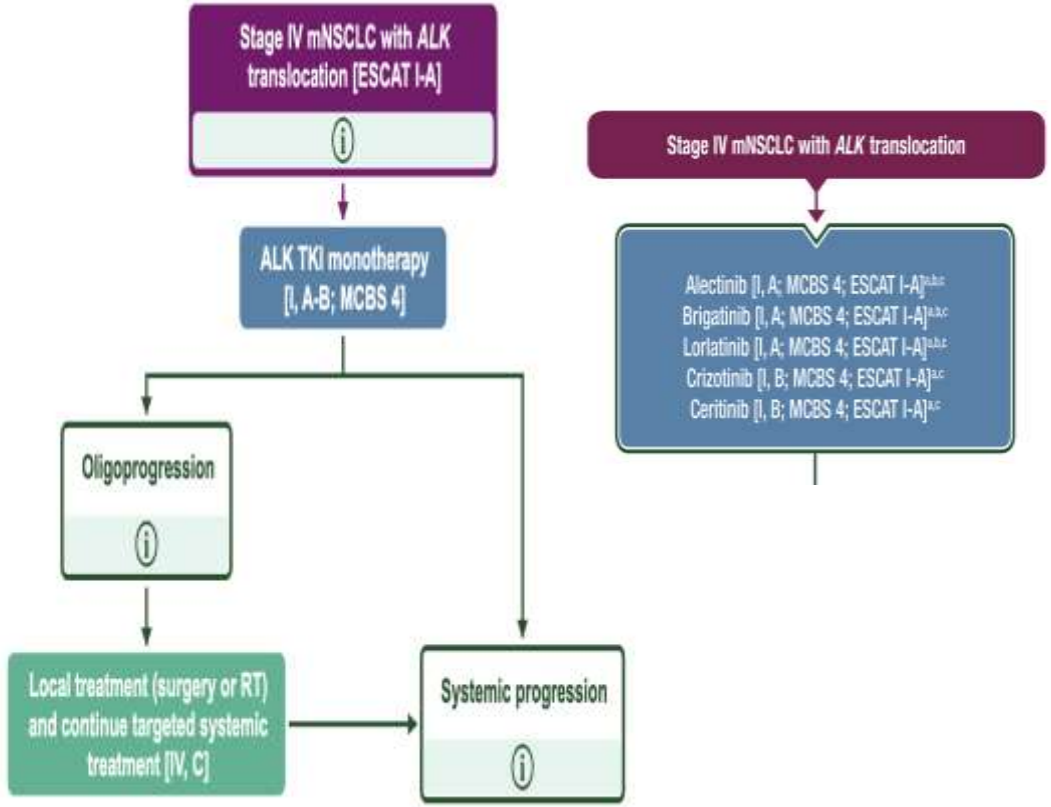


mOS: 81 months



PROFILE 1014: Crizotinib vs Chemo

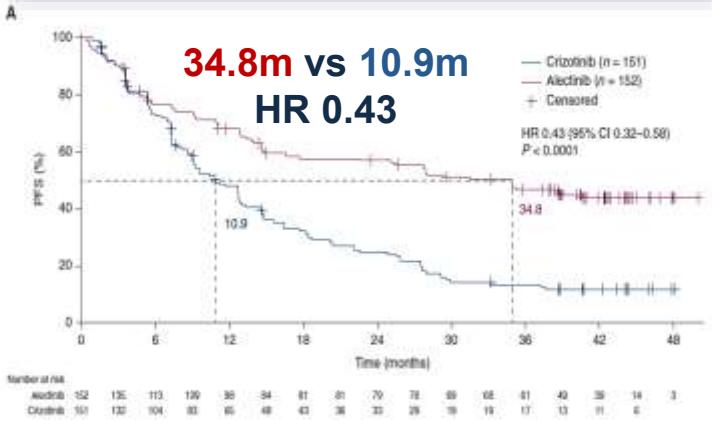




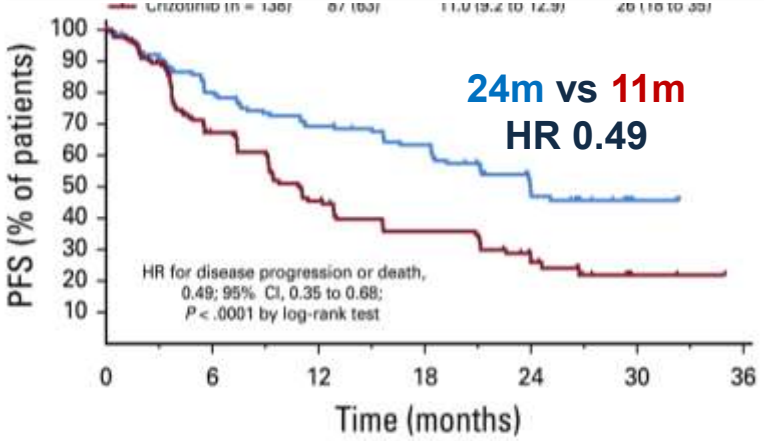
Barlesi et al ASCO 2013; Johnson et al; ASCO 2013; Sun JCO 2010; Pacheco et al, JTO 2018; Hendriks et al, ESMO Living Guidelines 2025

TOO MANY 2ND GENERATION TKI…THEY WORK FOR SURE COMPARED TO CRIZOTINIB

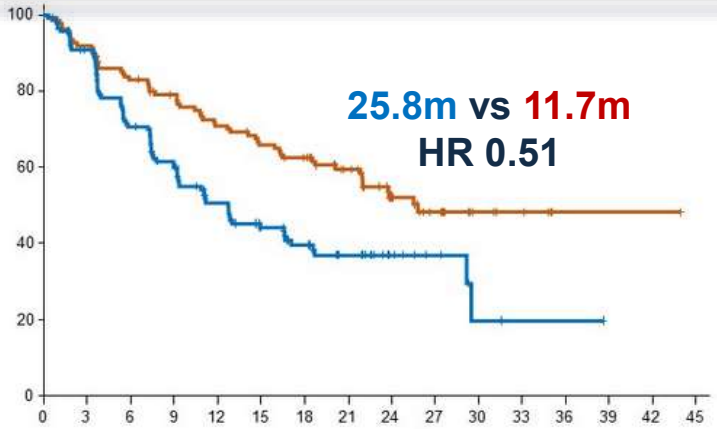
ALEX: Alectinib *vs*. Crizotinib



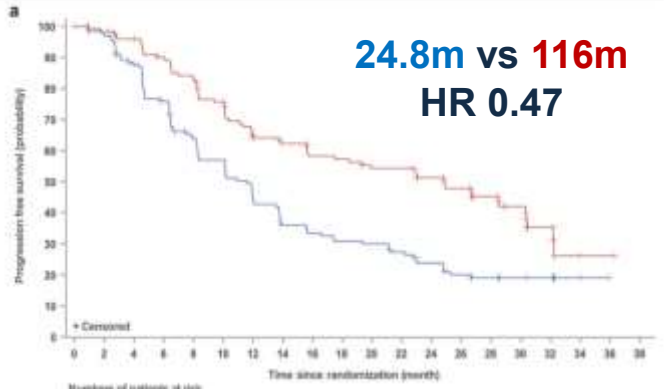
ALTA 1L: Brigatinib *vs*. Crizotinib



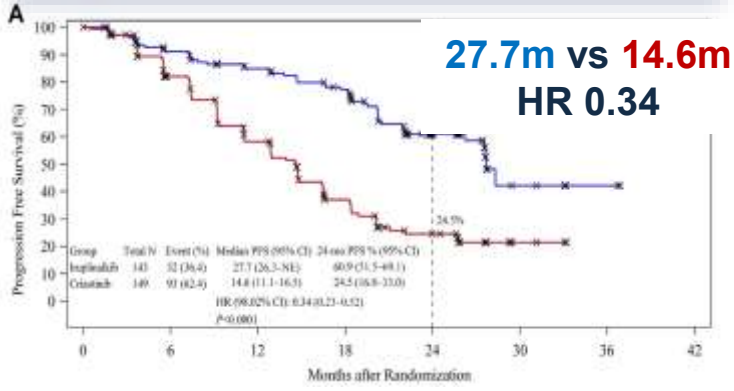
eXalt 3: Ensartinib *vs*. Crizotinib



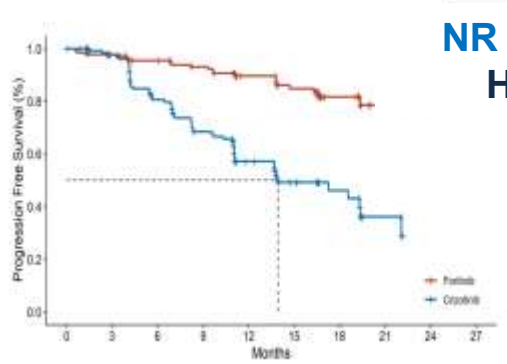
NCT04009317: Envonalkib *vs*. Crizotinib



INSPIRE: Iruplinalkib *vs*. Crizotinib



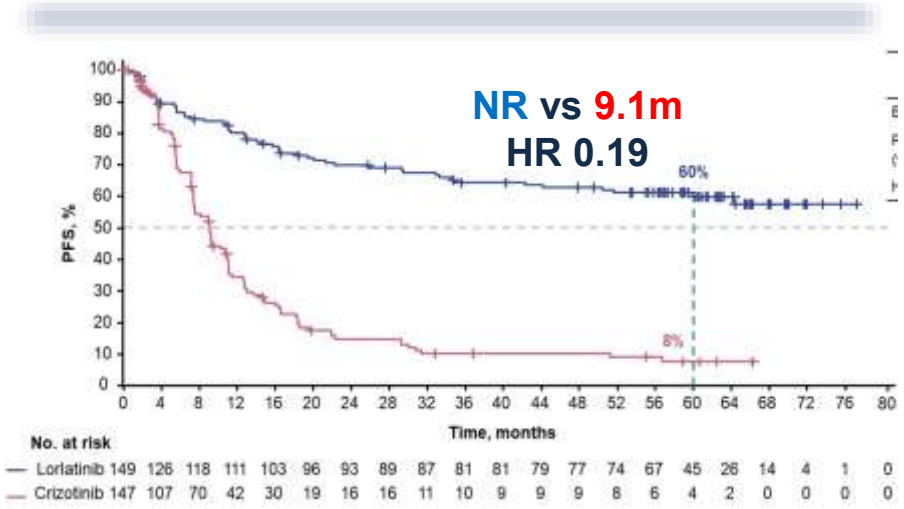
REMARK: Foritinib *vs*. Crizotinib



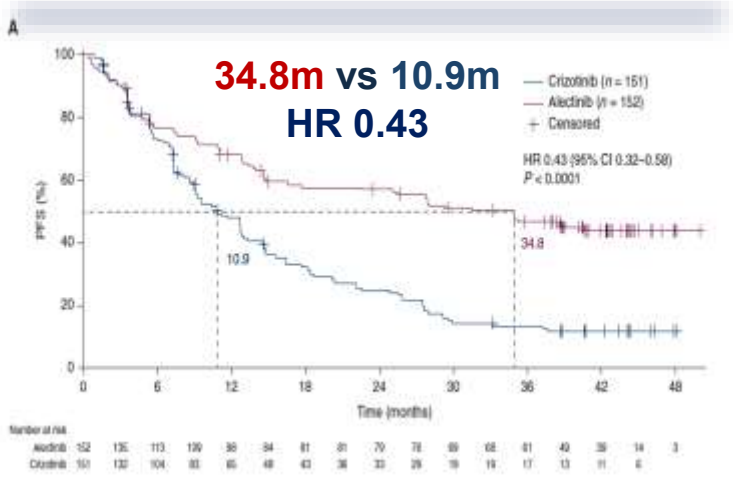
Mok Ann Oncol 2020; Camidge J Clin Oncol 2022; Horn et al. JAMA Oncol 2021; Yang Y et al Signal Transduct Target Ther 2023; Shi Y et al J Thorac Oncol 2024; Xiong A, et al. WCLC 2024

Next generation upfront…no many doubts

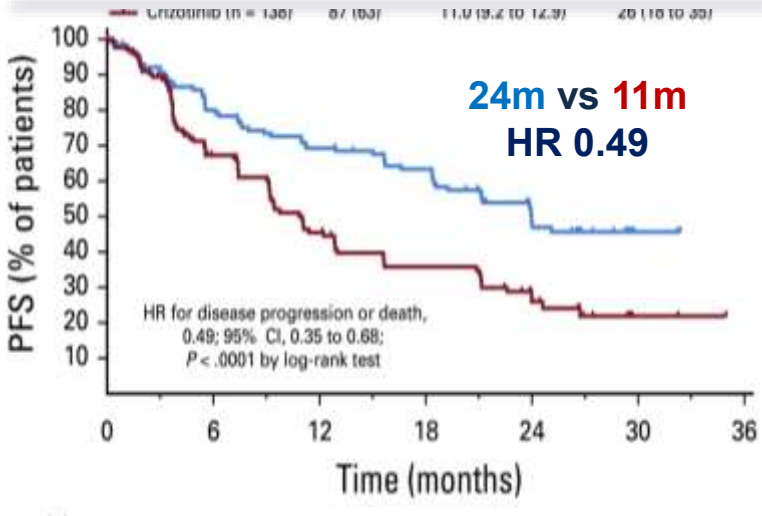
CROWN: Lorlatinib *vs*. crizotinib

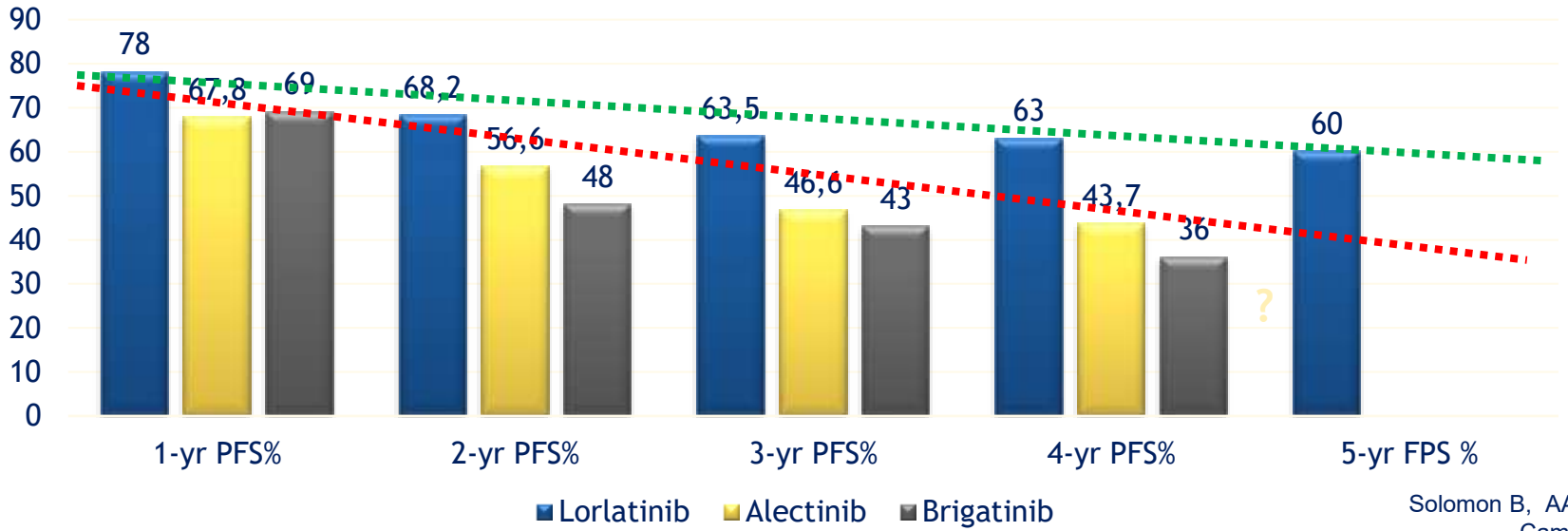


ALEX: Alectinib *vs*. Crizotinib



ALTA 1L: Brigatinib *vs*. Crizotinib



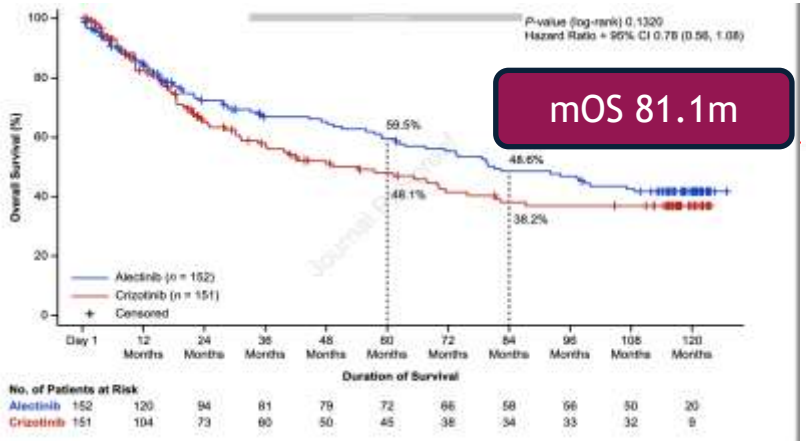


Solomon B, AACR 2022; Mok Ann Oncol 2020; Camidge J Clin Oncol 2022

How Relevant Is OS?

ALEX TRIAL

Alectinib vs Crizotinib

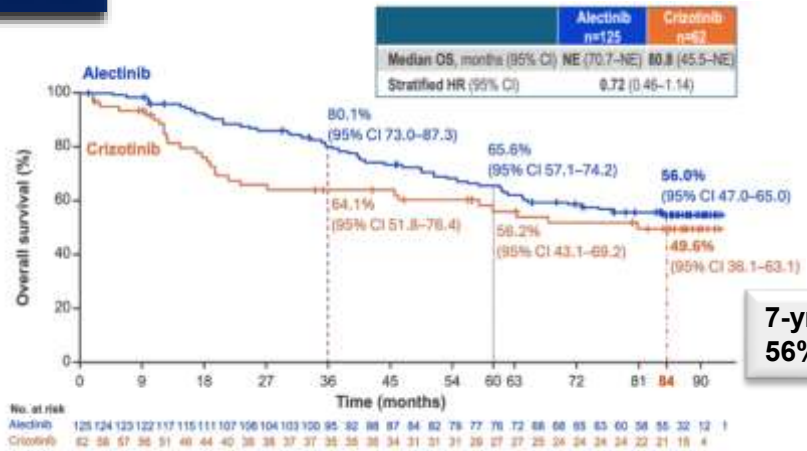


mOS 81.1m

5-yr OS: 62% vs 45%
7y OS: 48% vs 38% HR.78; p0.13

ALESIA TRIAL

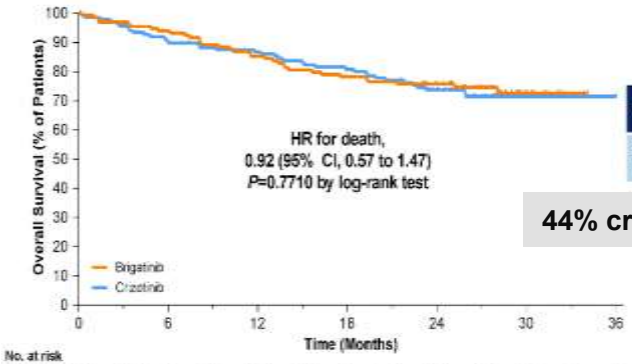
Alectinib vs Crizotinib



7-yr OS
56% vs 49%

ALTA1L TRIAL

Brigatinib vs Crizotinib



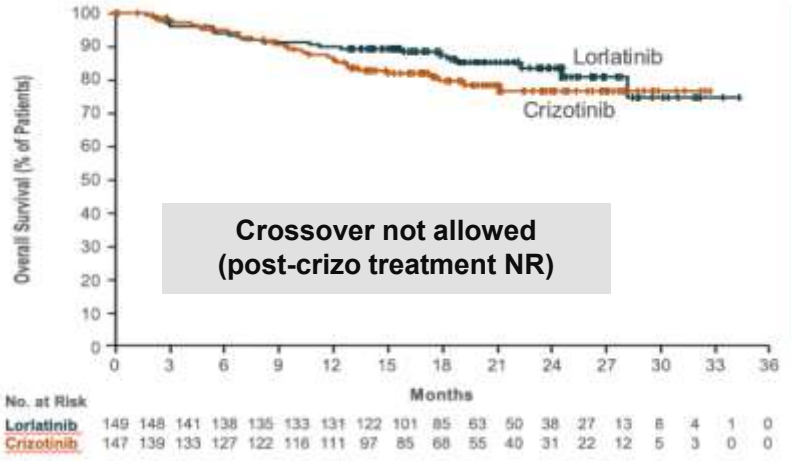
Treatment	Deaths, n (%)	2-year OS (95% CI), %
Brigatinib (n=137)	33 (24)	76 (67–82)
		74 (65–80)

44% crossover

• 44% of patients in the crizotinib arm crossed over to brigatinib therapy

CROWN TRIAL

Lorlatinib vs Crizotinib



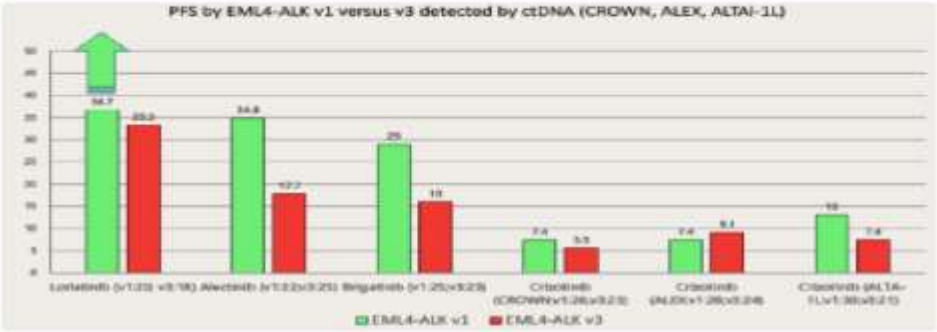
Crossover not allowed
(post-crizo treatment NR)

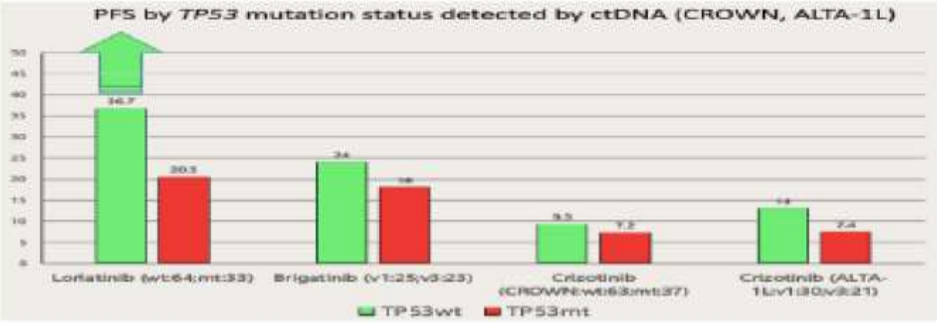
	Lorlatinib (n=149)	Crizotinib (n=147)
Patients with event, n (%)	23 (15)	28 (19)
Median OS, months (95% CI)	NE (NE–NE)	NE (NE–NE)
HR (95% CI)	0.72 (0.41–1.26)	

Mok T, et al ESMO 2025;Abstract 9518; Camidge R, et al. ESMO ASIA 2019; Solomon B, et al. ESMO 2020. Abstract LBA2; Zhou et al. ESMO ASIA 2024

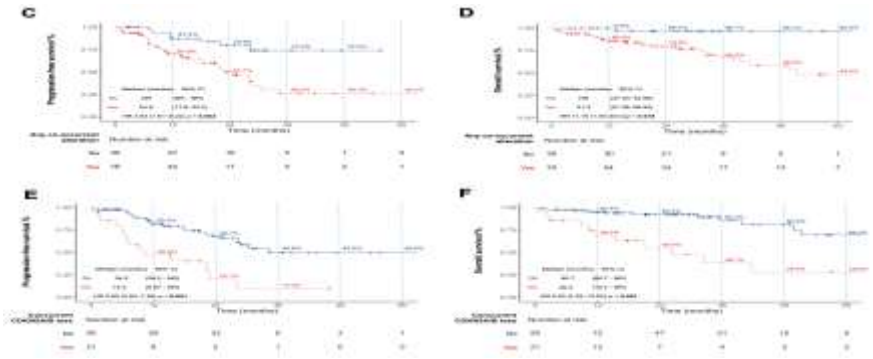
THE TYPE OF VARIANT MAY MATTERS…THE CONCOMITANT ALTERATIONS MAY ALSO IMPACT THE OUTCOME

PFS rates by ALK variant and TP-53 co-mutations

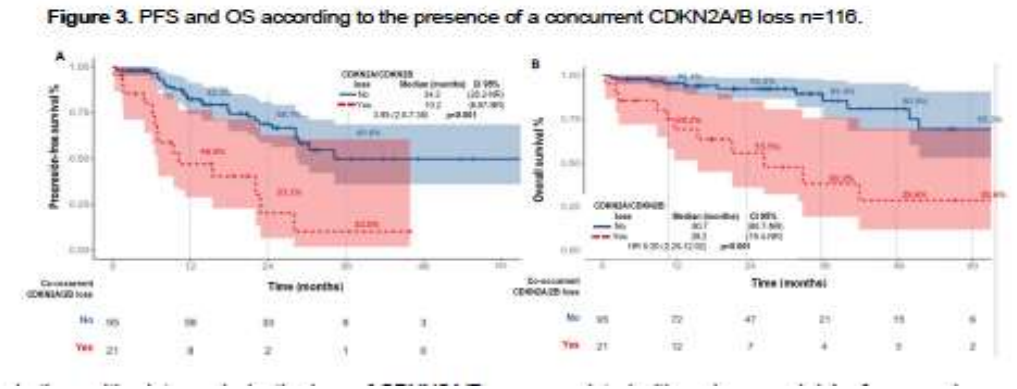




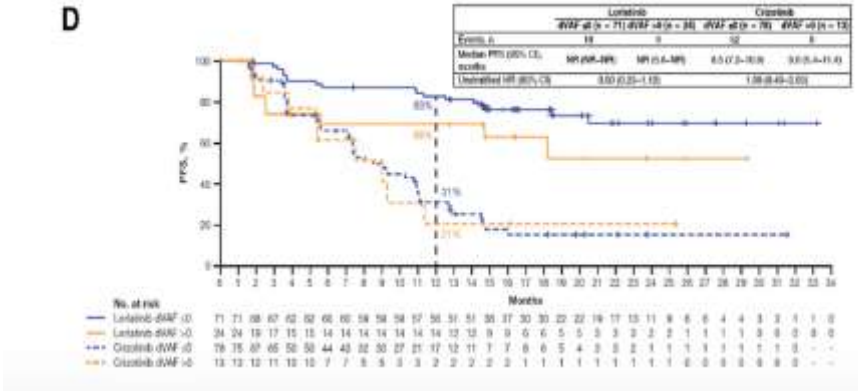
Not only mutations matters…VAF also important



CDKN2A/b Co-mutations may increase riks of TKI failure, BM and poor outome

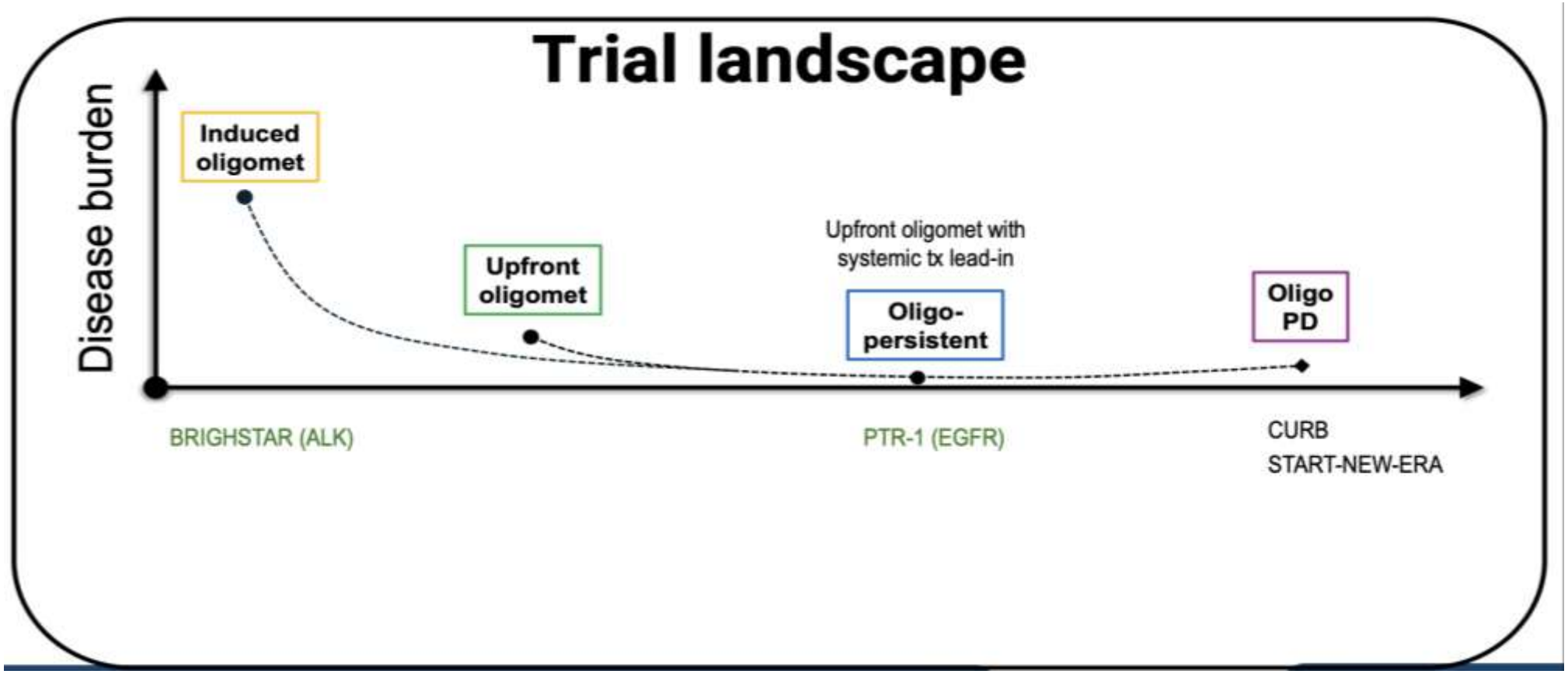


ctDNA clearance in CROWN related to outcomes



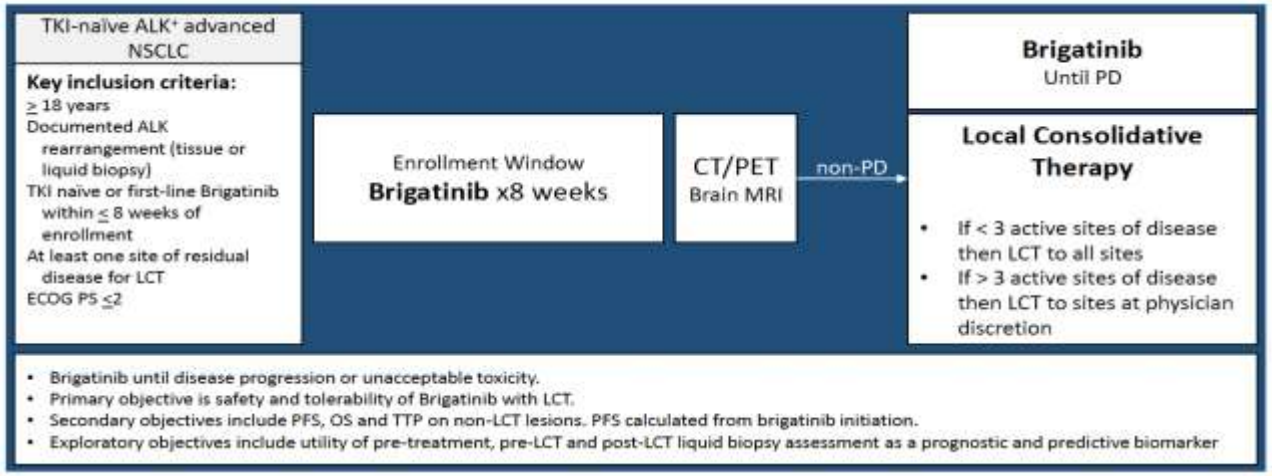
Ou S-H I, Lung Cancer Summit, Rome, 2023; Arrieta O, et al. JTO 2024; Soo R, et al. J Thorac Oncol 2023; Parikh et al. J Thorac Oncol 2024

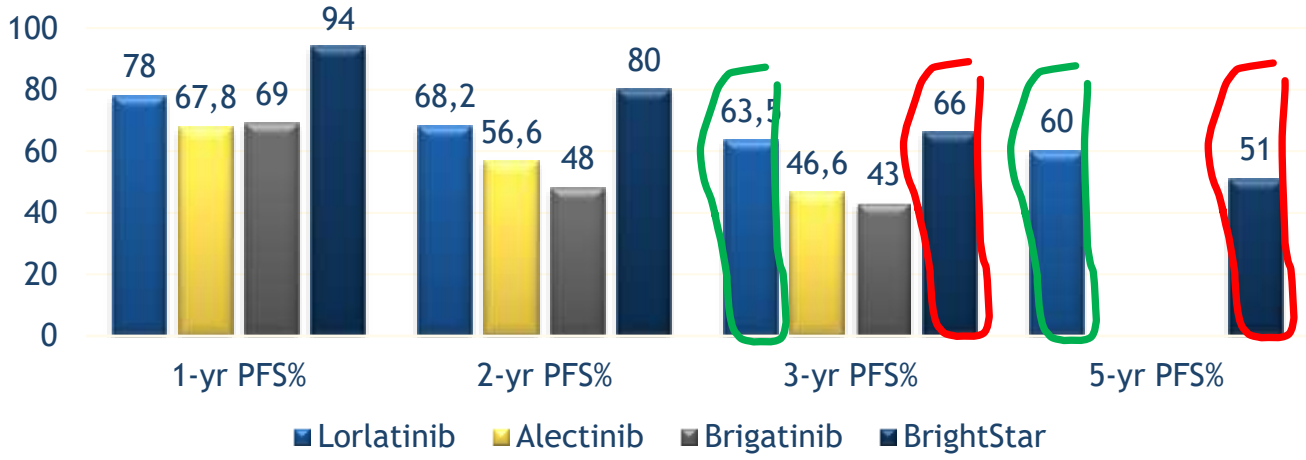
Integrating local therapies

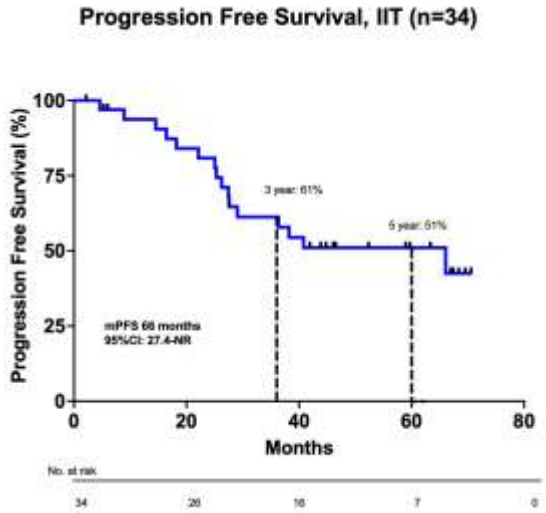


Lee, et al. WCLC 2024.

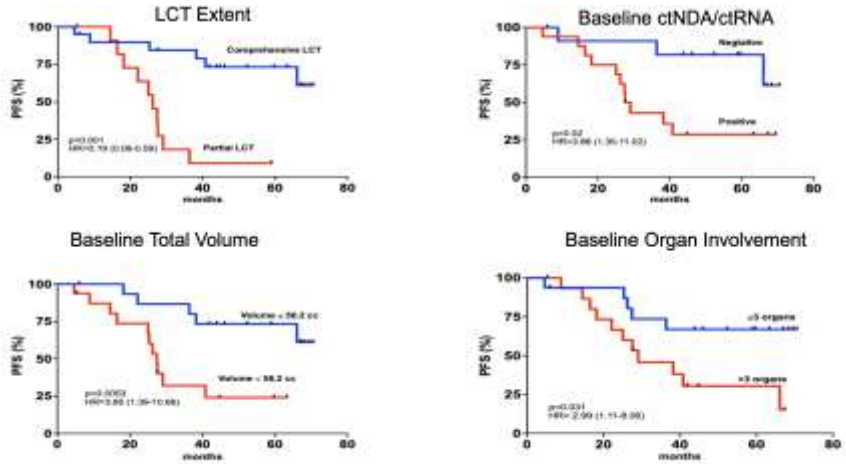
BRIGHTSTAR: a single arm phase II study of brigatinib+ LCT for ALK (oligo/poly) metastatic NSCLC







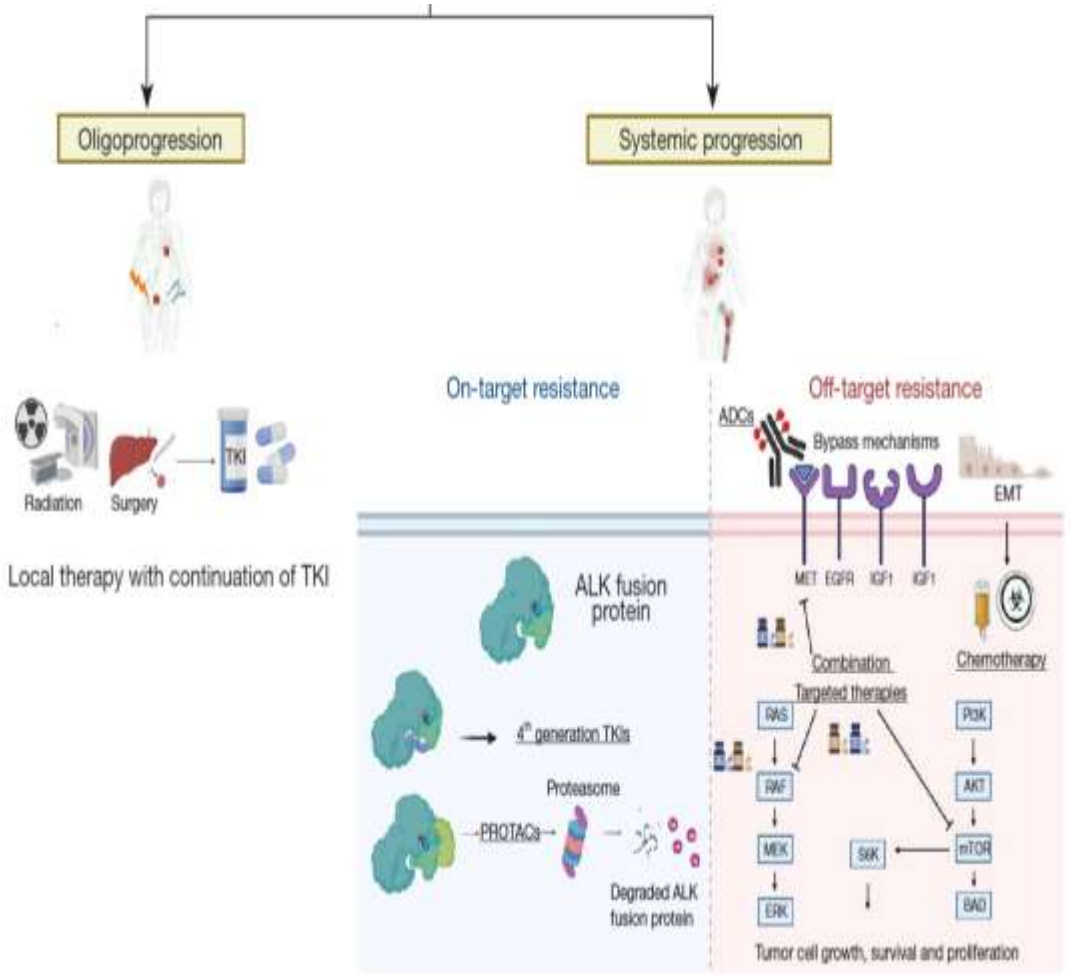
mPFS 66m



Elamin Y et al. WCLC 2025

Strategies to overcome resistance to ALK TKI

LOCAL ABLATIVE THERAPY + ALK INH.

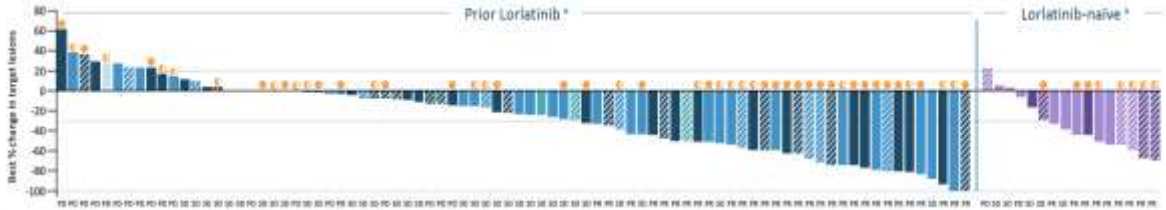


TRIAL/SITES	TREATMENT	N	NCT	YEARS
All Mets				
OPTALK (Phae II) (Groupo Francais De Pneumo-Cancerologie)	3-9 mos SD/PR to brigatinib → locally treat all remaining sites [PFS]	N=45	NCT06620835	2024-2030
A-SAB (Phase I-II) (Karolinska)	2-3 mos SD/PR to alectinib → locally treat all remaining sites [PFS]	N=70	NCT05724004	2023-2031
Bright-Star2 (RCT Phase II) (MDACC)	Brigatinib ,vs brigatinib + local consolidation therapy [PFS]	N=168	NCT06522360	2024-2031
COMLORLA (RWD) (Peking University)	Lorlatinib → any local treatment [TTD]	N=100	NCT06690541	2024-2030
Brain Mets				
DURABLE (Phase Ib-II) (USA/ Ohio/Stanford)	Delayed vs upfront brain SRS with alectinib for asymptomatic/min symp brain mets	N=56	NCT05987644	2024-2027

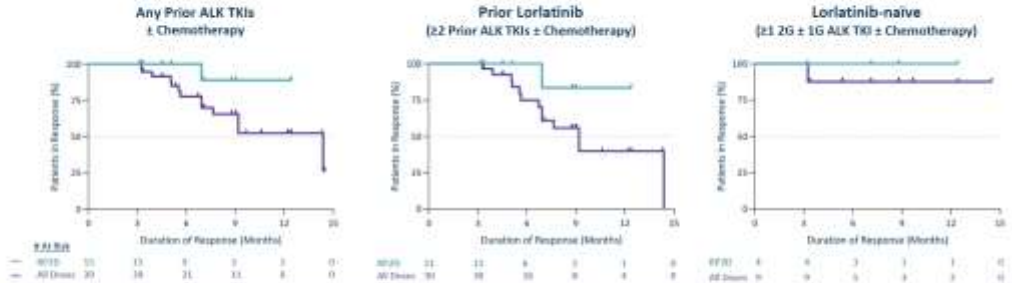
ALKove-1

Preliminary Activity: Radiographic Tumor Responses Across Previously Treated Patients with ALK+ NSCLC

RECIST 1.1 ORR, % (n/N) All patients ± chemotherapy	NSCLC Response-Evaluable (Any Prior ALK TKI, range 1 – 5)			Prior Lorlatinib (≥2 ALK TKIs)			Lorlatinib-naive (≥1 2G ± 1G)	
	All	Any ALK mutation	G1202R	All	Any ALK mutation	Compound ALK mutation	All	Any ALK mutation
All Doses	38% (39/103)	52% (30/58)	69% (22/32)	35% (30/85)	47% (23/49)	54% (15/28)	53% (9/17)	88% (7/8)
RP2D	38% (15/39)	55% (12/22)	71% (10/14)	35% (11/31)	50% (8/16)	64% (7/11)	57% (4/7)	80% (4/5)



Durable Tumor Response: Previously Treated Patients with ALK+ NSCLC

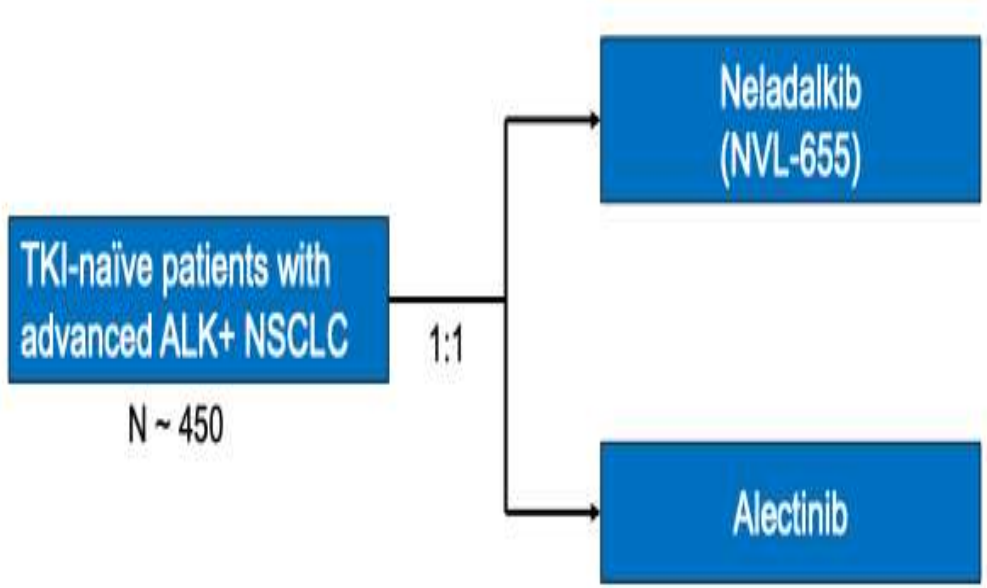


NSCLC Response-Evaluable	All Dose Levels	RP2D	All Dose Levels	RP2D	All Dose Levels	RP2D
Median DOR, m (95% CI)	14.4 (6.9, NE)	Not Reached (6.9, NE)	9.2 (6.9, NE)	Not Reached (6.9, NE)	Not Reached (3.3, NE)	Not Reached (NE, NE)
DOR ≥ 6 m (95% CI)	78% (58, 89)	100% (100, 100)	75% (52, 88)	100% (100, 100)	88% (39, 98)	100% (100, 100)

Drilon A, et al. ESMO 2024

GECP lung cancer research

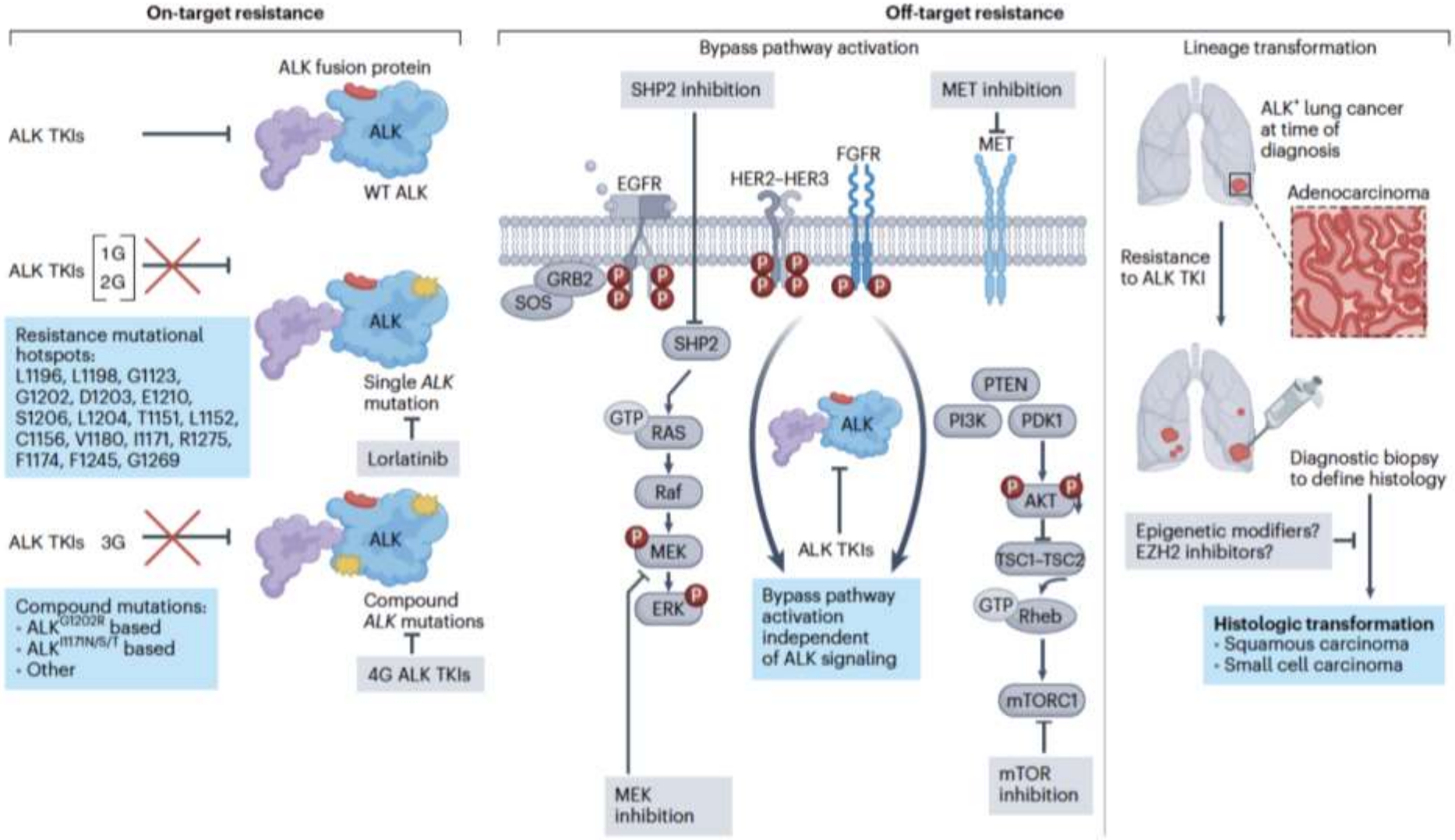
ALKAZAR PHASE III TRIAL



Primary endpoint: PFS per BICR
Secondary endpoints: PFS per investigator assessment, BICR-ORR, IC-ORR, OS, safety

Nuvalent press release

The real challenge: Understanding drug resistance

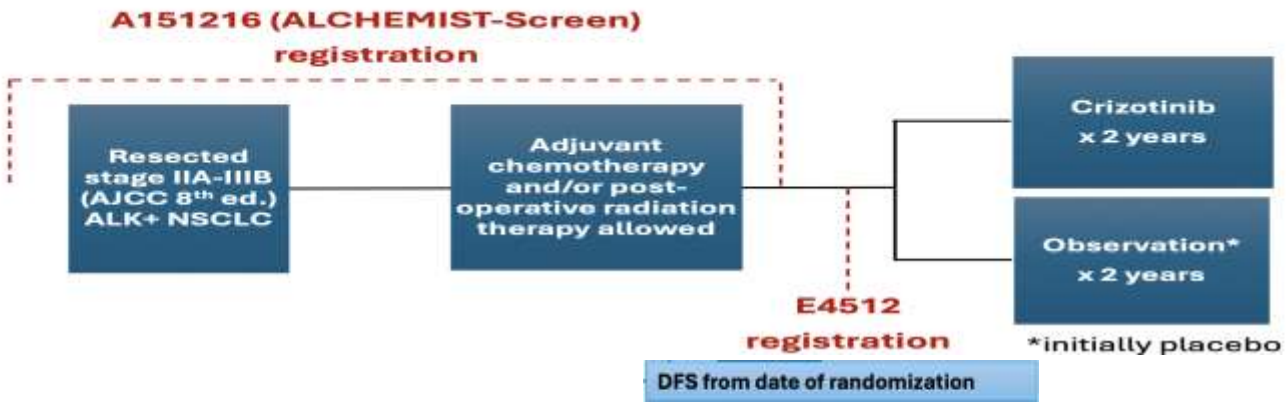


Schneider JL, et al. *Nat Cancer* 2023;4:330-343.

CAN WE INCREASE CURE RATE INTEGRATING THIS PERSONALIZED APPROACH IN EARLY STAGES?

ECOG-ACRIN 4512
Adjuvant Crizotinib..not an option

E4512 design and endpoints



Endpoints

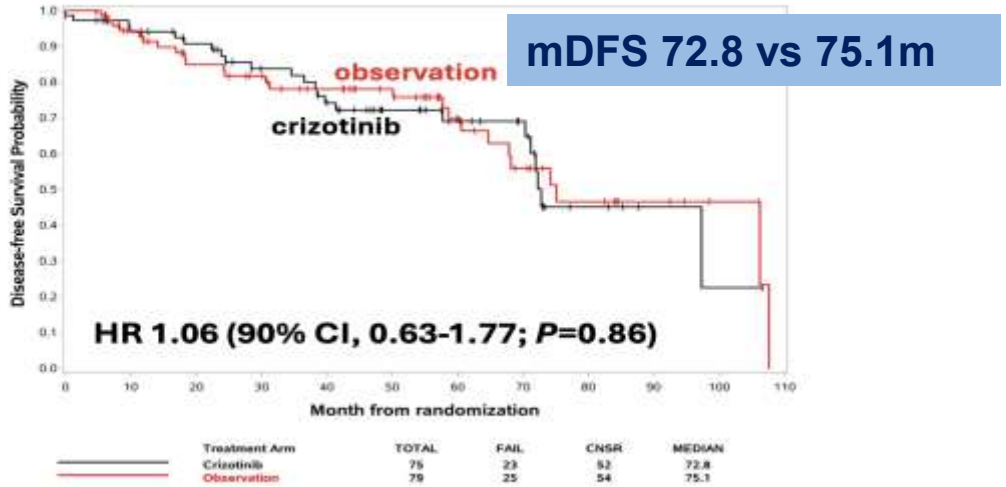
Primary

- DFS (in centrally ALK+ cases)

Secondary

- OS
- Toxicity

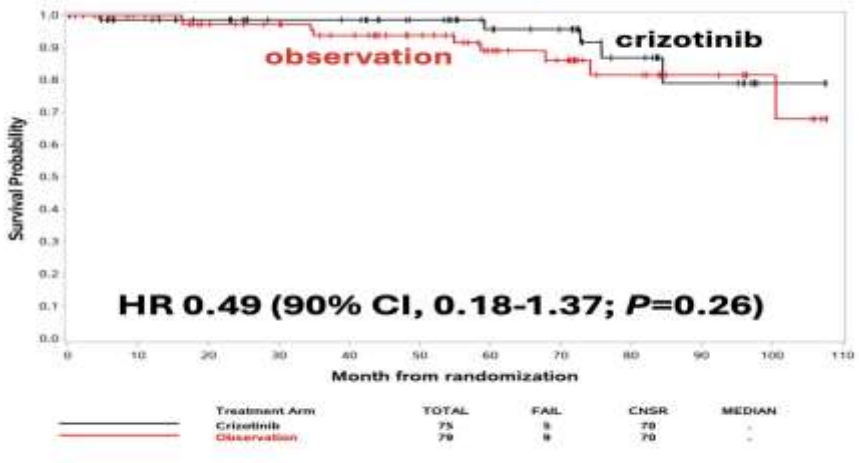
Disease-free survival



mDFS 72.8 vs 75.1m

Median follow-up = 58.3 months

Overall survival



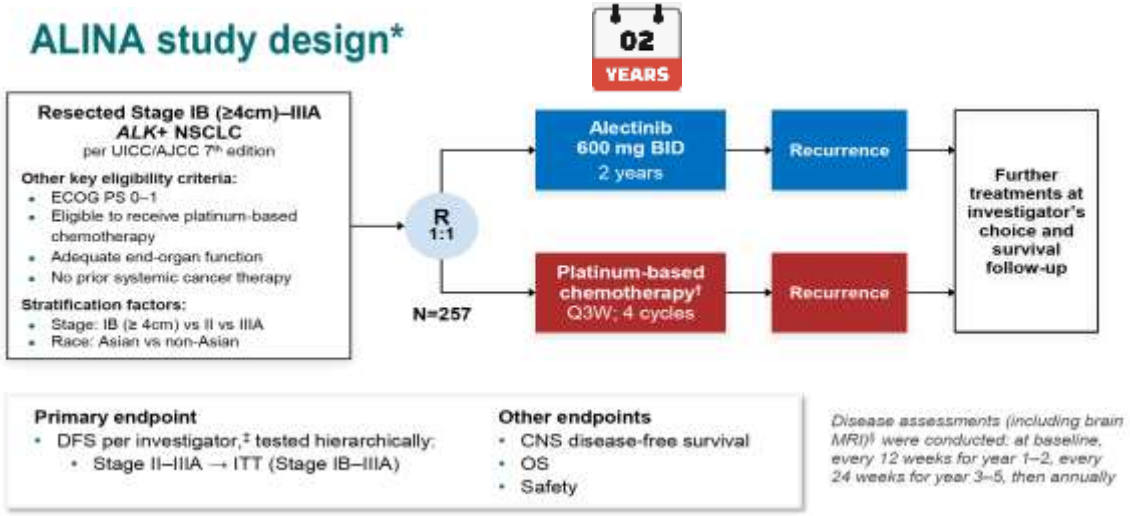
Median follow-up = 58.3 months

Gerber et al. WCLC 2025

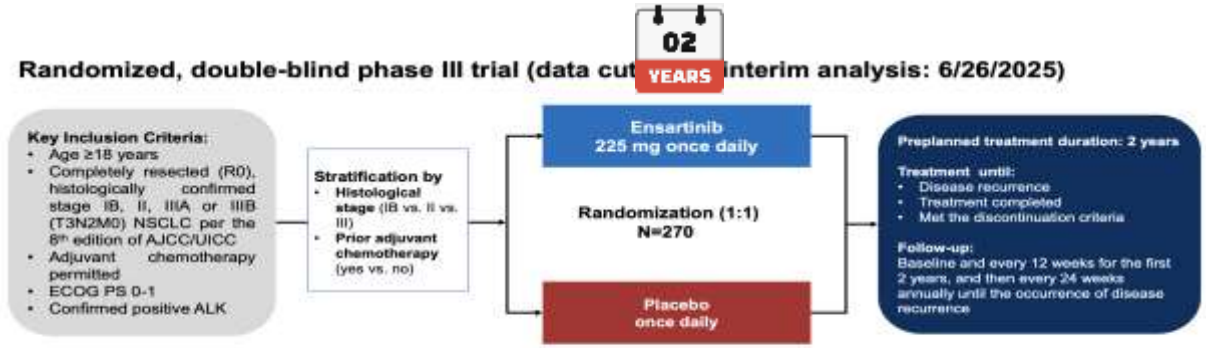
ALINA

ELEVATE

ALINA study design*



Randomized, double-blind phase III trial (data cut … interim analysis: 6/26/2025)



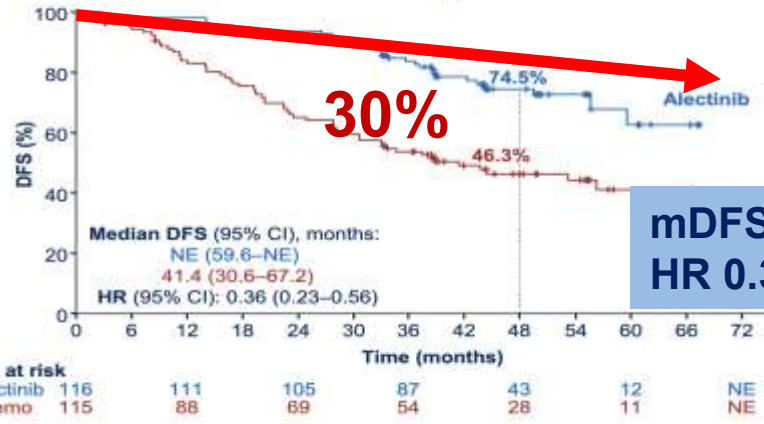
Primary endpoint: Investigator-assessed DFS* in patients with stage II to IIIB disease

Secondary endpoints: Investigator-assessed DFS in patients with stage IB-IIIB disease (ITT), 3/5-year DFS rate, OS, safety

Statistical analysis:

- This preplanned interim analysis was performed when 70% of events (57 events) were observed in patients with stage II-IIIB disease.

DFS in stage II–IIIA*

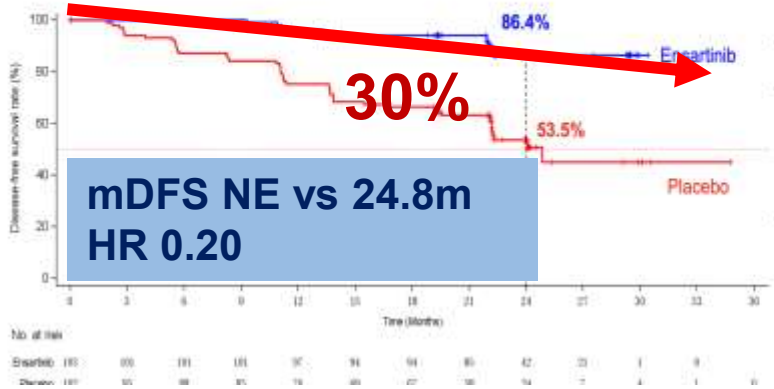


mDFS NR vs 41.4m
HR 0.36

76% reduction in risk of **disease recurrence** or **death** in **stage II / IIIA** population

Ensartinib showed an improved DFS in patients with II-IIIB disease

Investigator-assessed DFS

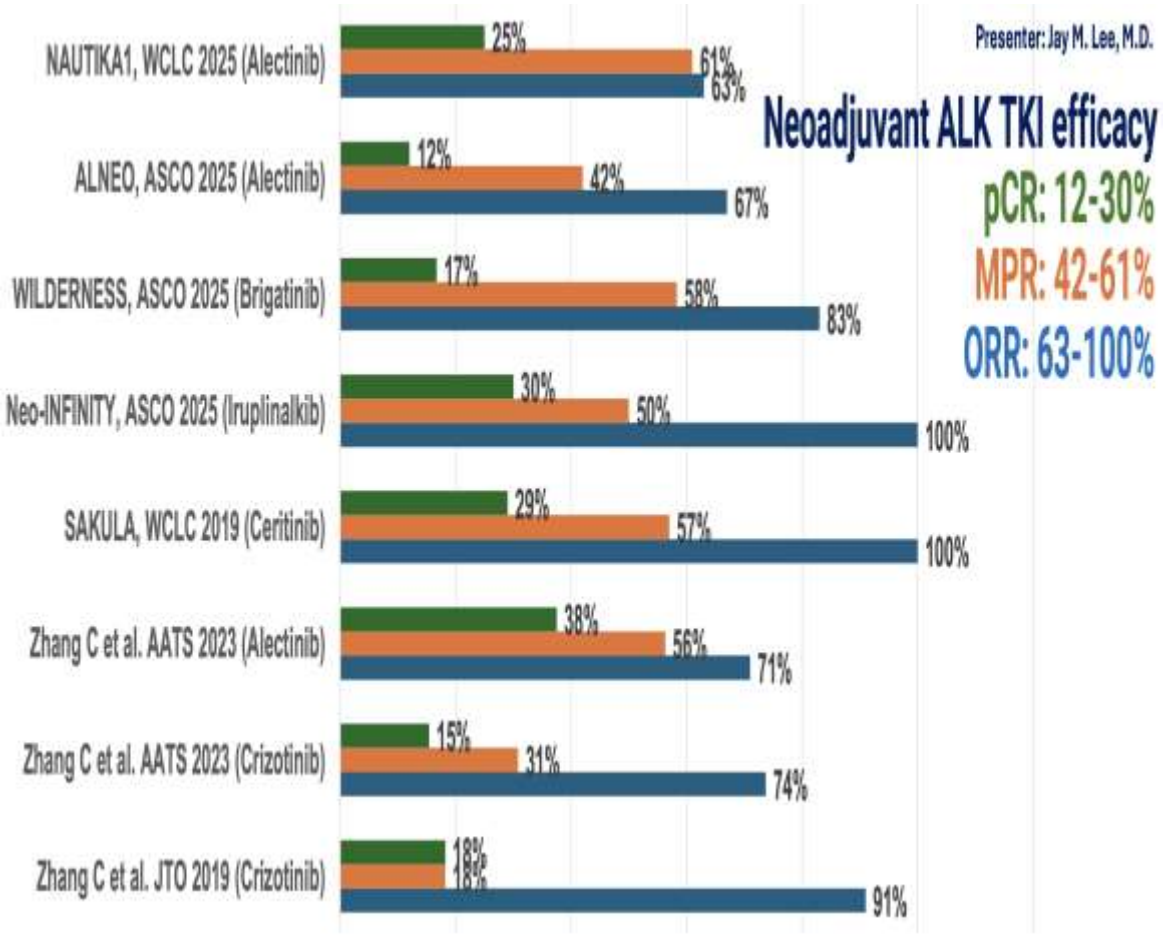


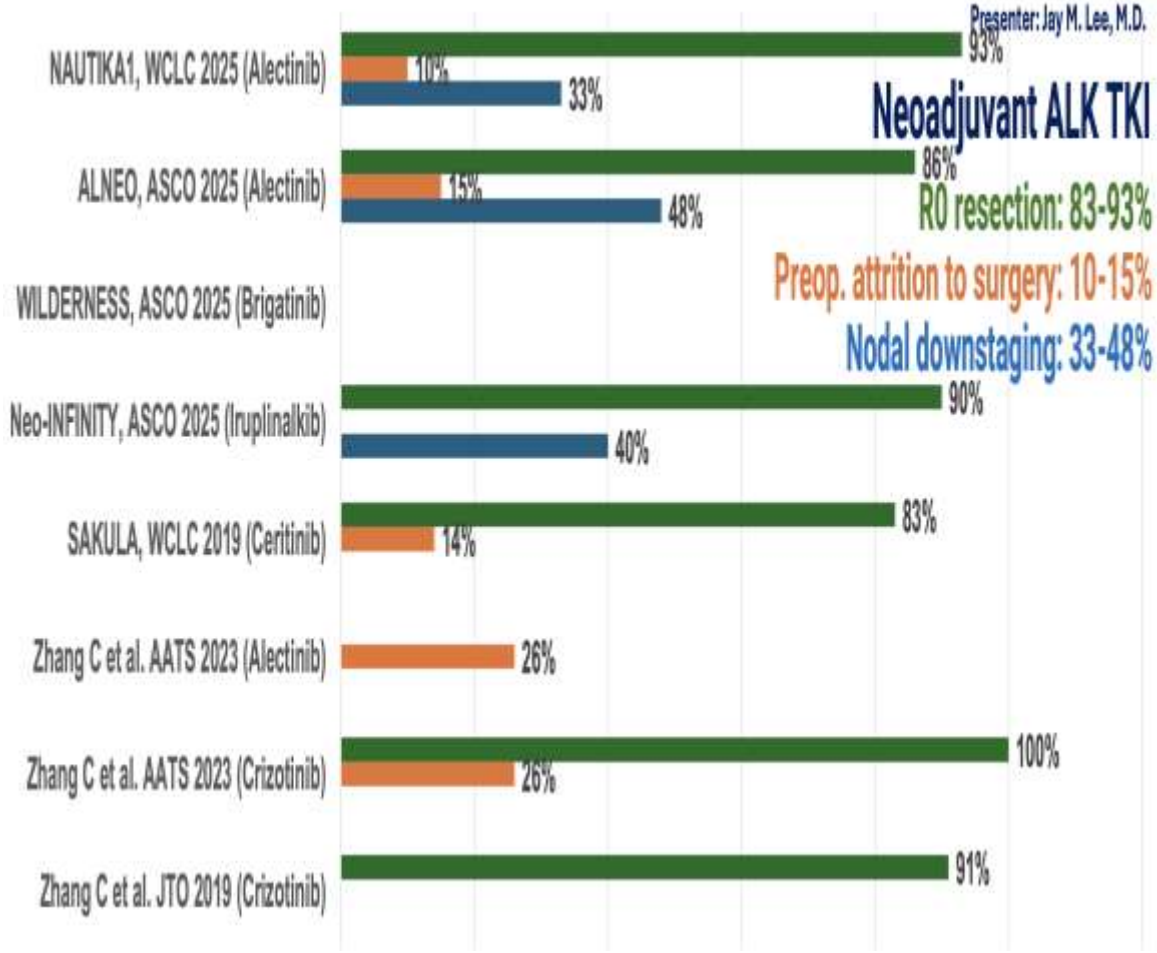
	Ensartinib (n=103)	Placebo (n=102)
Median follow-up	24.0 months	24.0 months
Events	12 (11.7%)	46 (45.1%)
Median DFS, months (95% CI)	NE (NE, NE)	24.8 (22.2, NE)
DFS HR (95% CI)	**0.20** (0.11, 0.38), p<0.0001	

mDFS NE vs 24.8m
HR 0.20

Wu et al NEJM 2024; Dziadziuszko et al, ESMO 2025; Yue et al, ESMO 2025

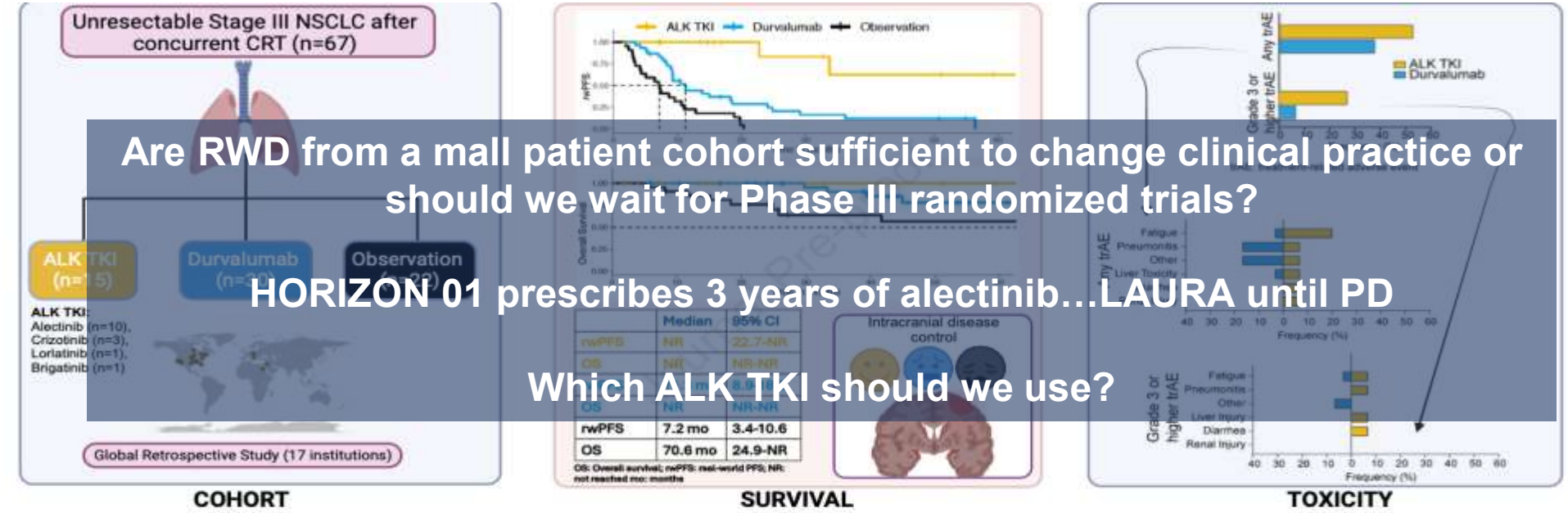
Neoadjuvant ALK TKIs…phase II trials





Lee J, WCLC 2025

CONSOLIDATION ALK TKI IN UNRESECTABLE STAGE III: RWD

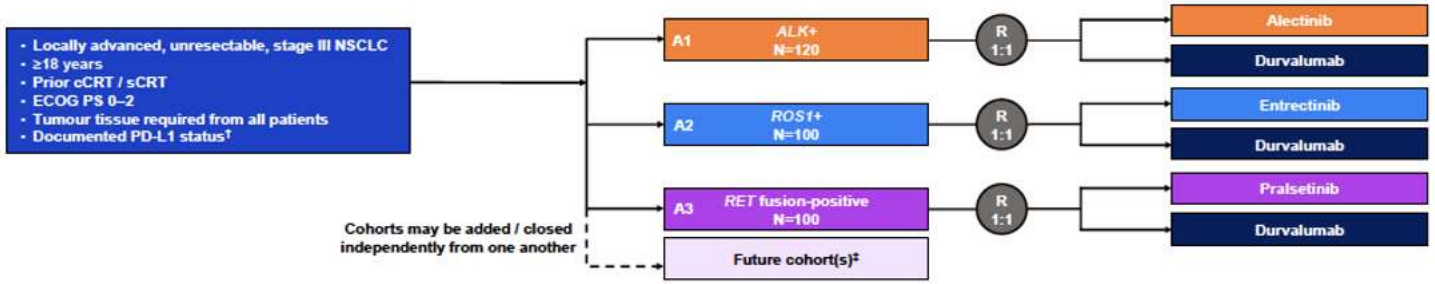


Are RWD from a mall patient cohort sufficient to change clinical practice or should we wait for Phase III randomized trials?

HORIZON 01 prescribes 3 years of alectinib…LAURA until PD

Which ALK TKI should we use?

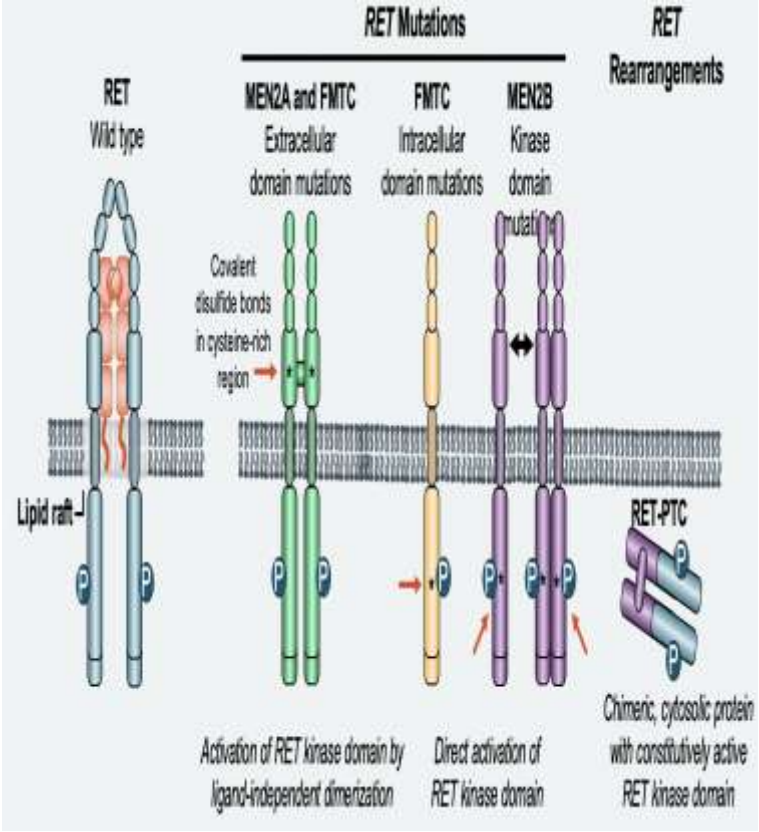
***CONCLUSION**: Consolidation ALK TKI treatment is associated with significantly improved real-world progression-free survival compared to Durvalumab or observation in patients with ALK+ NSCLC*



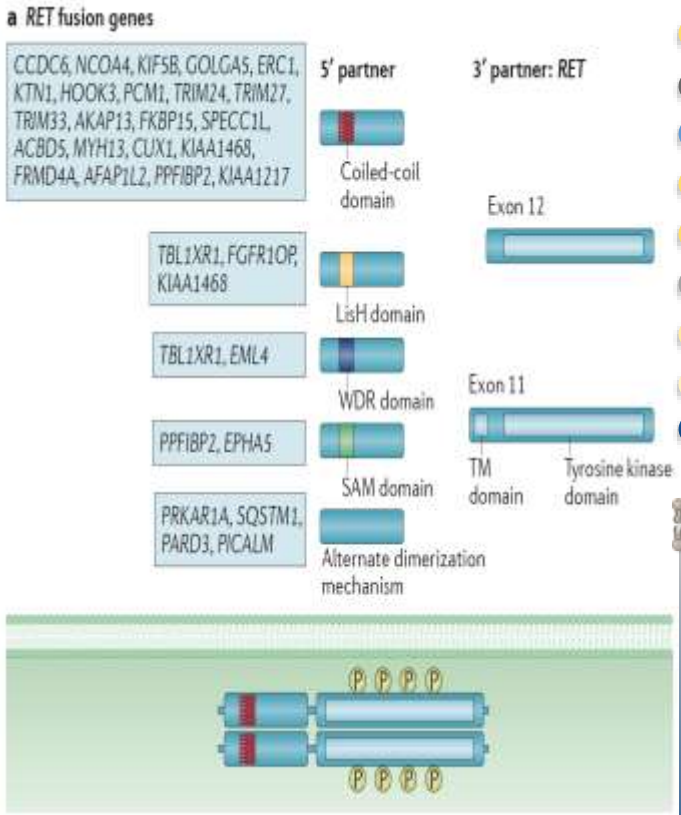
Nassar et al. J Thorac Oncol 2024; Arter et al. J Thorac Oncol 2024

Oncogenic RET fusions and mutations result in constitutive TK activity

RET ONOGENIC MECHANISMS

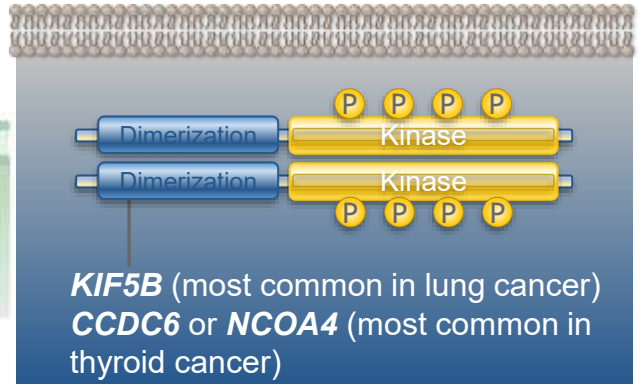


RET FUSIONS



Incidence of *RET* fusions

- **NSCLC (1%-2%)**
- **Papillary thyroid cancer (10%-20%)**
- Pancreatic cancer (<1%)
- Salivary gland cancer (<1%)
- Spitz tumors (<1%)
- Colorectal cancer (<1%)
- Ovarian cancer (<1%)
- Myeloproliferative disorders (<1%)
- Many others (<1%)



KIF5B (most common in lung cancer)
CCDC6 or ***NCOA4*** (most common in thyroid cancer)

Clinico-path characteristics

- **40% had a smoking history**
- **Poorly differentiated,**
- **‘solid-predominant subtype’**
- **Presence of signet ring cells**
- **BM: 25% upfront,**
- **25% concomitant p53 mut**

Subbiah et al. Cancer Disc 2020; Drilon A, et al. Nat Review 2018. Lipson et al. Nature 2012; Wang et al. J Clin Oncol 2012. Li Cancer Treat Rev 2019; Aldea JTO 2023; Cooper et al ASCO 2024

Randomized first line studies in mNSCLC RET+

AcceleRET Lung: Pralsetinib versus SOC **CLOSED and end of recruitment (23 Jan 2024)**

LIBRETTO-431 (NCT04194944)

- Stage IIIB-IV NSCLC harboring *RET* fusion in tissue or in ctDNA
- Non-squamous
- ECOG PS 0-2
- Asymptomatic BM

N=400

R 1:1

Selpercatinib (LOXO-292)

Stratify by:
- Brain metastases (yes vs. no)
- Race (Asian vs. non-Asian)
- Pembrolizumab (yes vs. no)

Platinum-Pemetrexed +/- pembrolizumab chemotherapy

- PD@
- Toxicity
- Other discontinuation criteria

@crossover to Selpercatinib allowed after IRC-assessed PD

Primary Endpoint: PFS by BIRC

ACCELE-RET (NCT04222972)

- Metastatic NSCLC harboring *RET* fusion in tissue and ctDNA
- Non-squamous and squamous
- ECOG PS 0-1
- Asymptomatic BM

N=250

R 1:1

Pralsetinib (BLU-667) 400 mg QD

Stratify by:
- Brain metastases (yes vs. no)
- Pembrolizumab (yes vs. no)
- ECOG (0 vs. 1)

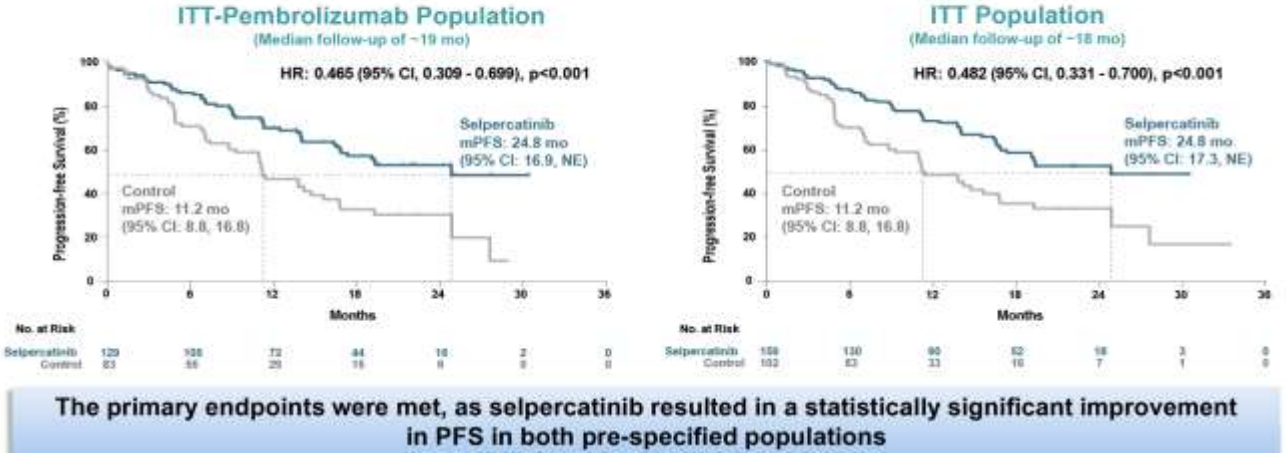
Platinum-Pemetrexed / Gemcitabine +/- pembrolizumab

- PD@
- Toxicity
- Other discontinuation criteria

@crossover to Pralsetinib allowed after IRC-assessed PD

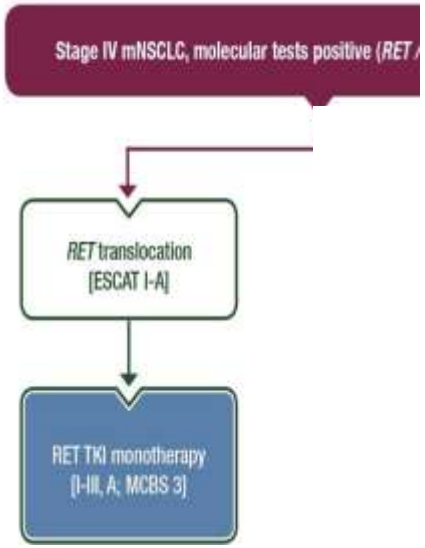
Primary Endpoint: PFS by BIRC

Progression-free survival (PFS) assessed by BICR



The primary endpoints were met, as selpercatinib resulted in a statistically significant improvement in PFS in both pre-specified populations

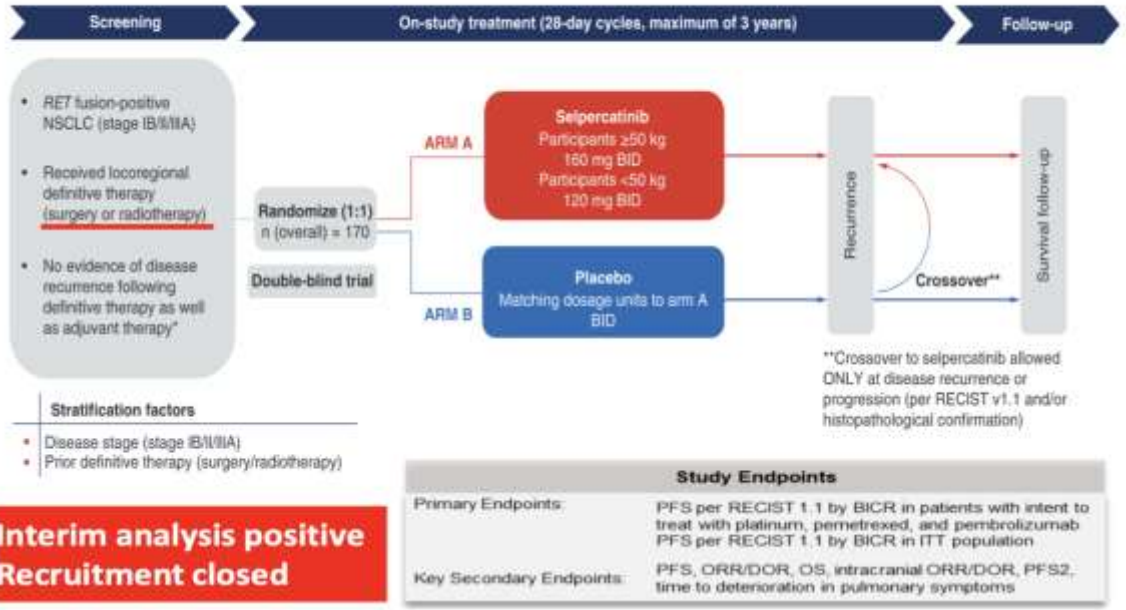
ORR: 83.7% vs 65.1%
mPFS: 24.8m vs 11.2m
mDoR: 24.2m vs 11.5m
icORR: 82.4% vs 58.3%
icPFS 16.1m vs 10.4m



Zhou C, NEJM 2023; Hendriks et al. ESMO Living Guideline 2025

MOVING TO EARLY STAGES

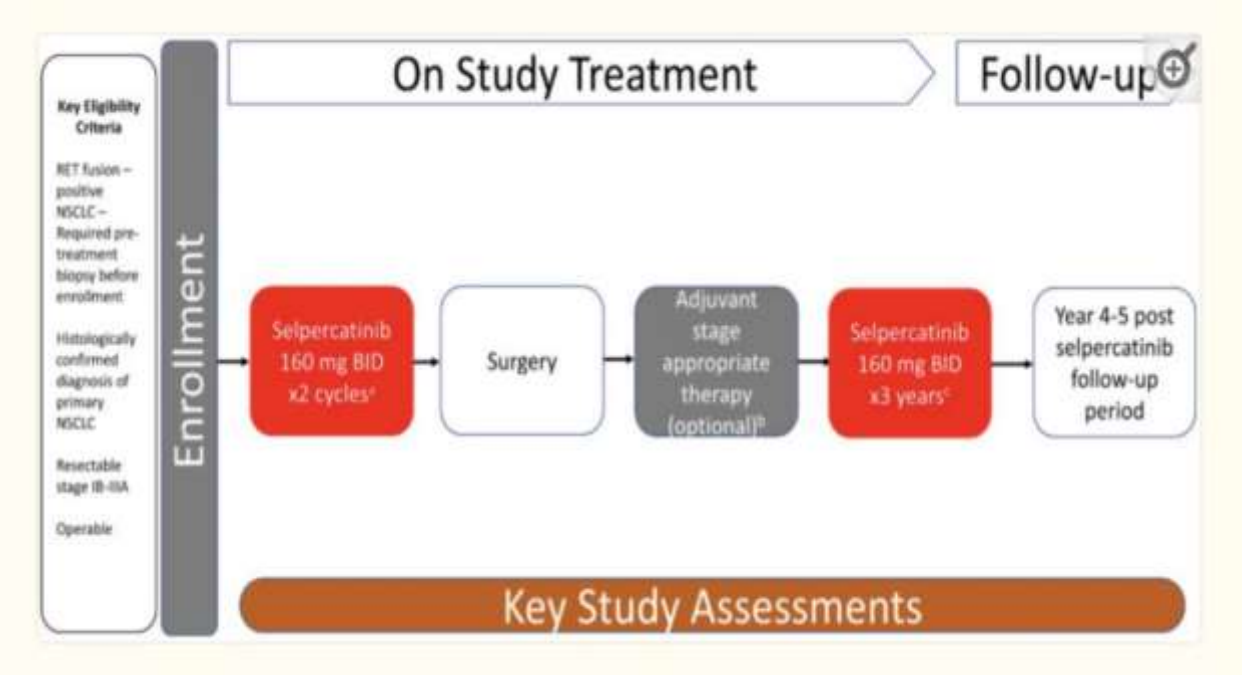
LIBRETTO-432 (Adj, Phase III, RET+. NCT04819100)



Interim analysis positive
Recruitment closed

Study Endpoints	
Primary Endpoints:	PFS per RECIST 1.1 by BICR in patients with intent to treat with platinum, pemetrexed, and pembrolizumab PFS per RECIST 1.1 by BICR in ITT population
Key Secondary Endpoints:	PFS, ORR/DOR, OS, intracranial ORR/DOR, PFS2, time to deterioration in pulmonary symptoms

Ph 2: LIBRETTO-001 (Periop Selpercatinib *RET*+. NCT03157128)



Next generation selective RET TKIs development

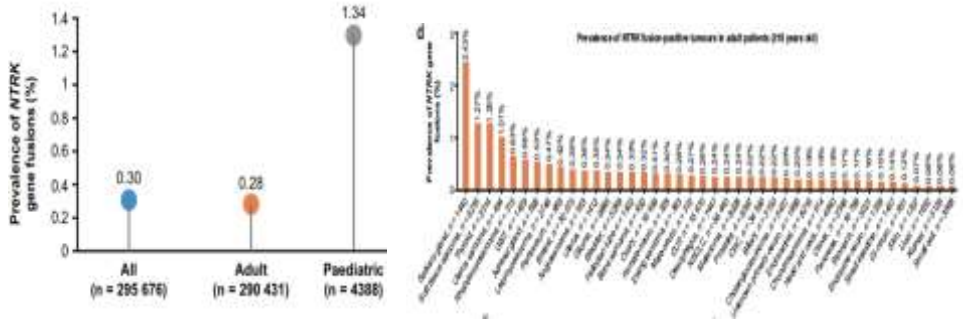
Agent	ClinicalTrials.gov identifier	Patient population, No.	Phase/ design	Status
TPX0046 (RET/ SRC inhibitor)	NCT04161391	Advanced solid tumors harboring *RET* fusions or mutations, 41	Phase 1/ 2, FIH	Terminated (adverse change in risk/benefit)
BOS172738	NCT03780517	Advanced solid tumors with *RET* gene alterations including NSCLC and MTC, 117	Phase 1	Completed recruitment
TAS0953/HM06	NCT04683250	Advanced solid tumors with *RET* gene abnormalities, 202	Phase 1/2	Recruiting in US and Japan
LOXO-260	NCT05241834	Advanced *RET* fusion–positive solid tumors, MTC, and other tumors with *RET* activation refractory to selective RET inhibitors, 110	Phase 1	Active, not recruiting
SY-5007	NCT05278364	Advanced solid tumors, including *RET* fusion–positive NSCLC or *RET*-mutated NSCLC or other *RET*-altered advanced solid tumors, 184	Phase 1/ 2, FIH	Recruiting in China
EP0031	NCT05443126	Advanced *RET*-altered malignancies, 265	Phase 1/2	Recruiting in US and Europe
APS03118	NCT05653869	Unresectable locally advanced or metastatic solid tumors harboring *RET* mutations or fusions, 35	Phase 1, FIH	Recruiting in China
TY-1091	NCT05675605	Advanced *RET*-altered NSCLC, MTC, and other *RET*-altered solid tumors that have progressed after standard therapy, 248	Phase 1/ 2, FIH	Recruiting in China
HEC169096	NCT05451602	Advanced solid tumors, including *RET* fusion–positive NSCLC, MTC, and other tumors with *RET* activation, 456	Phase 1/2	Recruiting in China
HS-10365	NCT06147570	Treatment-naive locally advanced or metastatic *RET* fusion–positive NSCLC, 62	Phase 2	Recruiting in China
HS269	NCT05058352	Advanced solid tumors that fail or where no standard treatment is available, 36	Phase 1, FIH	Recruiting in China
KL590586	NCT05265091	Advanced solid tumors carrying *RET*-fusion or -mutant genes, 414	Phase 1/2	Recruiting in China
HA121-28	NCT05117658	*RET* fusion–positive NSCLC after at least one line of therapy, 83	Phase 2	Recruiting in China

ACTIVATING *NTRK* FUSIONS RESULTS IN TRK FUSION PROTEINS THAT ARE ONCOGENIC DRIVERS

SEVERAL CANCER HISTOLOGIES, DIVERSE AGES…

Prevalence by age

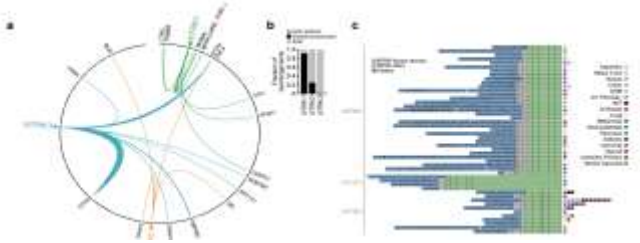
Prevalence by cancer

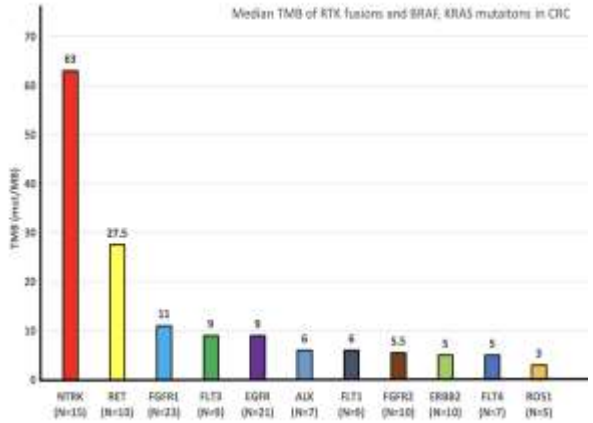


RARITY OF CO-OCCURRENCE OF TRK FUSION WITH OTHER CANONICAL ONCOGENES

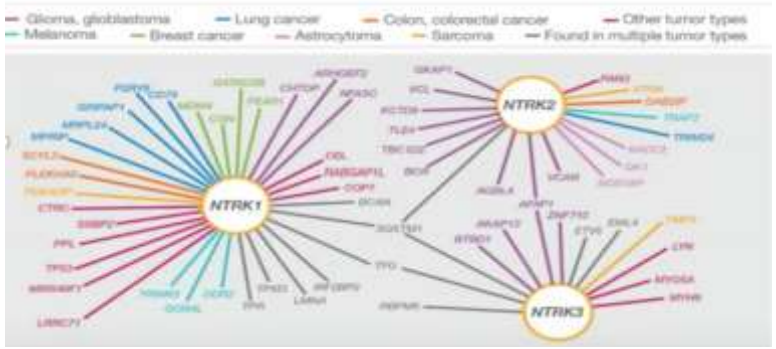
NRTK+ AND RET+ CRC POSSESS HIGHER TMB THAN OTHER RTK+ CRC OR NTRK+/RET+ NON-CRC SOLID TUMORS

ˣNTRK CRC (H-TMB, MSI-H)

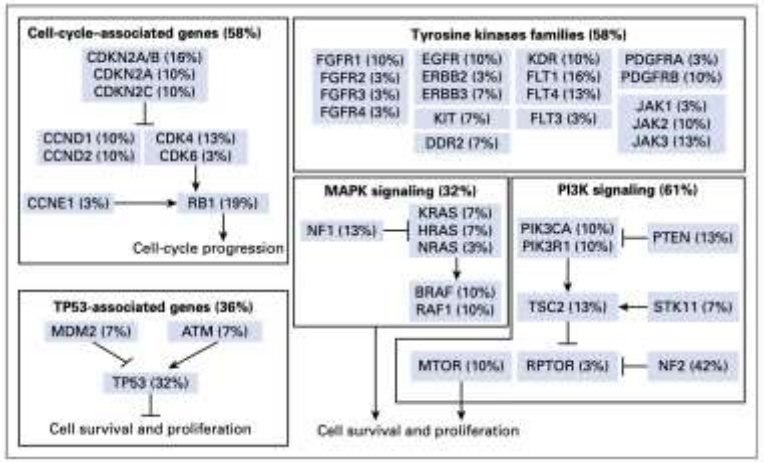


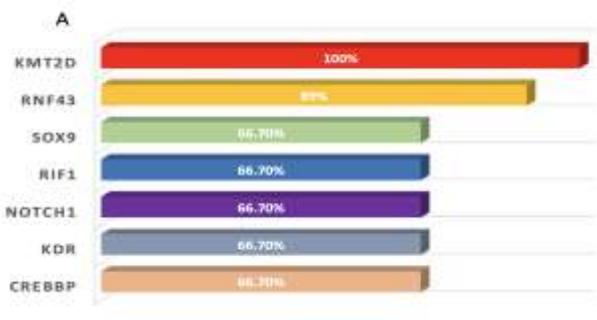


MORE THAN 80 FUSION PARTNERS



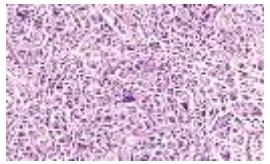
GENOMIC CO-ALTERATIONS ASSOCIATED WITH NTRK





Amatu et al. ESMO Open 2016; Vaishnavi et al. Cancer Disc 2015; Penautl-Llorca et al. J Clin Pathol 2019; Hymann et al. ASCO 2017; Rosen EY, et al. Clin Cancer Res 2020; Westphalen et al. Nature Precision Oncol 2021; Okamura et al, JCO Prec Oncol 2018; Arter et al. Cancer Med 2025

NTRK NSCLC patients



82% ADC

~60% NTRK1

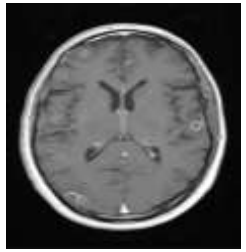
55% *vs.* 45%

73% Never-S

Higher NTRK in Asian ancestry

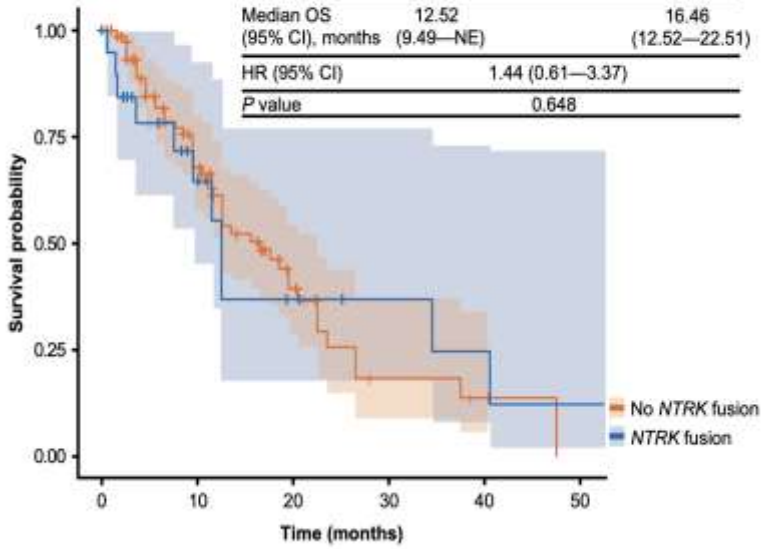
Median age ~50 years

ROS1



~30% BM

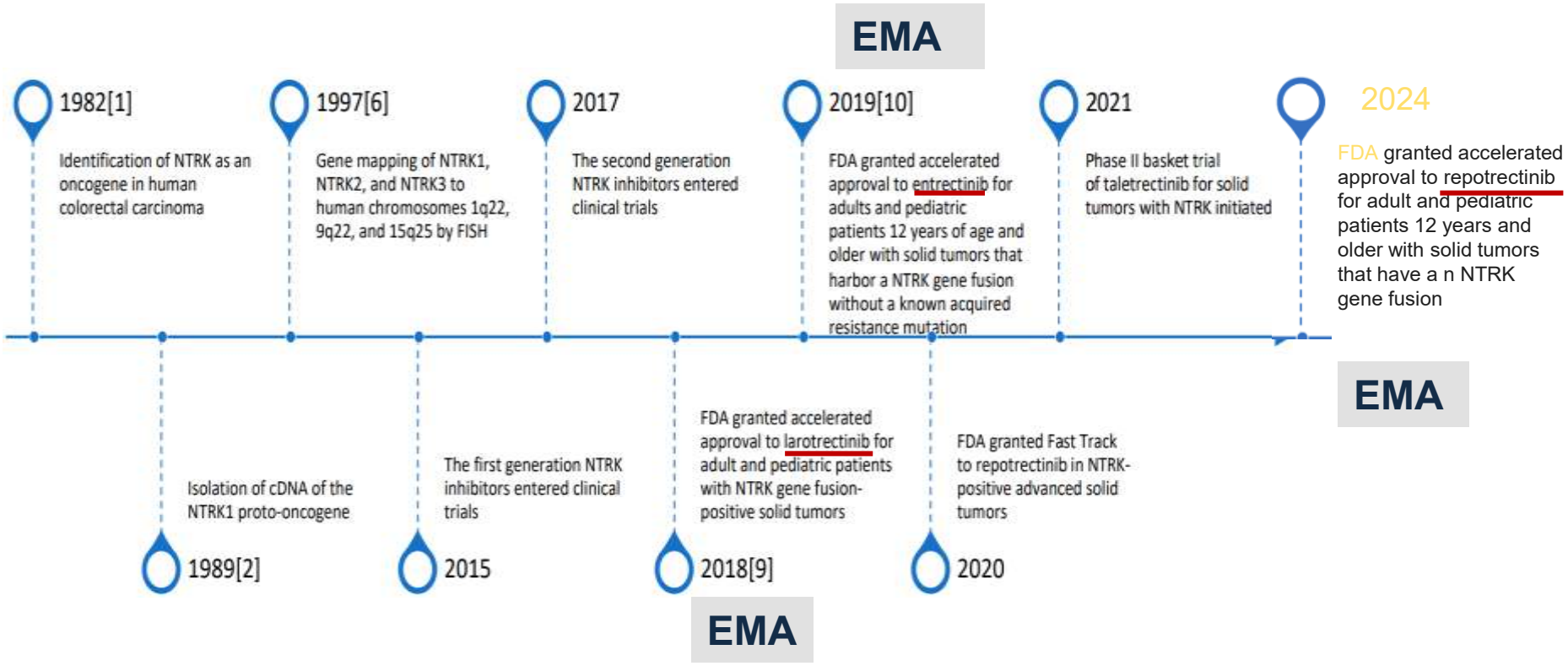
	NTRK fusion (*n* = 27*)	*NTRK* wild-type (Matched) (*n* = 107)
Events, *n* (%)	11 (41)	45 (42)
Median OS (95% CI), months	12.52 (9.49—NE)	16.46 (12.52—22.51)
HR (95% CI)	1.44 (0.61—3.37)	
P value	0.648	

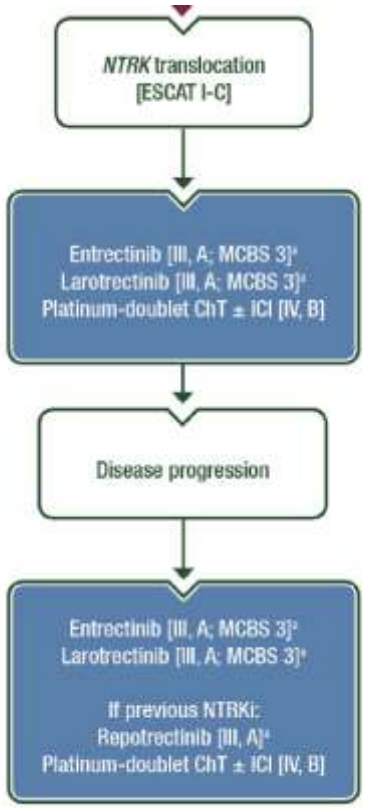


Patients with NTRK fusions do no have better or worse prognosis than those patients with NTRK WT

Farago et al, JCO Prec Oncol 2018; Westphalen et al. Nature Prec Oncol 2021 Overbeck et al, Cancers 2023; Bazhenova et al. Target Oncol 2021

Timeline of NTRK genes discovery and NTRK therapy approvals





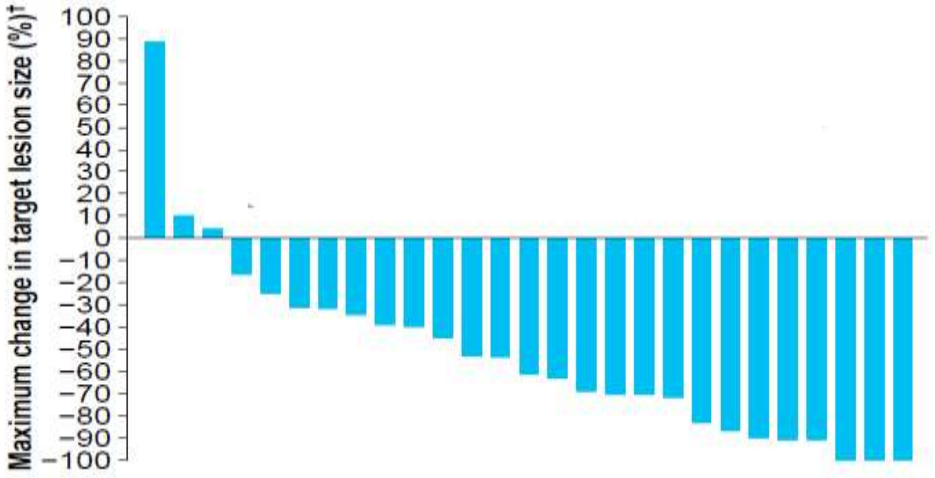
Adapted from Patel, Int. J. Mol. Sci. 2022
Hendricks et al. ESMO Living Guidelines 2025

1st generation TKI updated efficacy in NSCLC

LAROTRECTINIB

Pooled analysis
LOXO-TRK, SCOUT, NAVIGATE

N=32 adults, 38% >3 lines, 38% BM



ORR 66%
mPFS 22m (95% CI 10-NE
mOS 39m (95% CI 17-NE)

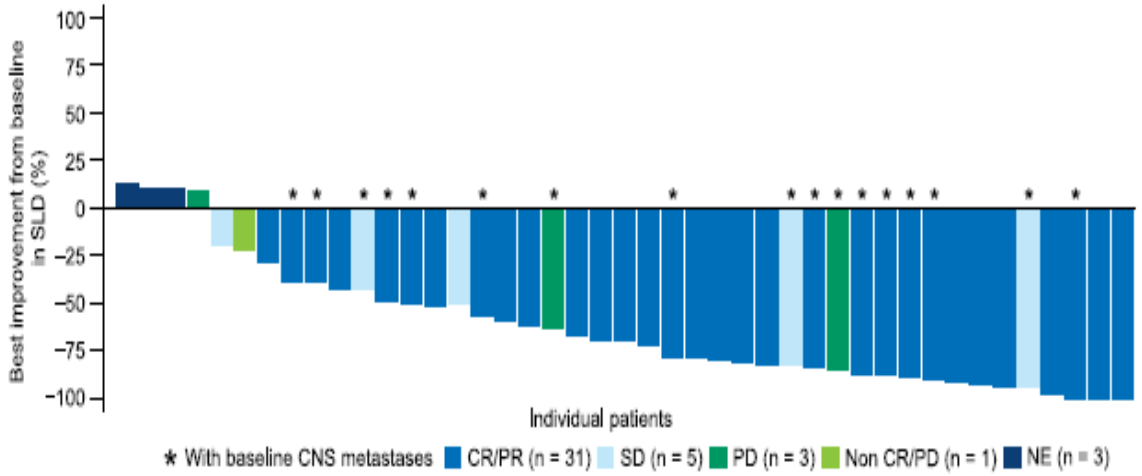
12p

icORR 67% (PR 100%)
mPFS 9.9m
mOS 19.4m

ENTRECTINIB

Pooled analysis
ALKA 372-001, STARTRK-1, STARTRK-2

N=51 adults, 40% >2 lines, 39% BM



ORR 63% (w/o BM 78%, w BM 54%)
mPFS 28m (95% CI 15.8-30.4)
mOS 41.5m (95% C 30.9-NE)

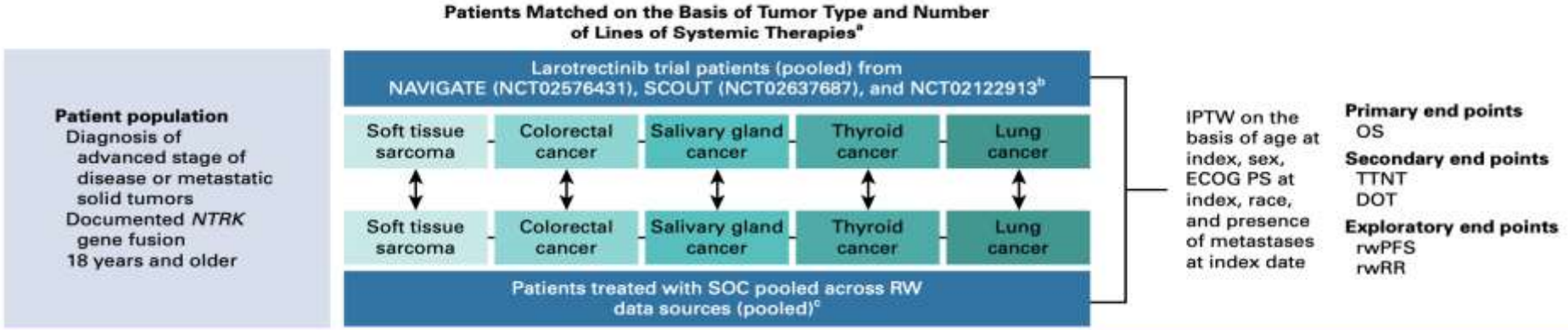
20p

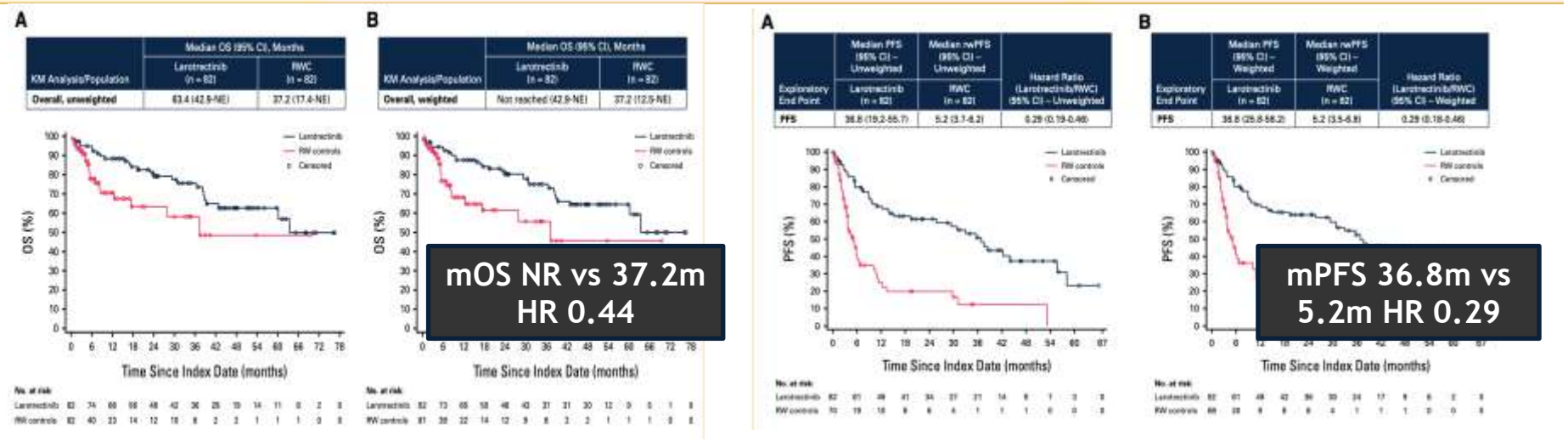
icORR 64% (CR 50%)
icDoR 55.7m
icPFS 32.7m

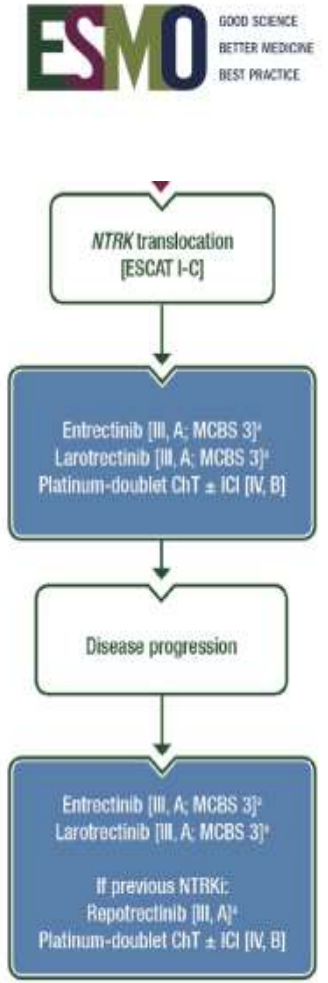
Lin et al WCLC 2024; Cho et al. Clin Lung Cancer 2024

We don´t have a comparative phase III trial NTRK TKI vs SOC…

164 pts, 5 tumor types, matched 1:1



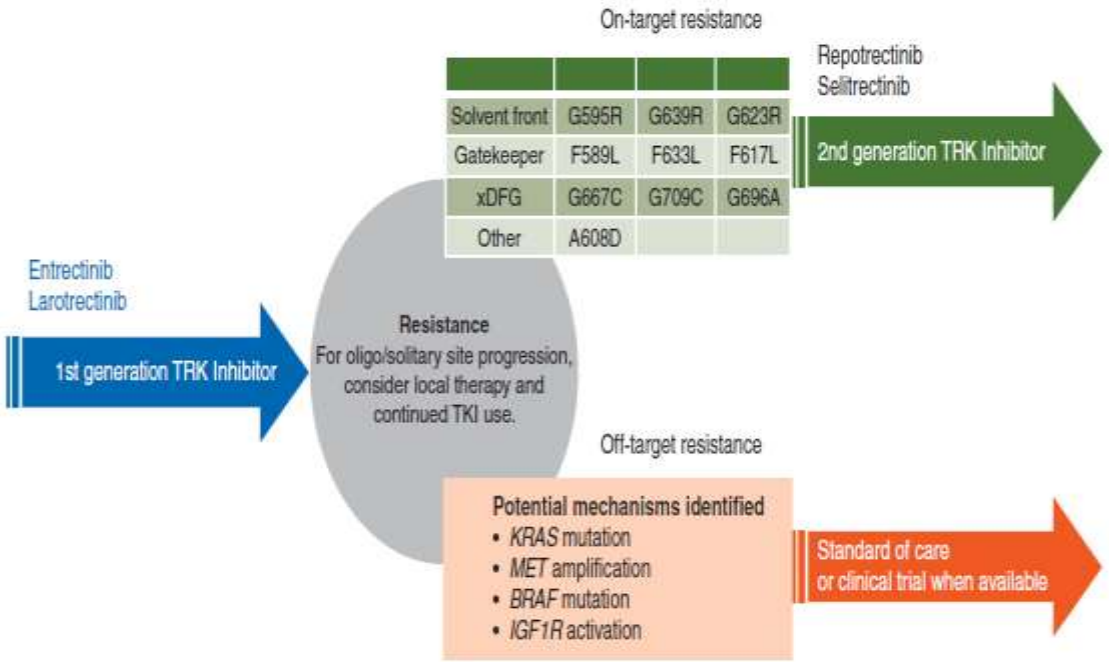




In the absence of randomized clinical trial, this analsys demonstrates the feasibility of external control-arm on the basis of RWD, even in the context of ultra-rare alterations

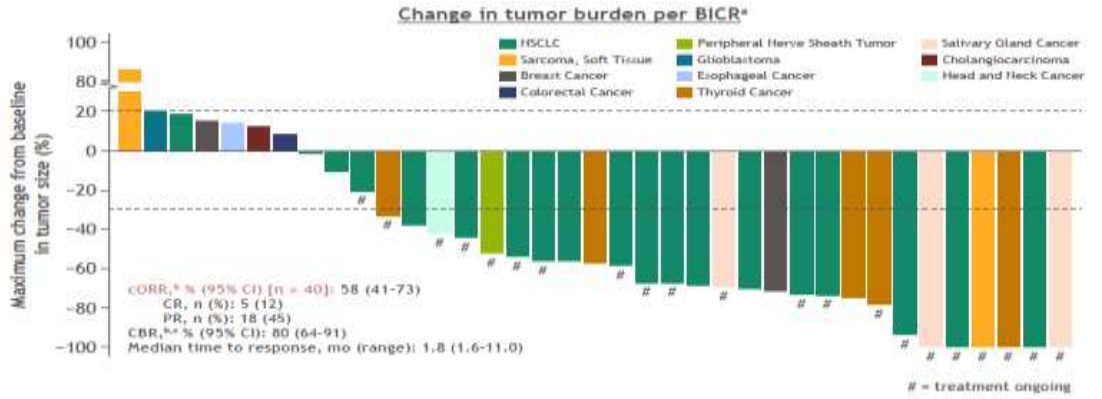
Brose M, et al. JCO Prec Oncol 2025

MANAGING SECONDARY RESISTANCE: REPOTRECTINIB



REPOTRECTINIB: TRIDENT1

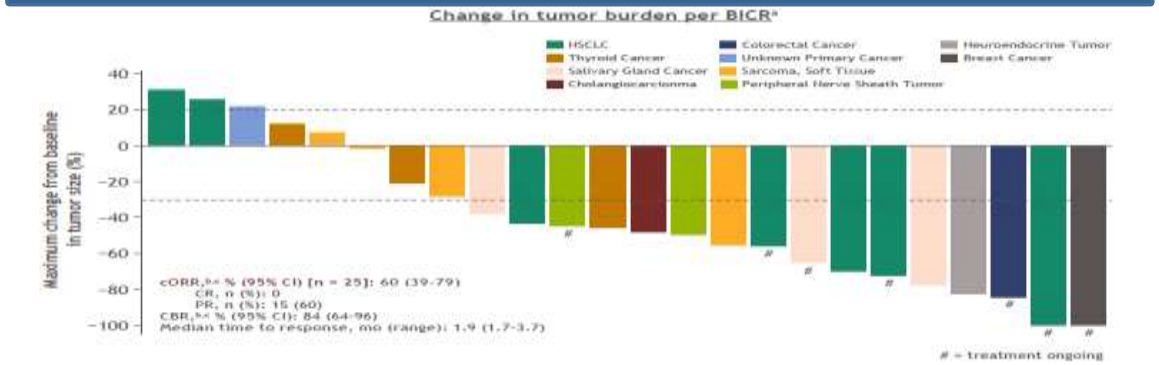
n=17 NSCLC TKI naive ORR 79%



REPOTRECTINIB in acquired resistance

n=7 NSCLC TKI pretreated with solvent front mutations

ORR = 71%

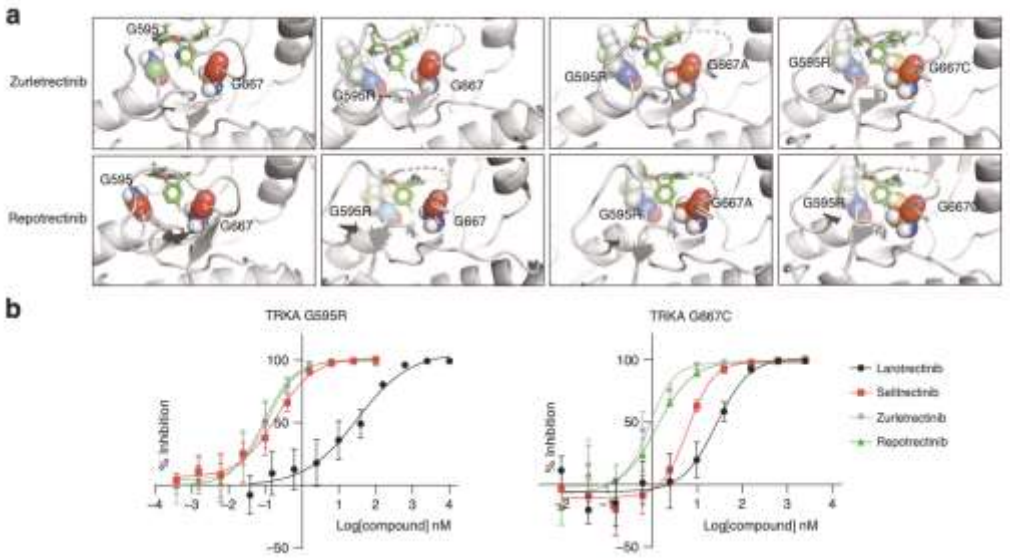


Drilon Ann Onc 2019; Solomon et al ESMO 2023

Personalizing the managing “on target” resistance Zuletrectinib…A new player

Cell lines/*NTRK* status	IC_{50} [nM]			
	Larotrectinib	Selitrectinib	Zuletrectinib	Repotrectinib
Ba/F3 *LMNA-NTRK1*	21.8	3.38	1.2	2.23
Ba/F3 *ETV6-NTRK2*	53.6	9.1	5.61	5.94
Ba/F3 *ETV6-NTRK3*	14.2	1.89	1.4	1.46
Ba/F3 *LMNA-NTRK1*-F589L	614	46.9	40.8	4.43
Ba/F3 *ETV6-NTRK2*-F633L	3113	119	163	11.9
Ba/F3 *ETV6-NTRK3*-F617L	893	21.1	31.2	2.41
Ba/F3 *LMNA-NTRK1*-G595R	3204	18.1	11	16.2
Ba/F3 *ETV6-NTRK2*-G639R	3809	81.2	40	60.1
Ba/F3 *EVT6-NTRK3*-G623R	962	6.7	6.45	11.6
Ba/F3 *ETV6-NTRK3*-G623E	61.3	3.23	0.69	3.06
Ba/F3 *LMNA-NTRK1*-G667A	118	21.1	5.27	6.48
Ba/F3 *LMNA-NTRK1*-G667C	1368	163.6	38.8	68.1
Ba/F3 *LMNA-NTRK1*-G667S	3195	537	193	139
Ba/F3 *ETV6-NTRK2*-G709C	2788	323	72.6	116
Ba/F3 *ETV6-NTRK3*-G696A	45.7	5.1	2.24	2.84
Ba/F3 *ETV6-NTRK3*-G696C	780	51.6	15.5	32.2
Ba/F3-*LMNA-NTRK1*-V573M	45.4	5.47	2.33	2.22
Ba/F3 *LMNA-NTRK1*-A608D	19.7	3.16	1.18	2.12
Ba/F3 *ETV6-NTRK2*-V689M	13.9	1.44	1.01	1.24

▲ Solvent front substitutions ★ Gate keeper substitutions ✿ xDFG mutations



News | Article | May 2, 2025

Zurletrectinib Receives Priority Review from NMPA for NTRK Gene Fusion+ Advanced Solid Tumors

Author(s): Ashley Chan

Abstract
2025 ASCO Annual Meeting

Updated efficacy and safety of zurlectrectinib in adult patients (pts) with locally advanced or metastatic NTRK fusion–positive (NTRK+) solid tumors.

The full, final text of this abstract will be available at 5:00 PM ET on May 22, 2025

Roa et al. BJC 2024; Wei et al. ASCO 2022

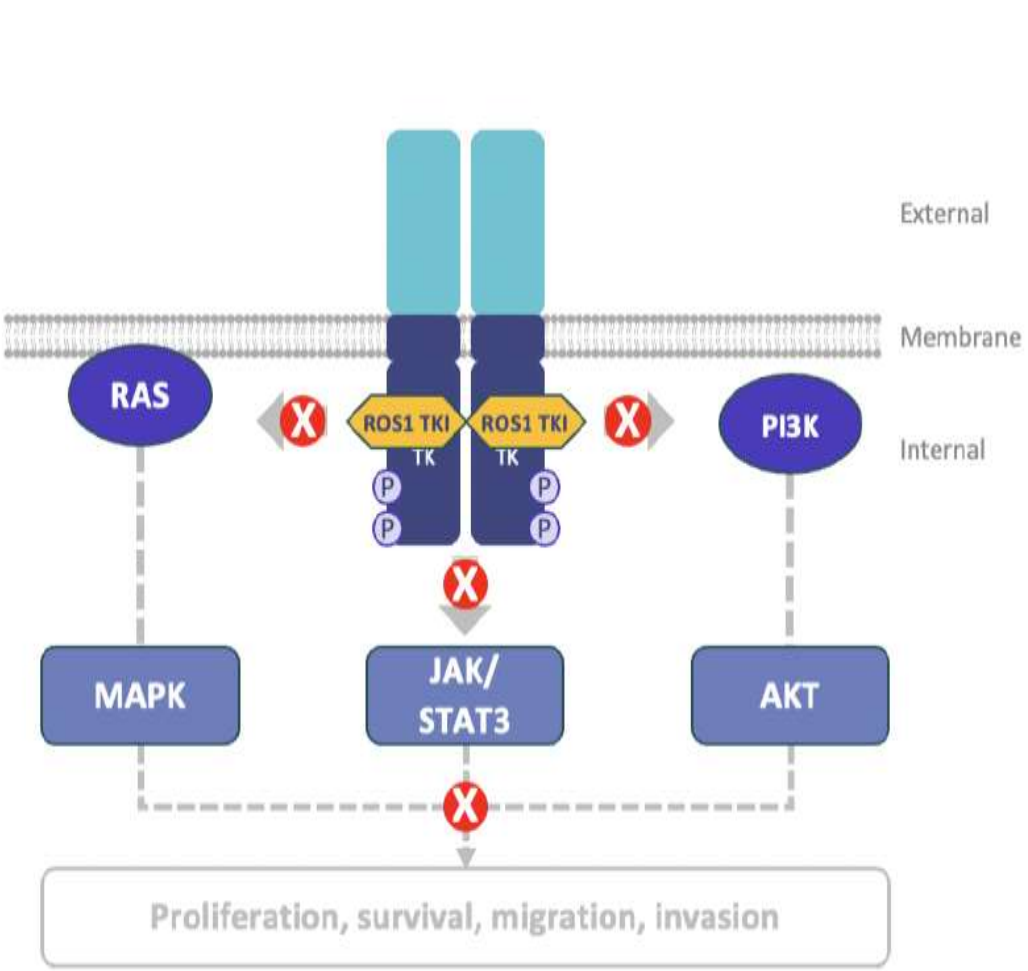
So crouded scenario…

Study	Phase	Drug	Gene alteration	Tumor Type	Status
NCT04671849	I	SIM1803-1A	NTRK, ROS1 or ALK fusion	All Solid	Recruiting
NCT04687423	II	FCN-011	NTRK fusion	All Solid	Recruiting
NCT05212987	I	FCN-098	NTRK fusion	All Solid	Recruiting
NCT04996121	I/II	XZP-5955	NTRK, ROS1 Fusion	All Solid	Recruiting
NCT05745623	I/II	ICP-723	NTRK fusion	All Solid	Recruiting
NCT05769075	I	TY-2136b	NTRK, ROS1 or ALK fusion	All Solid	Recruiting
NCT04879121	II	Larotrectinib	NTRK Amplification	All Solid	Recruiting
NCT01639508	II	Cabozantinib	NTRK, ROS1 fusions, MET/AXL	NSCLC	Recruiting
NCT06010342	II	TL118	NTRK fusion	All Solid	Not Yet Recruiting
NCT05302843	I	BPI-28592	NTRK fusion	All Solid	Recruiting
NCT03556228	I	VMD-928	NTRK fusion	All Solid	Recruiting

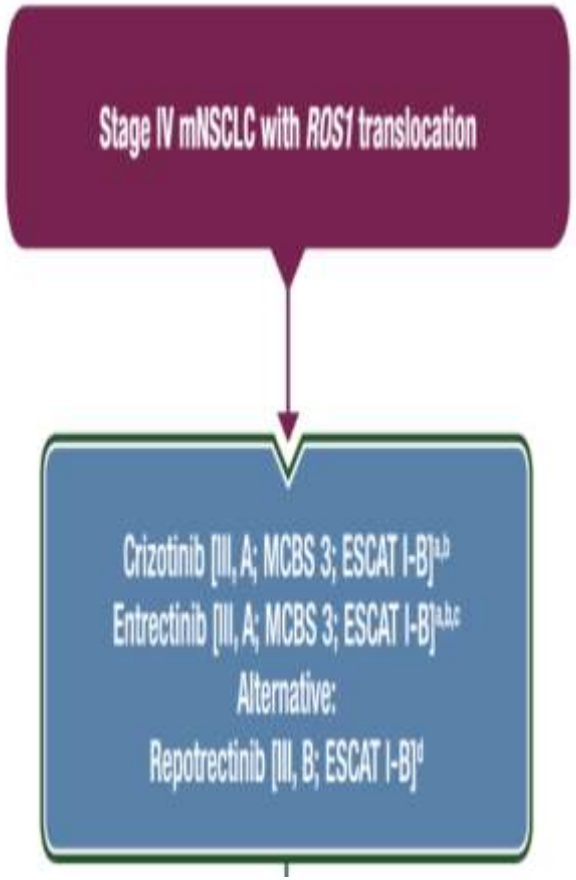
Adapted from Hagopian et al. Crit Rev Oncol Hematol 2024

ALK & ROS-1 fusion kinases: high degree of homology

- Located on chromosome 6q22, the ROS1 gene encodes an orphan receptor tyrosine kinase for which an activating ligand has yet to be described.
- ROS1 gene fusions account for 1-2% of NSCLC.
- 40% baseline CNS metastases
- Poor outcomes with platinum-based CT
- ROS1 and ALK are phylogenetically related, leading to a biological rationale for the use of some ALK inhibitors in ROS1 NSCLC.



ROS1 fusions: general outcomes with current options (1L)



	CRIZOTINIB	ENTRECTINIB	REPOTRECTINIB
N	270	67	71
ORR (%)	65-73	77	79
mPFS, months (95%CI)	19-24.7	17.7 (11.8-39.4)	35.7m (27.4-NR)
mOS, months (95%CI)	17-51.4	47.7 (43.2-NR)	NA *88% surv. rate @18m*
TRAEs (Gr3/4), %	94-98 (23-36)	92 (34)	96 (29)

Hendriks et al. ESMO Living Guidelines 2025; Ou I, et al. JTO 2024; Shaw A et al, NEJM 2014; Mazieres et al, J Clin Oncol 2015; Barlesi F, et al. ELCC 2019; Drilon A, Lancet Oncol 2020; Drillon A, et al. NEJM 2024

Efficacy of ROS1 TKI in naive patients

ROS1 Inhibitor	ORR (%)	Median PFS	BM Prevalence	Intracranial ORR
Crizotinib (PROFILE 1001)	72%	19.2 mo	Not eligible	-
Crizotinib (OxOnc)	72%	15.9 mo	Not eligible	-
Brigatinib	71%	12.0 mo	11% (3/28)	0%
Entrectinib	68%	15.7 mo	29% (48/168)	52%
Ceritinib	62%	19.3 mo	25% (8/32)	25%
Lorlatinib	62%	21.0 mo	52% (11/21)	64%

Shaw et al, NEJM 2014; Wu et al, JCO 2018; Niho et al, ESMO Open 2024; Drilon et al, JTO Clin Res Rep 2022; Lin et al, JCO 2017; Shaw et al, Lancet Oncol 2019

Lorlatinib after failure of first line ROS1: IFCT -2003 ALBATROS study

54 patients; 57% Brain Mts; 94% prior crizotinib

Efficacy of lorlatinib after failure of a first-line ROS1 tyrosine kinase inhibitor (ROS1 TKI) in patients (pts) with advanced ROS1-positive non-small cell lung cancer (ROS1+ NSCLC): IFCT-2003 ALBATROS trial

NCT04621188 / EU CT 2024-512028-12-00

Single-arm, multicenter phase II trial

- ROS1-positive advanced NSCLC according to IHC and confirmed with FISH or NGS (local)
- Progression after a first-line therapy with ROS1 TKI
- PS 0, 1 or 2
- Stable and asymptomatic brain metastases allowed
- Measurable disease according to RECIST 1.1

Mandatory centralized tumor tissue and blood samples

- Lorlatinib 100 mg once daily
- Planned inclusion of 84 patients *
- Until progression or intolerable toxicity
- Required blood samples at time of progression

Primary endpoint: investigator-assessed confirmed Overall Response Rate (cORR)

Secondary endpoint: BICR cORR, DCR, PFS, DoR, OS, CNS ORR, safety

Results

- cORR of 30%
- median PFS 7.4 months
- median DoR 20.4 months
- median OS 42.3 months

Secondary endpoints: inv-assessed CNS ORR

	Investigator assessed N = 13 patients with measurable CNS disease
Complete Response	7 (53.8%) [26.7% ; 80.9%]
Partial Response	5 (38.5%) [12.0% ; 64.9%]
Objective Response	**12 (92.3%) [77.8% ; 100.0%]**
Stable Disease	1 (7.7%) [0.0% ; 22.2%]
Disease Control	**13 (100.0%) [100.0% ; 100.0%]**
Progressive Disease	0

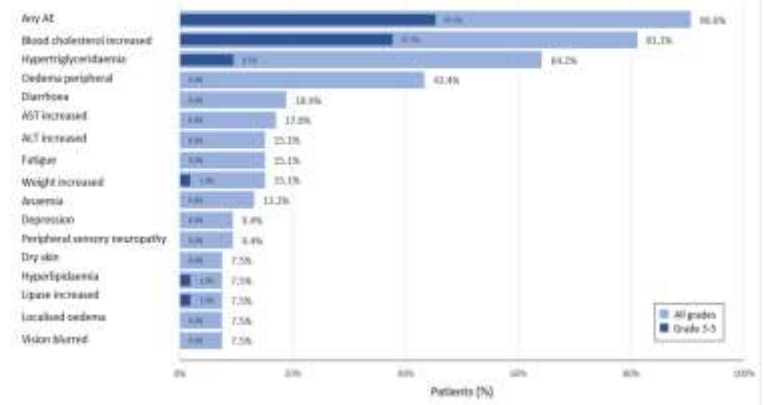
Safety

TRAE grade ≥ 3 rate: 45%

Dose modification rate: 30%

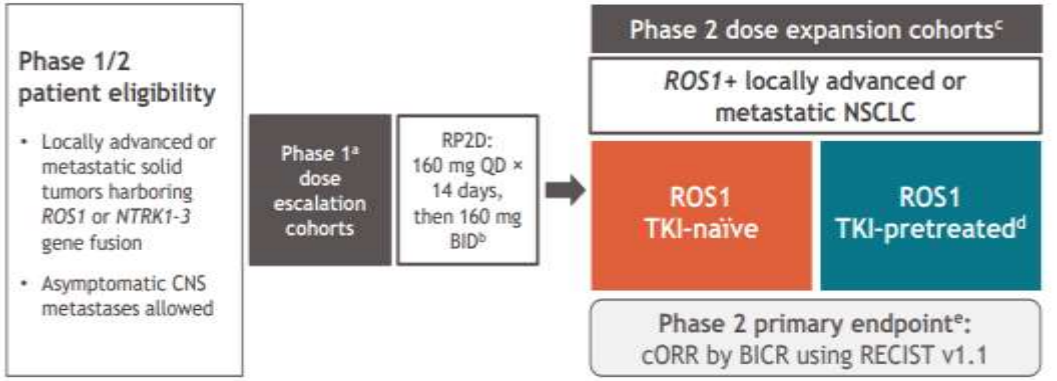
TRAE permanent discontuation rate: 1%

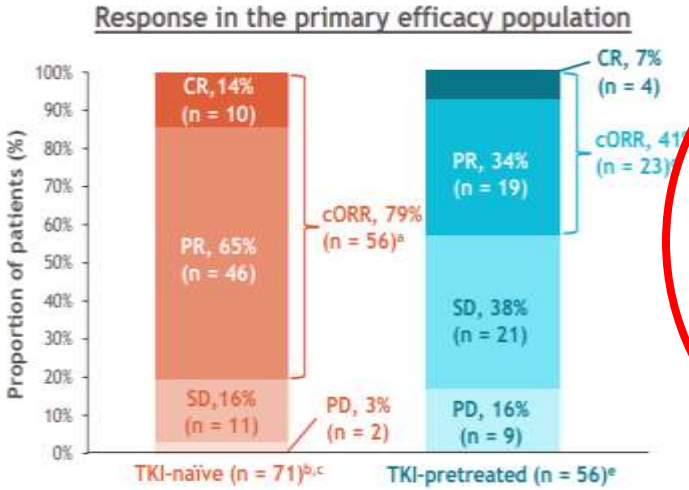
TRAEs with incidence ≥7.5%



Durisseaux M. ESMO 2025

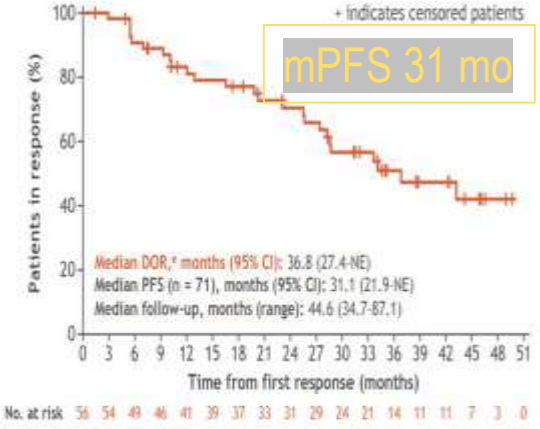
REPOTRECTINIB: long-term FW from phase 1/2 TRIDENT-1 trial



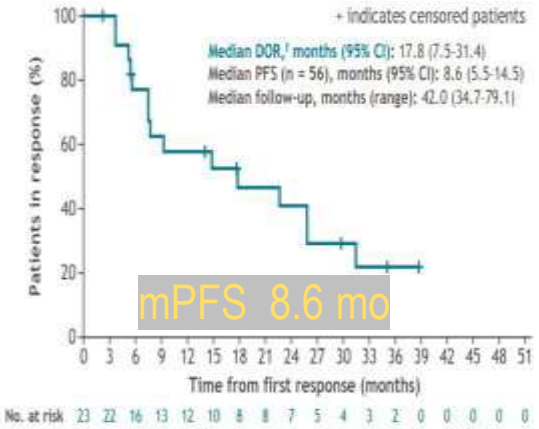


- Among patients with measurable brain metastases at baseline[f]:
  - icORR was 89% in TKI-naïve patients (95% CI, 52-100; n/N = 8/9)[g]; median icDOR was 43.2 months (95% CI, 11.1-NE)
  - icORR was 38% in TKI-pretreated patients (95% CI, 14-68; n/N = 5/13)[h]; median icDOR was NE (95% CI, 3.0-NE)
- Among patients with the *ROS1* G2032R (solvent front) mutation across the 3 TKI-pretreated cohorts (n = 17), cORR was 59% (95% CI, 33-82)

DOR in TKI-naïve patients in the primary efficacy population[d]

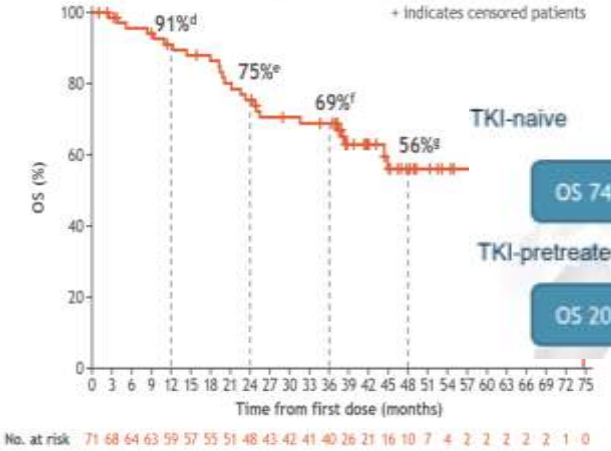


DOR in TKI-pretreated patients in the primary efficacy population

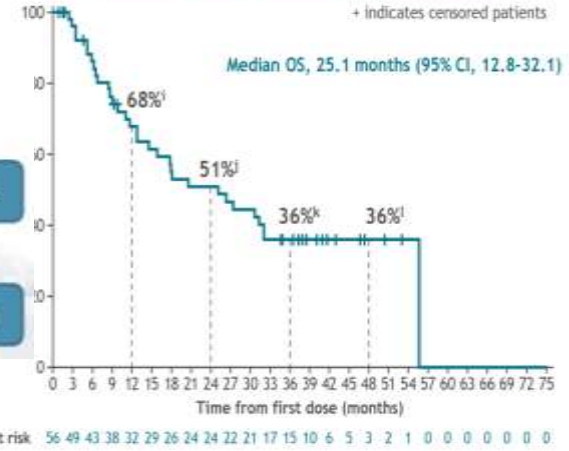


- In the expanded efficacy population, median follow-up was 37.7 months for TKI-naïve patients (n = 121) and 34.8 months for TKI-pretreate patients (n = 107)
  - Median DOR[s] was 33.6 months (95% CI, 25.5-NE) for TKI-naïve patients and 14.9 months (95% CI, 7.7-31.4) for TKI-pretreated patients
  - Median PFS was 30.2 months (95% CI, 19.3-38.6) for TKI-naïve patients and 9.2 months (95% CI, 7.4-11.3) for TKI-pretreated patients

TKI-naïve patients in the primary efficacy population[c]



TKI-pretreated patients in the primary efficacy population[h]



- In the expanded efficacy population,[m] median OS was 74.6 months (95% CI, 44.4-NE) for TKI-naïve patients (n = 121) and 20.5 months (95% CI, 17.8-31.4) for TKI-pretreated patients (n = 107)

TALETRECTINIB in *ROS1*+ Non-Small Cell Lung Cancer: TRUST

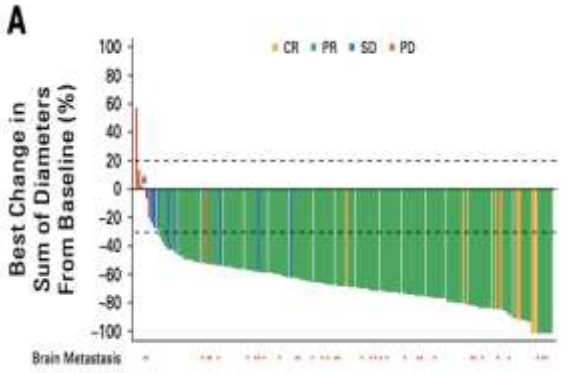
273 mNSCLC TRUST-1 and TRUST-2

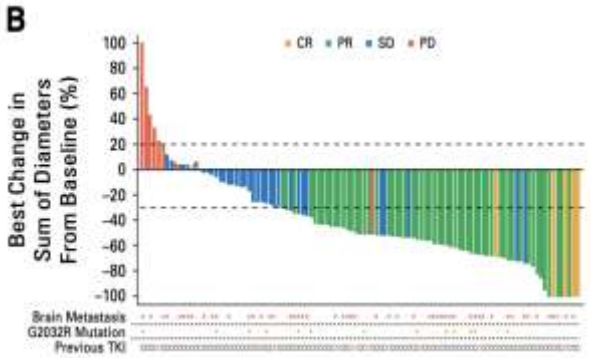
160 TKI naive

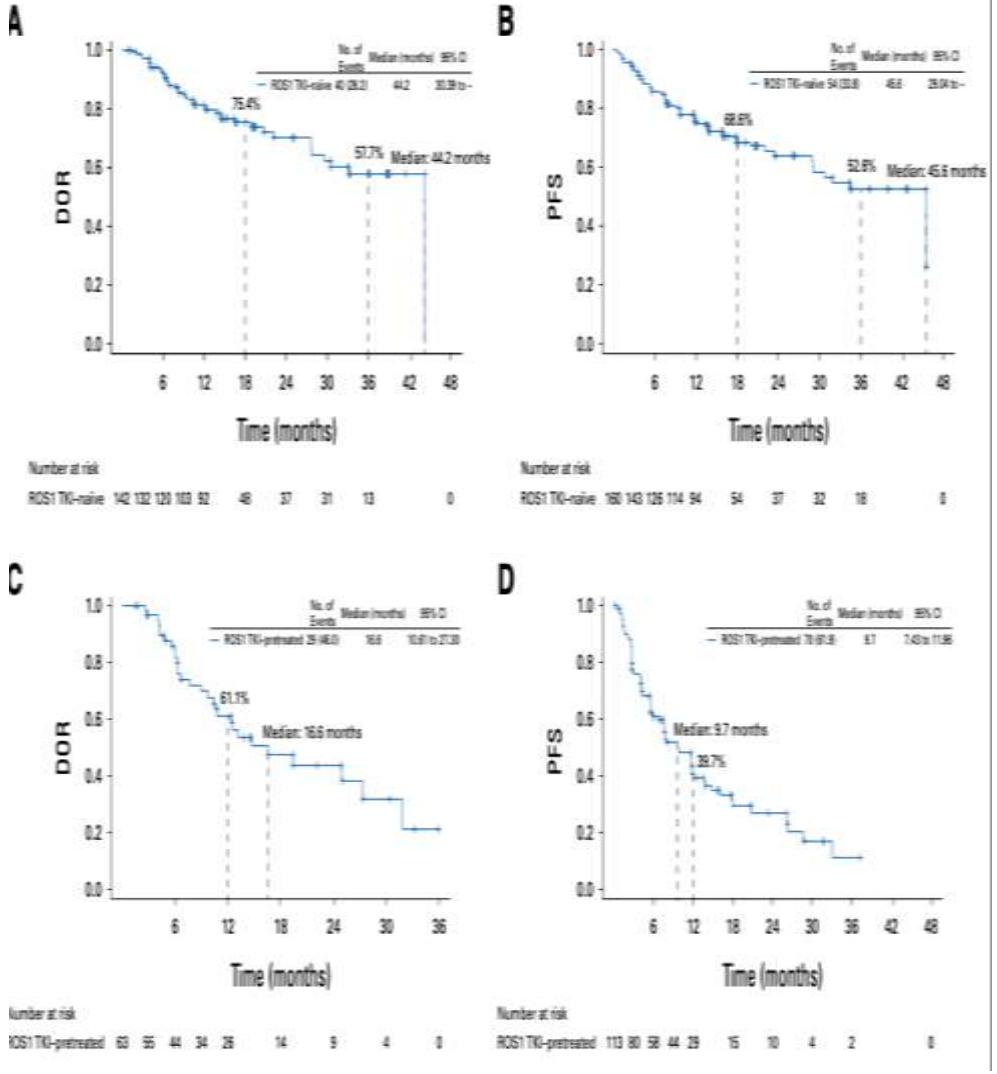
ORR 88.8%
mDoR 44.2m
mPFS 44.6m
icORR 76.5%

113 TKI pretreated

ORR 55.8%
mDoR 16.6m
mPFS 9.7m
icORR 65.6%







Perol et al, J Clin Oncol 2025

ZIDESAMTINIB in TKI Pretreated Patients with Advanced/Metastatic ROS1+ NSCLC: ARROS1

ZIDESAMTINIB DESIGN GOALS:

- ROS1 Activity
- \+
- ROS1 Mutant Activity
- \+
- Brain Penetrance
- \+
- Avoiding TRK

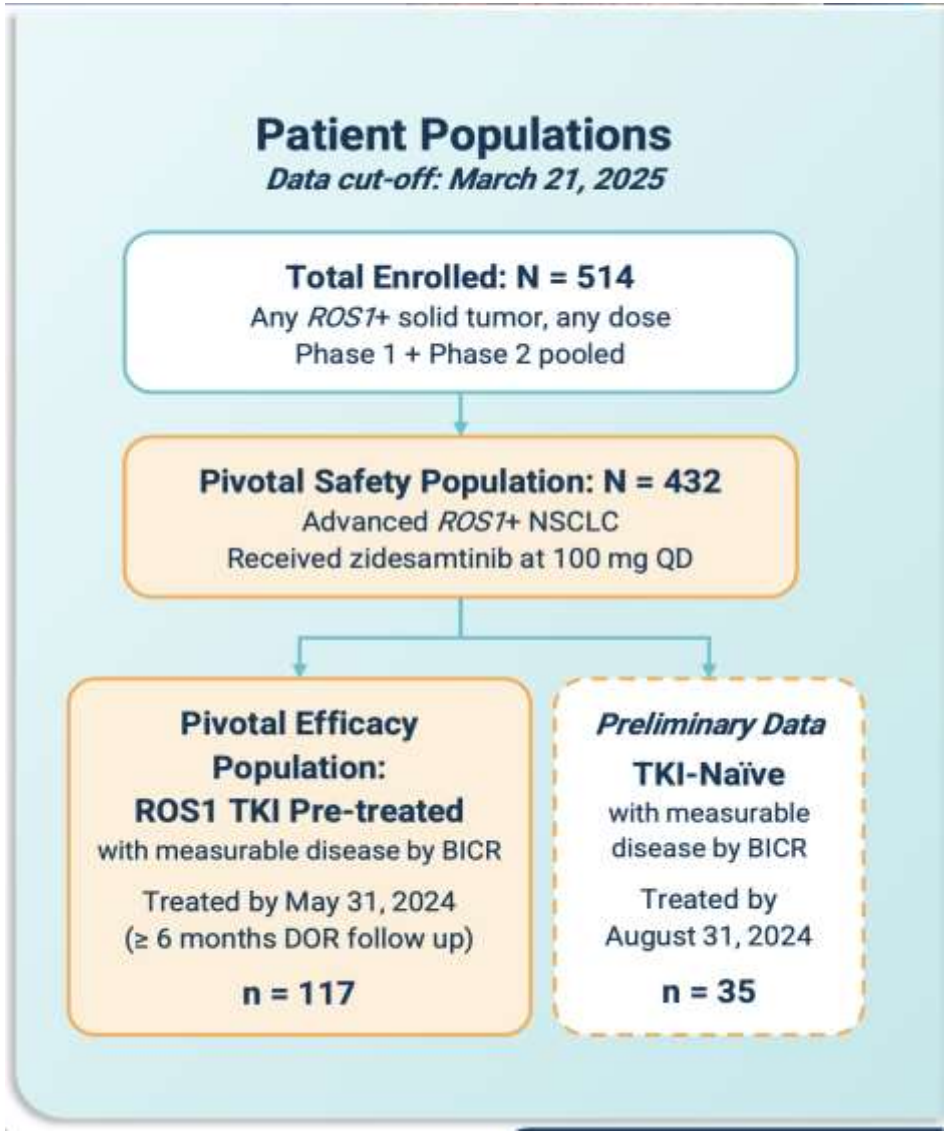
PHASE 1: Zidesamtinib dose escalation (25 – 150 mg QD) in ROS1 TKI pre-treated patients with advanced *ROS1+* solid tumors

PHASE 2: Zidesamtinib 100 mg QD (RP2D)

ARROS-1 PHASE 2 PATIENT POPULATION	PRIOR ROS1 TKI	PRIOR CHEMO/I-O
ROS1+ NSCLC	ROS1 TKI-naïve [a]	≤ 1
	1 prior ROS1 TKI [b]	None
		1 [c]
	≥ 2 Prior ROS1 TKIs [d]	≤ 1
Any *ROS1+* Solid Tumor [e]	Any	Any

PHASE 2 OBJECTIVES

- **Primary:** ORR by blinded independent central review (BICR)
- **Secondary:** Additional efficacy measures (DOR, TTR, CBR, PFS, OS), intracranial activity, overall safety and tolerability, confirmation of PK profile, PROs



Drilon et al, WCLC 2025

Advanced *ROS1*+ NSCLC	PIVOTAL DATASET: Any prior ROS1 TKIs (range 1-4) ± chemotherapy	PIVOTAL DATASET: 1 prior ROS1 TKI (crizotinib or entrectinib) ± chemotherapy	*Preliminary*: ROS1 TKI-naïve
ORR (BICR)	44%	51%	89%
% DOR ≥ 12 months	78%	93%	96%
% DOR ≥ 18 months	62%	93%	
% PFS ≥ 12 months	48%	68%	
% PFS ≥ 18 months	40%	68%	
Intracranial Activity	**Any prior ROS1 TKIs ± chemotherapy**	**Prior crizotinib only ± chemotherapy**	**ROS1 TKI-naïve**
IC-ORR (BICR)	48%	85%	83%
IC-DOR	71% ≥ 12 months	91% ≥ 12 months	No CNS progression among confirmed CNS responders

ARROS-1: Objective Response in ROS1 TKI Pre-treated Patients

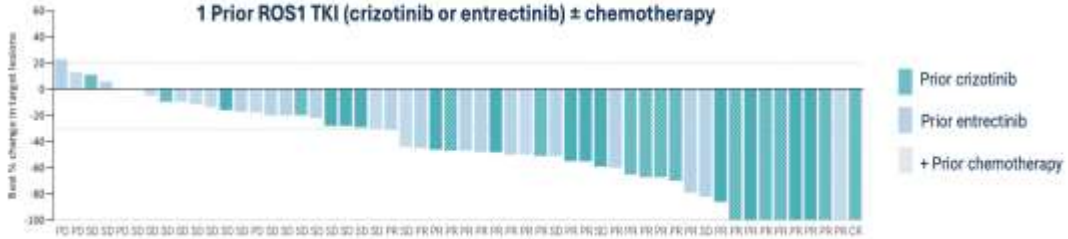
Advanced *ROS1*+ NSCLC RECIST 1.1 by BICR	Any prior ROS1 TKI (range 1 – 4) ± chemotherapy	1 prior ROS1 TKI (crizotinib or entrectinib) ± chemotherapy
ORR, % (n/N) [95% CI]	44% (51/117) [34, 53]	51% (28/55) [a] [37, 65]
CR, % (n/N)	1% (1/117)	2% (1/55)

[a] Prior crizotinib only ± chemotherapy: ORR = 68% (19/28). Prior entrectinib only ± chemotherapy: ORR = 33% (9/27).

Responses were also observed in patients previously treated with:

- **≥2 prior ROS1 TKIs ± chemotherapy:** ORR = 38% (22/58; 95% CI: [26, 52])
- **Prior repotrectinib: ORR = 47% (8/17),** DOR range 3.5 to 17.2 months
- **Prior taletrectinib: ORR = 43% (3/7),** DOR range 5.2 to 7.0+ months

1 Prior ROS1 TKI (crizotinib or entrectinib) ± chemotherapy



***ROS1* G2032R Resistance Mutation**

Advanced *ROS1*+ NSCLC Analysis by BICR	Any prior ROS1 TKI ± chemotherapy	1 prior ROS1 TKI (crizotinib or entrectinib) ± chemotherapy [a]
ORR, % (n/N) [95% CI]	54% (14/26) [33, 73]	83% (5/6) [36, 100]
% DOR ≥ 6 months [95% CI] [b]	79% [47, 93]	80% [c] [20, 97]
% DOR ≥ 12 months [95% CI] [b]	60% [28, 81]	80% [c] [20, 97]

Responses were also observed in patients with:

- ROS1 G2032R mutation following ≥2 prior ROS1 TKIs ± chemotherapy, including lorlatinib or repotrectinib
- Other ROS1 resistance mutations, including G1957A, L1982V, S1986F, F2004C/V, G2032K, and D2033N

[a] Patients received zidesamtinib as their first TKI designed with activity against ROS1 G2032R.
[b] Analyses of DOR based on Kaplan-Meier estimates.
[c] One progression event among responders.

Measurable CNS lesions by BICR at baseline

Advanced *ROS1*+ NSCLC Analysis by BICR	Any prior ROS1 TKI ± chemotherapy	Prior crizotinib only ± chemotherapy
IC-ORR, % (n/N) [95% CI]	48% (27/56) [a] [35, 62]	85% (11/13) [55, 98]
IC-CR, % (n/N)	20% (11/56)	54% (7/13)
% IC-DOR ≥ 6 months [95% CI] [b]	79% [56, 91]	91% [c] [51, 99]
% IC-DOR ≥ 12 months [95% CI] [b]	71% [46, 87]	91% [c] [51, 99]

- CNS responses also observed in patients who had received ≥1 prior brain-penetrant TKI, including prior entrectinib, lorlatinib, repotrectinib, or taletrectinib: IC-ORR: 37% (16/43 [a]; [95% CI 23, 53]), including 4 IC-CRs
- No CNS progression was observed among patients who entered the study without brain metastases at baseline per BICR

[a] Includes 2 unconfirmed intracranial partial responses (PR).
[b] Analyses of DOR based on Kaplan-Meier estimates.
[c] One CNS progression event among CNS responders (n=11).

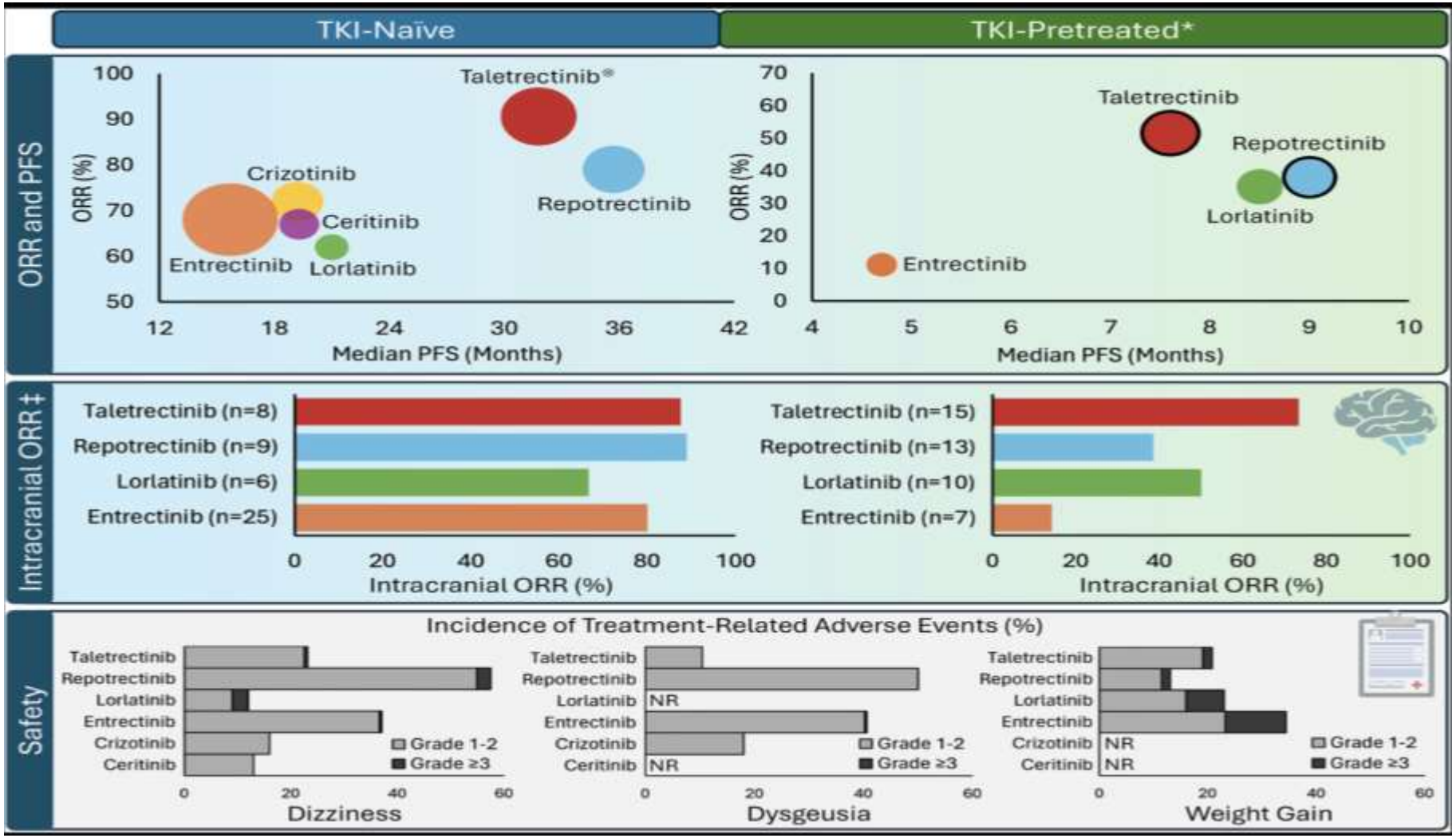
Drilon et al, WCLC 2025

Efficacy of ROS1 TKI in naive patients

ROS1 Inhibitor	ORR (%)	Median PFS	BM Prevalence	Intracranial ORR
Crizotinib (PROFILE 1001)	72%	19.2 mo	Not eligible	-
Crizotinib (OxOnc)	72%	15.9 mo	Not eligible	-
Brigatinib	71%	12.0 mo	11% (3/28)	0%
Entrectinib	68%	15.7 mo	29% (48/168)	52%
Ceritinib	62%	19.3 mo	25% (8/32)	25%
Lorlatinib	62%	21.0 mo	52% (11/21)	64%
Repotrectinib	**79%**	**31 mo**	**13% (9/71)**	**89%**
Taletrectinib	**88%**	**44.6 mo**	**57% (17/130)**	**76.5%**
Zidesamtinib	**89%**	**NR**		**83%**

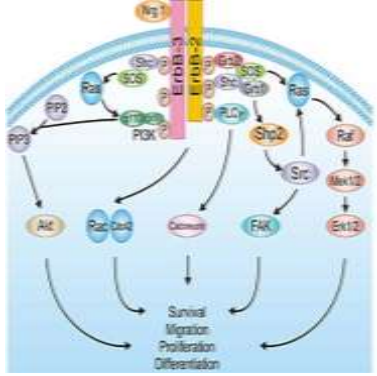
Shaw et al, NEJM 2014; Wu et al, JCO 2018; Niho et al, ESMO Open 2024; Drilon et al, JTO Clin Res Rep 2022; Lin et al, JCO 2017; Shaw et al, Lancet Oncol 2019; Drilon et al, NEJM 2024; Perol et al, JCO 2025; Drilon et al, WCLC 2025

EFFICACY AND TOXICITY of ROS1 TKI INH

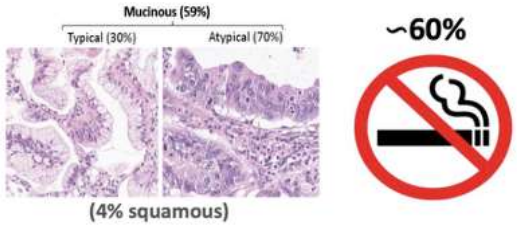


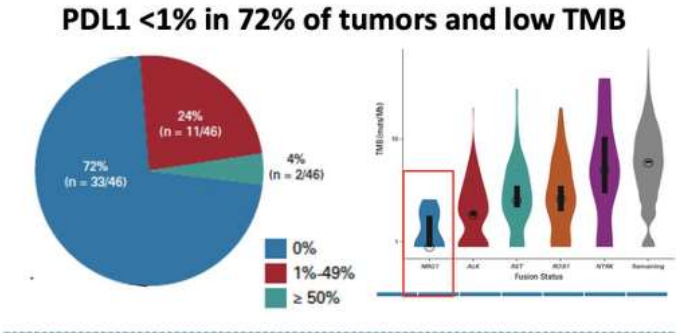
Waliany S, JCO 2024

NRG-1 (Neuregulin-1) Fusions



- Part of a large family of growth factors with EGF-like sequence
- NRG-1 binds to ErbB3 and ErbB4 receptors inducing dimerization and activation
- Actionable genomic driver in various tumor types (overall incidence 0.2%), enriched NSCLC ADC and Kras^{wt} pancreatic cancer
- 8–32% of NSCLC Invasive Mucinous ADC
- DETECTION: NGS/RNA
- Mucinous adenocarcinoma with CD74-NRG-1 fusion expressed phosphorylated ErbB3 protein (pErbB3) → pErbB3 IHC as a screening for NRG-1 fusion ??





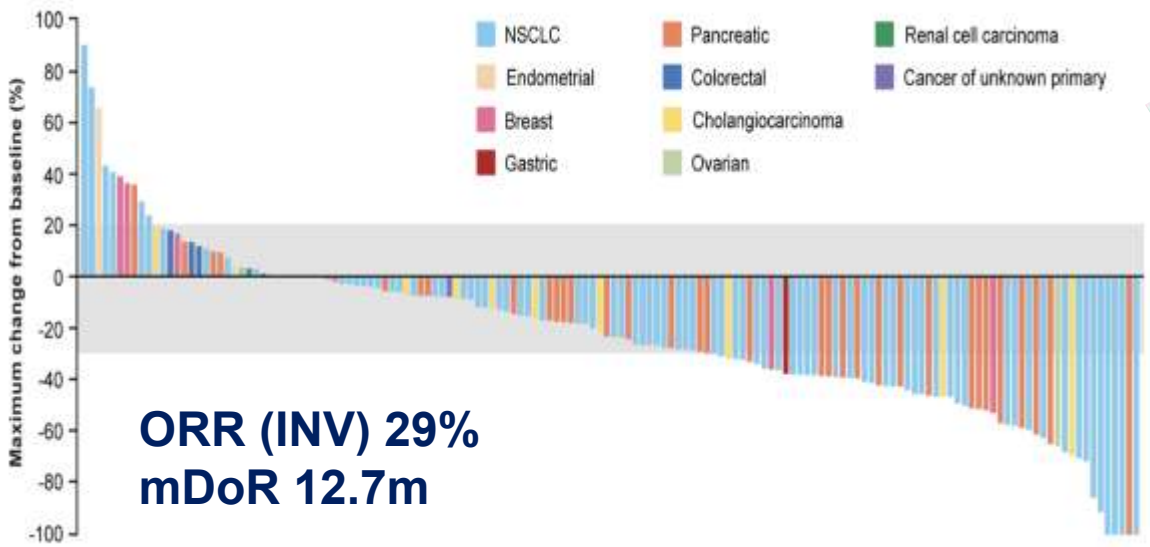
ENERGY1 REGISTRY: CLINICAL OUTCOMES
THE PAST…NRG1-FUSION NSCLC an unmet need

Response, n (%)	Plat-doublet chemo (N = 15)	Taxane-based chemo (N = 7)	Combined chemo/immunotx (N = 9)	Single-agent immunotx (N = 5)	Targeted therapy with afatinib (N = 20)
CR	0	0	0	0	0
PR	2 (13)	1 (14)	0	1 (20)	5 (25)
SD	7 (47)	1 (14)	4 (44)	1 (20)	3 (15)
PD	6 (40)	5 (71)	5 (56)	3 (60)	12 (60)
ORR	2 (13)	1 (14)	0	1 (20)	5 (25)
Survival, mo (95% CI)	**Plat-doublet chemo**	**Taxane-based chemo**	**Combined chemo/immunotx**	**Single-agent immunotx**	**Targeted therapy with afatinib**
Median PFS	5.8 (2.2-9.8)	4.0 (0.8-5.3)	3.3 (1.4-6.3)	3.6 (0.9-undefined)	2.8 (1.9-4.3)

Filetti, Precis Cancer Med 2022; Drilon A, et al J Clin Oncol 2021; Shim WCLC 2023

Zeconotuzumab and Seribantumab

ZENOCOTUZUMAB (anti HER3-HER2 Ab) 750mg iv Q2W
Agnostic Phase II registrational trial
161p, 95 NSCLC



G≥ 3 TRAE 2%, Discontinuation: <1
Most TRAE were grade 1 and 2, commonly diarrhea, fatigue

CRESTONE
Phase II SERIBANTUMAB (HER3 mAB IgG2) in NRG fusion Cancers

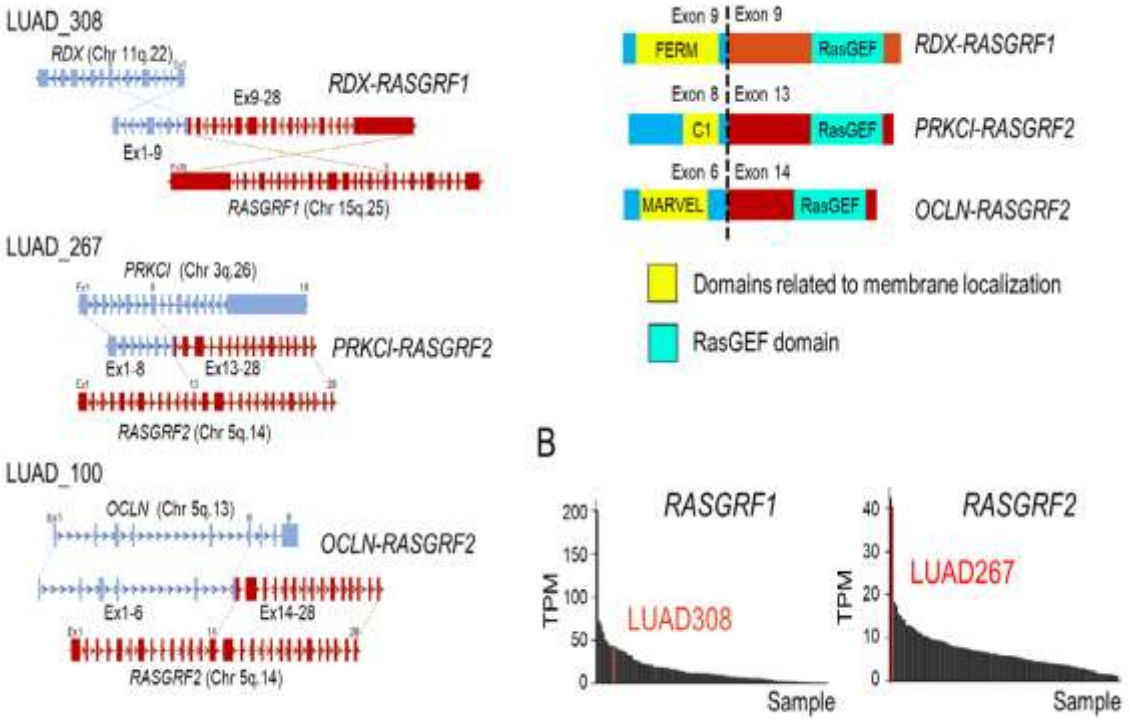
G≥ 3 TRAE: 6%. Discontinuation 0%
Most TRAE were grade 1 and 2, commonly diarrhea, fatigue, rash.

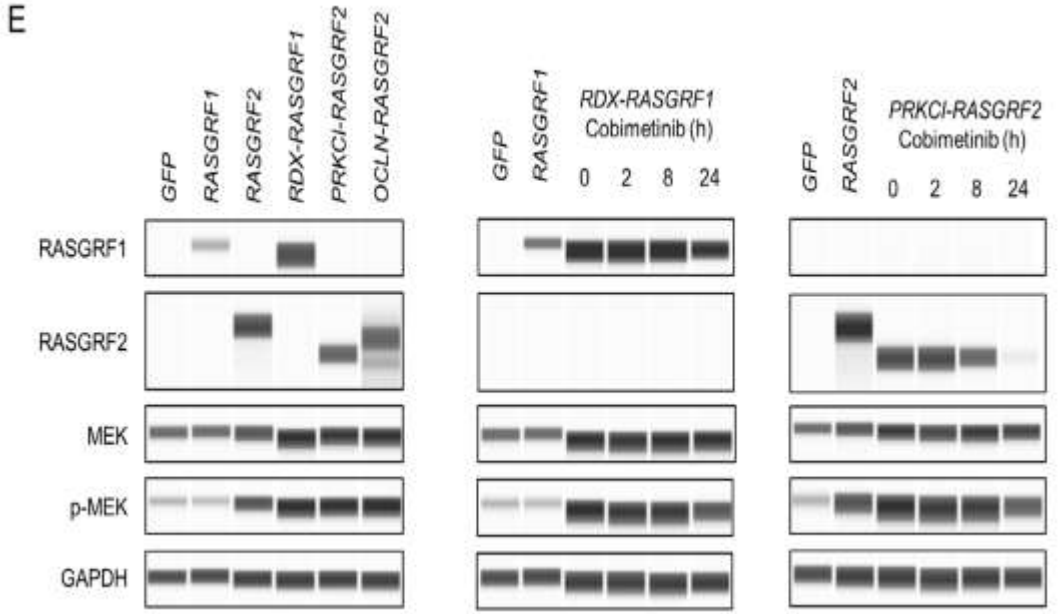
Schram et al. NEJM 2025; Carricosa et al, ASCO 2022

ORIGINAL ARTICLE OPEN ACCESS

Discovery of Novel *RASGRF2* Fusions as a Therapeutic Target in Lung Adenocarcinoma of Never or Light Smokers

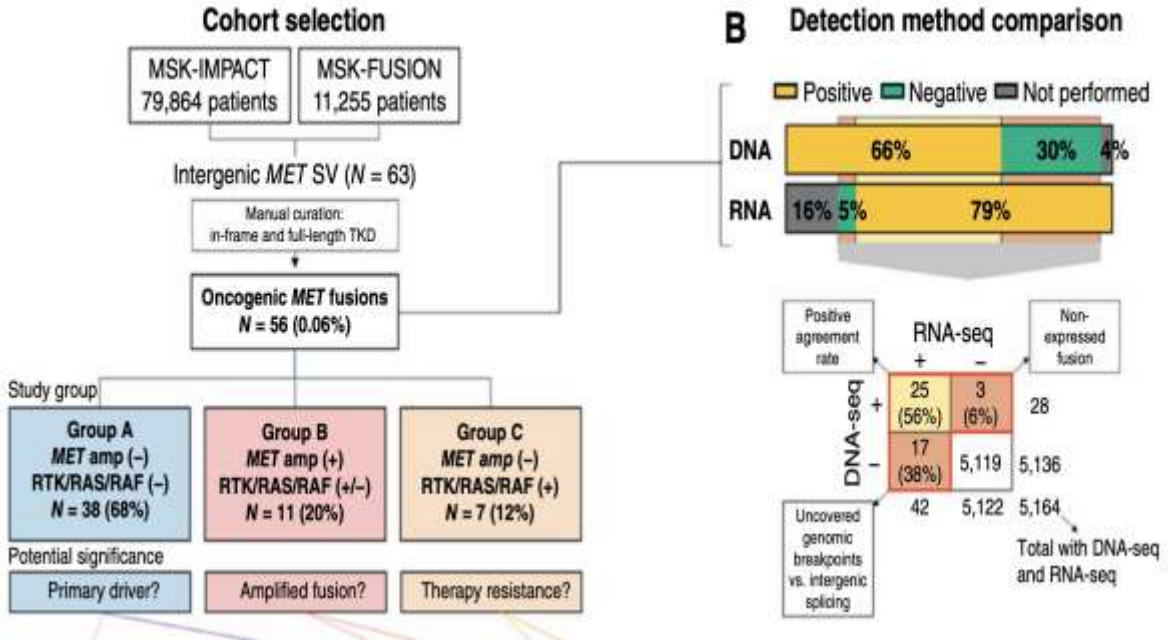
Yuki Terashima[1,2] | Soohwan Park[1,3] | Hiroshi Ikeuchi[1,3] | Takuo Hayashi[4] | Shinya Kojima[1] | Toshihide Ueno[1] | Masachika Ikegami[1] | Rina Kitada[1] | Yoshiyuki Suehara[1] | Shinya Tanaka[2,5] | Kenji Suzuki[3] | Hiroyuki Mano[1] | Kazuya Takamochi[3] | Shinji Kohsaka[1]

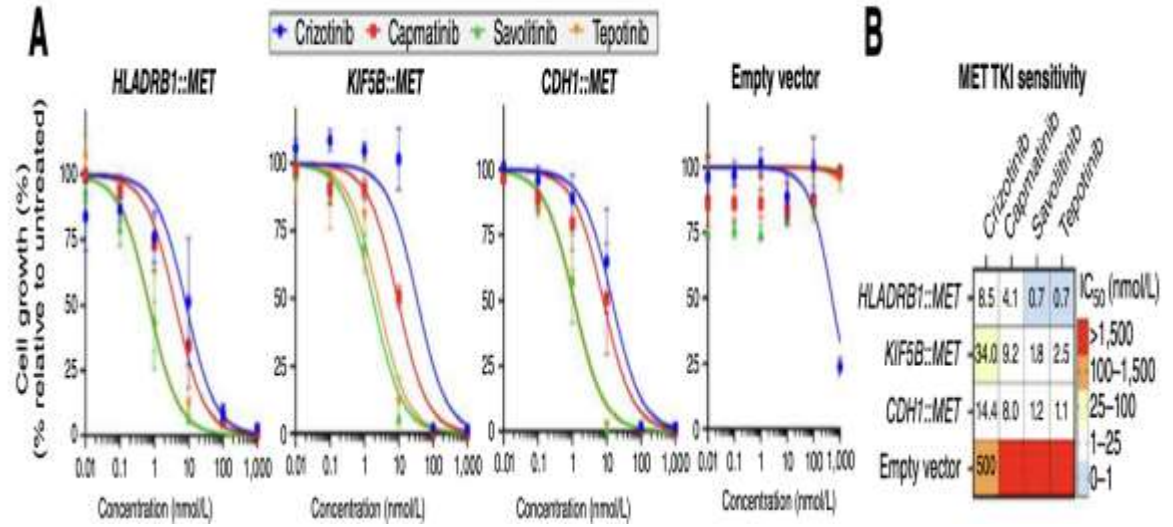




Tersshima et al, Cancer Sci 2025

Pan-Cancer Analysis of Oncogenic MET Fusions Reveals Distinct Pathogenomic Subsets with Differential Sensitivity to MET-Targeted Therapy





Febres-Aldana et al. Can Discov 2025

Knowledge, Testing, Access….
EVEN IF YOU ARE RARE, THE LAST, THE END OF THE WORLD...

- Relevant number of oncogenic fusions with SCAT I in NSCLC

- NGS-RNA based testing-blood based are the best choice
- 4 fusions with targeted therapy approved
- Moving more potent new TKI to first line settting…sequencing approach, toxicities…
- How are we going to treat these patients in early/ locally advanced disease?
- Undoubtedly managing these tumors will require expertise, comprehensive analytical approach and funding.
- Be aware of unusual toxicities
- Resistance…always resistance

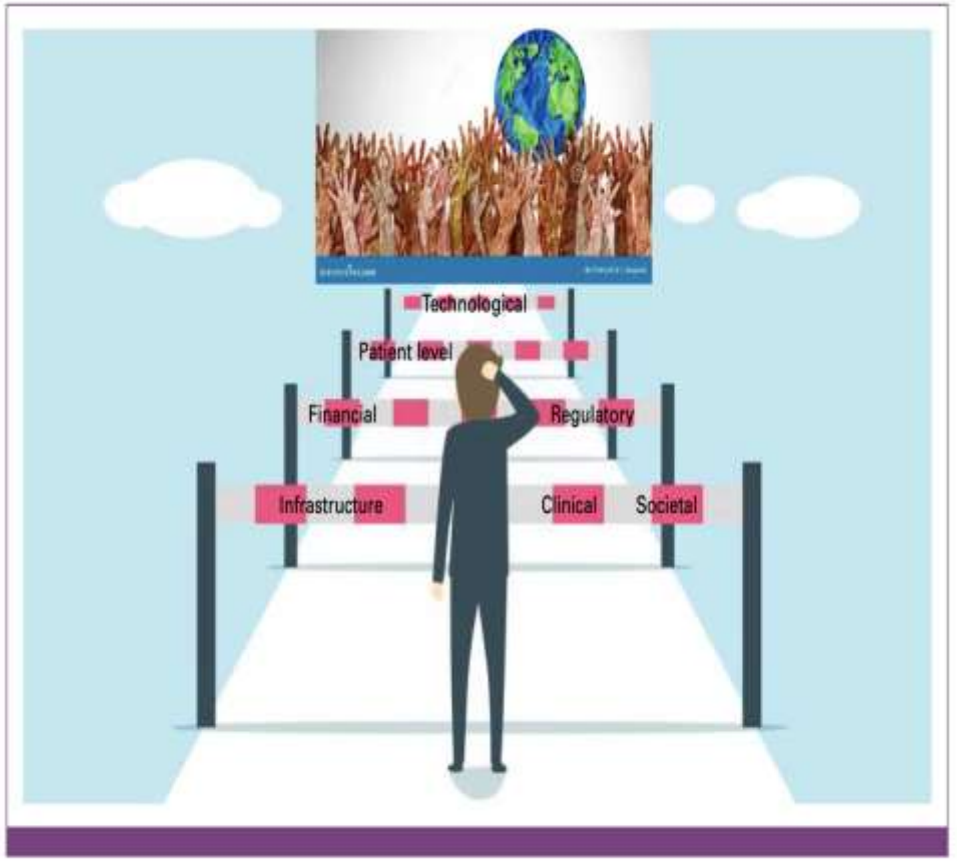


FIG 1. Barriers to globalizing precision oncology.

Horgan et al. JCO Global Oncol 2025

16th CONGRESS Lung ON CANCER

BARCELONA 27 / 28 NOVEMBER 2025

THANK YOU

GECP lung cancer research