Metastatic Lung Cancer Targeted Therapy Update

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Agenda

- Increasing Targets in NSCLC

- mEGFR and OSI resistance
  - MET inhibition (Chrysalis 2, Insight 2)
  - Chemo+ (AGAIN, Keynote 789)

- Antibody Drug Conjugates (ADC)
  - Her2 mutation (Destiny)
  - TROP2 (Tropion-Lung02)

- Novel Inhibitors (YK-029A, APG-2449, SI B-001)

- BRAF 4600E (Pharos)

- KRAS G12C + Chemo (Scarlet)

Lung Cancer Targets

EGFR **exon 19 deletion or exon 21 L858R mutation positive**
EGFR **S768I, L861Q, and/or G719X mutation positive**
EGFR **exon 20 insertion mutation positive**
KRAS **G12C mutation positive**
ALK **rearrangement positive**
ROS1 **rearrangement positive**
BRAF **V600E mutation positive**
NTRK1/2/3 **gene fusion positive**
METex14 **skipping mutation positive**
RET **rearrangement positive**
ERBB2 (HER2) **mutation positive**
PD-L1 ≥1% and negative for actionable molecular biomarkers above
PD-L1 <1% and negative for actionable molecular biomarkers above

MOLECULAR AND BIOMARKER-DIRECTED THERAPY FOR ADVANCED OR METASTATIC DISEASE[a,b]

***EGFR* Exon 19 Deletion or Exon 21 L858R**
- First-line therapy
  - Afatinib[1]
  - Erlotinib[2]
  - Dacomitinib[3]
  - Gefitinib[4,5]
  - Osimertinib[6]
  - Erlotinib + ramucirumab[7]
  - Erlotinib + bevacizumab[c] (nonsquamous)[8]
- Subsequent therapy
  - Osimertinib[9]

***EGFR* S768I, L861Q, and/or G719X**
- First-line therapy
  - Afatinib[1,10]
  - Erlotinib[2]
  - Dacomitinib[3]
  - Gefitinib[4,5]
  - Osimertinib[6,11]
- Subsequent therapy
  - Osimertinib[9]

***EGFR* Exon 20 Insertion Mutation**
- Subsequent therapy
  - Amivantamab-vmjw[12]
  - Mobocertinib[13]

***KRAS* G12C Mutation**
- Subsequent therapy
  - Sotorasib[14]
  - Adagrasib[15]

***ALK* Rearrangement**
- First-line therapy
  - Alectinib[16,17]
  - Brigatinib[18]
  - Ceritinib[19]
  - Crizotinib[16,20]
  - Lorlatinib[21]
- Subsequent therapy
  - Alectinib[22,23]
  - Brigatinib[24]
  - Ceritinib[25]
  - Lorlatinib[26]

***ROS1* Rearrangement**
- First-line therapy
  - Ceritinib[27,28]
  - Crizotinib[29]
  - Entrectinib[30]
- Subsequent therapy
  - Lorlatinib[31]
  - Entrectinib[30]

***BRAF* V600E Mutation**
- First-line therapy
  - Dabrafenib/trametinib[32]
  - Dabrafenib[32]
  - Vemurafenib
- Subsequent therapy
  - Dabrafenib/trametinib[33,34]

***NTRK1/2/3* Gene Fusion**
- First-line/Subsequent therapy
  - Larotrectinib[35]
  - Entrectinib[36]

***MET* Exon 14 Skipping Mutation**
- First-line therapy/Subsequent therapy
  - Capmatinib[37]
  - Crizotinib[38]
  - Tepotinib[39]

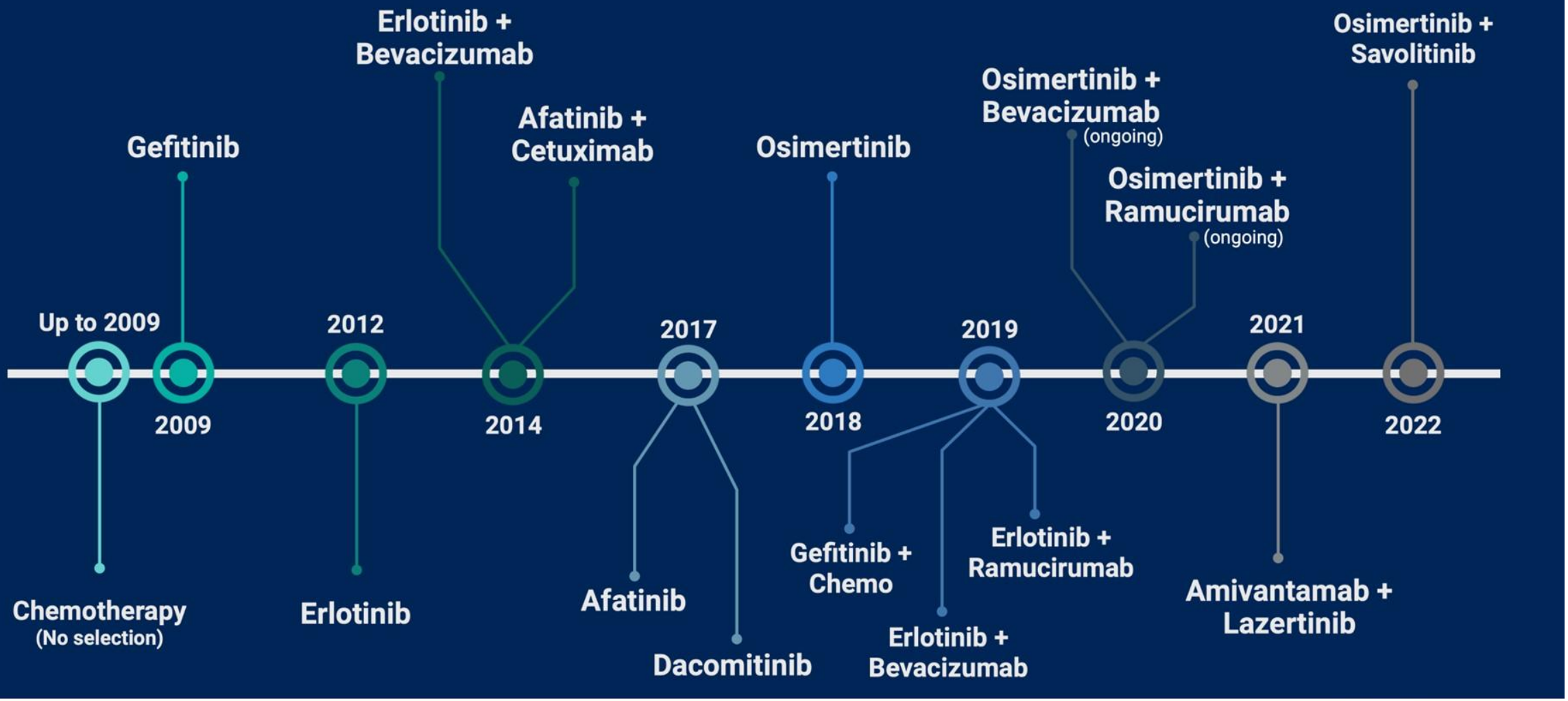
***RET* Rearrangement**
- First-line therapy/Subsequent therapy
  - Selpercatinib[40]
  - Pralsetinib[41]
  - Cabozantinib[42,43]

***ERBB2 (HER2)* Mutation**
- Subsequent therapy
  - Fam-trastuzumab deruxtecan-nxki[44]
  - Ado-trastuzumab emtansine[45]

PD-L1 ≥50% First-line Therapy

PD-L1 ≥1-49% First-line Therapy

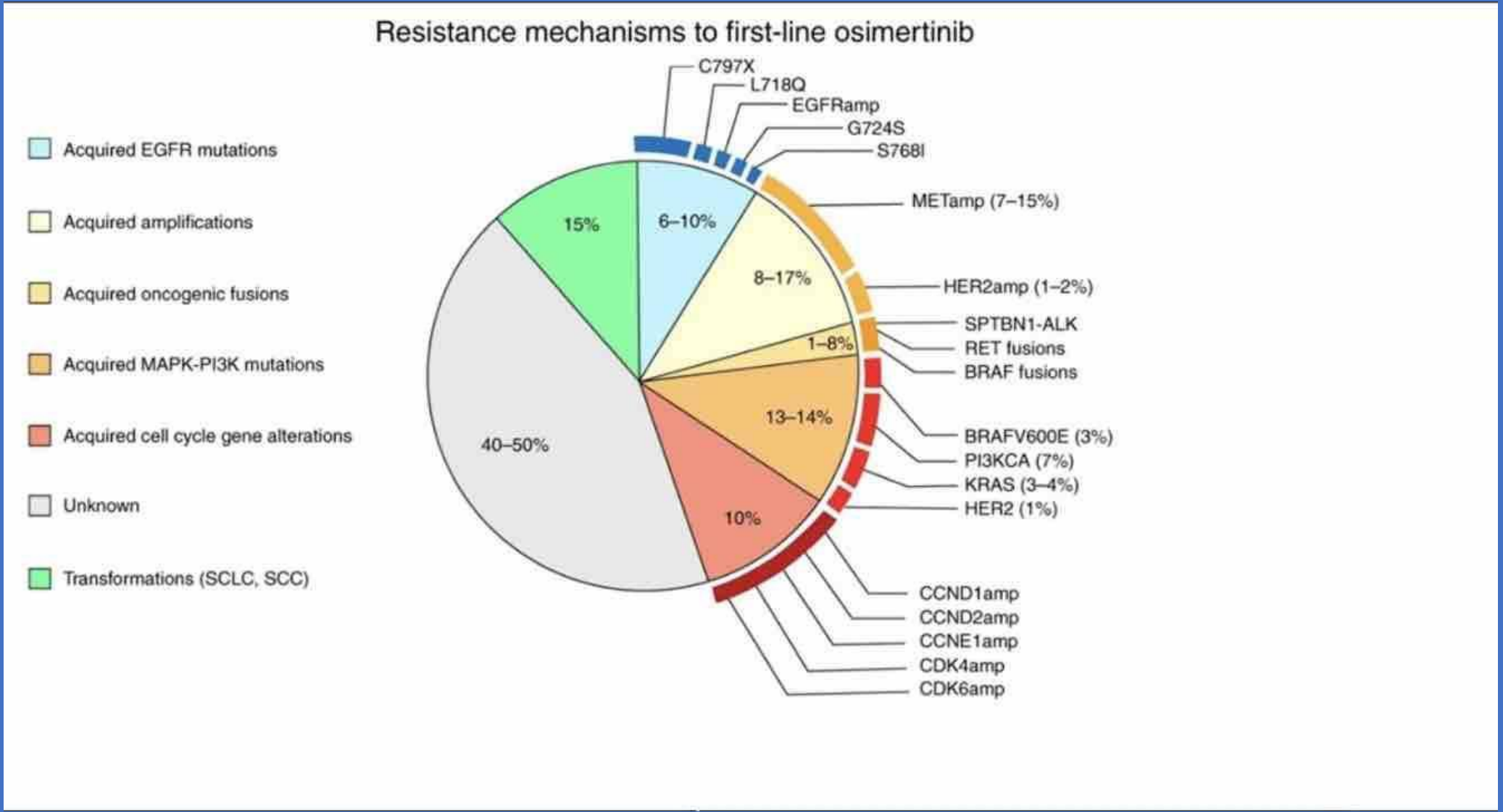
EGFR Mutations *Evolution of Treatment* in advanced NSCLC



PRESENTED BY: Carlos G. M. Ferreira, MD, PhD

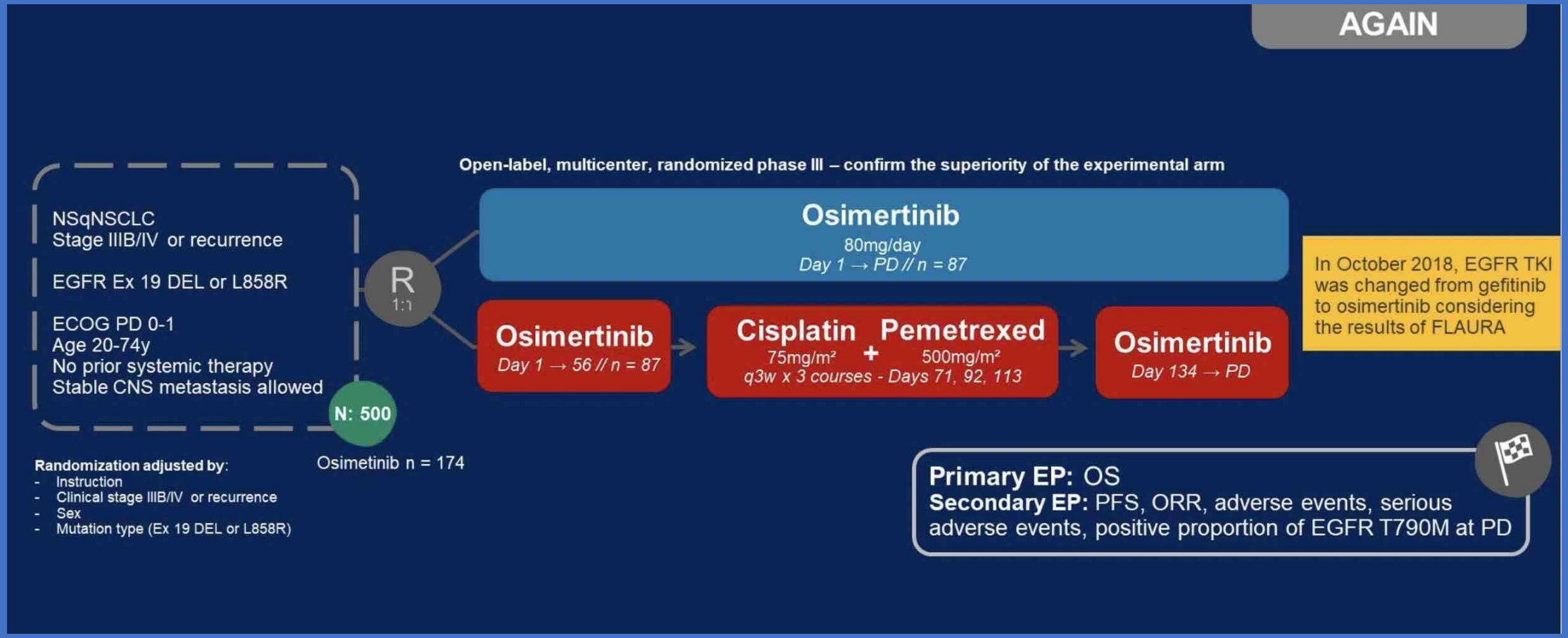
Resistance to Osimertinib in mEGFR NSCLC

Schoenfeld Clin Cancer Res 2020, 26, 2654



AGAIN Study combining chemo and Osi

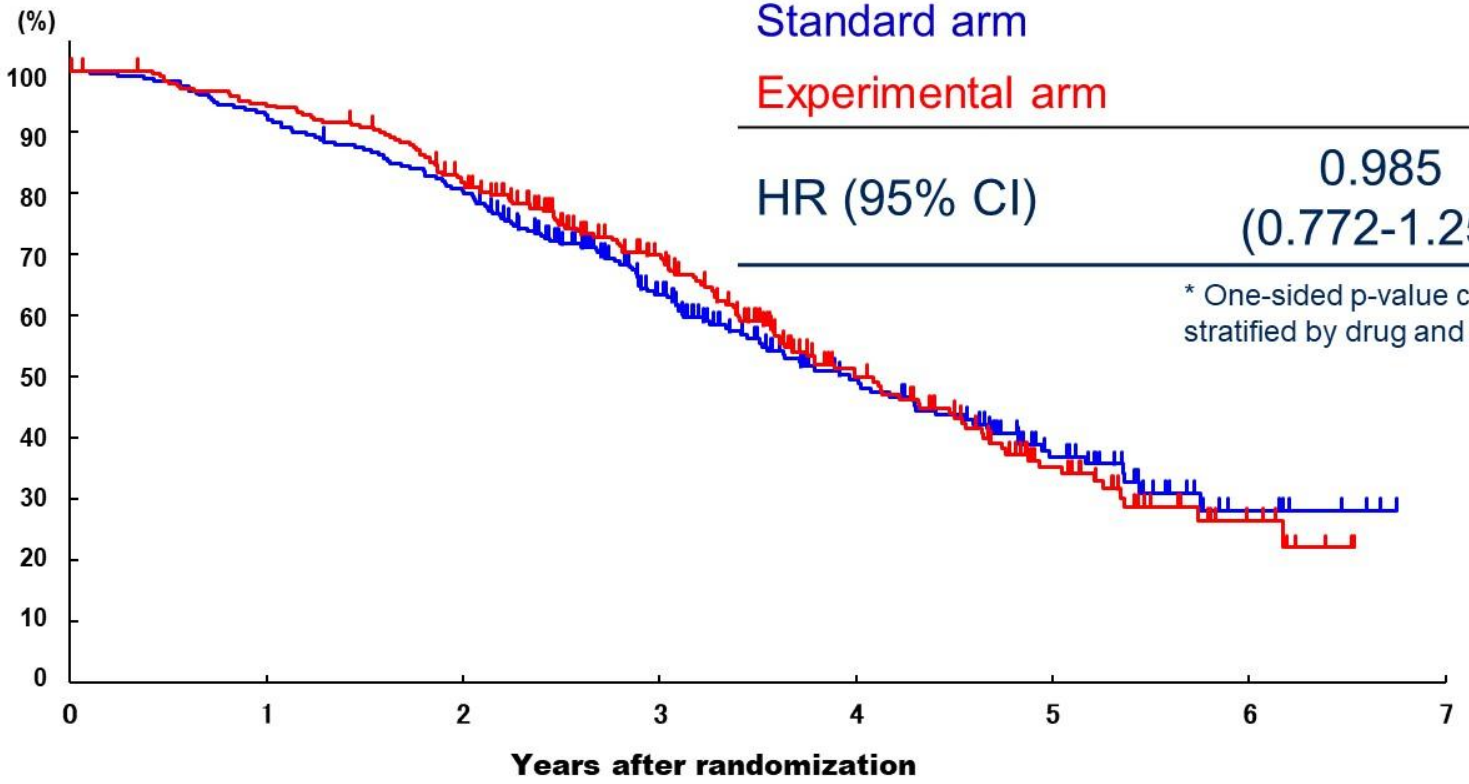
JCOG1404/WJOG8214L



Primary endpoint: Overall survival (ITT)

	Median OS (95% CI), months	
Standard arm	48.0	(40.8-55.2)
Experimental arm	48.0	(43.2-54.0)
HR (95% CI)	0.985 (0.772-1.257)	p=0.4496*

* One-sided p-value calculated from log-rank test stratified by drug and sex and *EGFR* mutation type.

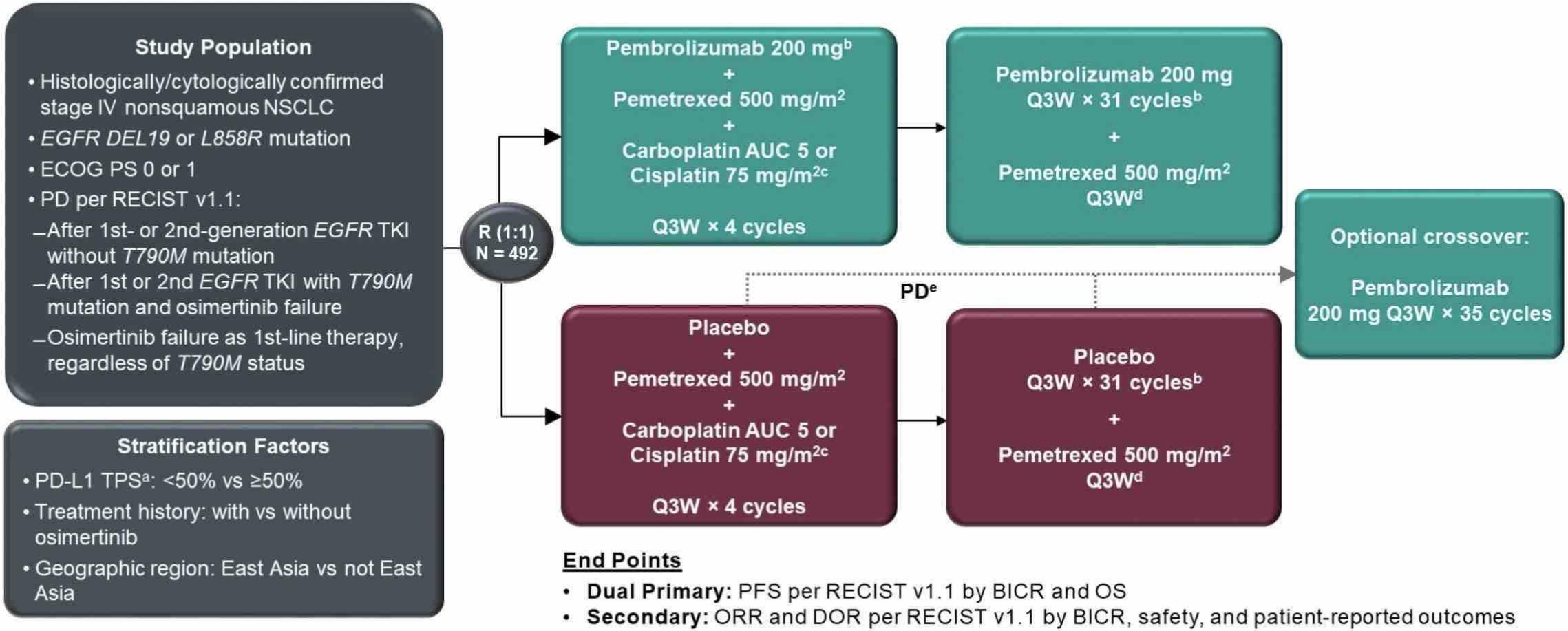


Standard arm	250	232	200	124	70	36	7	0
Experimental arm	251	234	198	134	70	33	8	0

Overcoming EGFR TKI Resistance

Chemo+/-IO in TKI refractory mEGFR NSCLC

KEYNOTE-789: Phase 3 Randomized Study (NCT03515837)



End Points

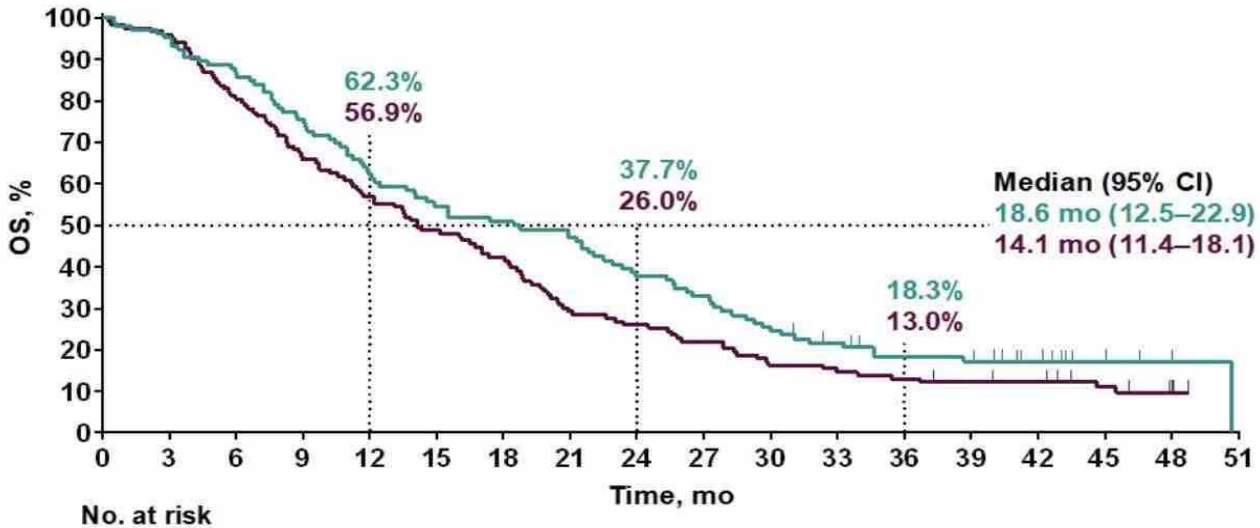
- **Dual Primary:** PFS per RECIST v1.1 by BICR and OS
- **Secondary:** ORR and DOR per RECIST v1.1 by BICR, safety, and patient-reported outcomes

Chemo+/-IO in TKI refractory mEGFR NSCLC, KN 789

Overall Survival in PD-L1 TPS ≥1% and <1% at FA

PD-L1 TPS ≥1%

	Events, n (%)	HR (95% CI)
Pembrolizumab + chemo	88 (83.0)	0.77 (0.58–1.02)
Placebo + chemo	110 (89.4)	

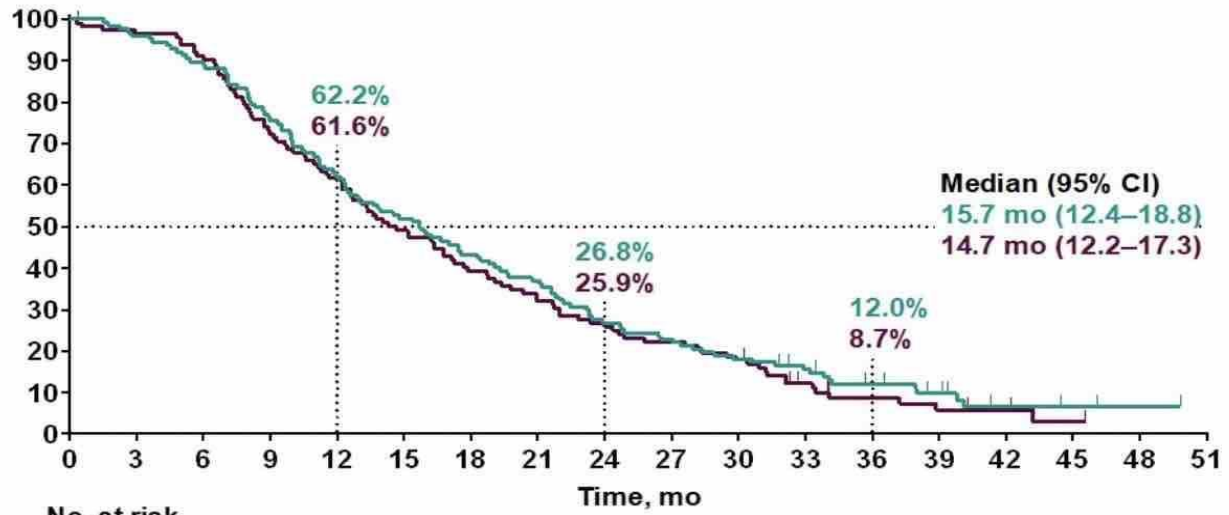


No. at risk

106	101	92	80	66	58	54	50	40	35	26	21	16	15	10	5	2	0
123	118	99	81	70	60	52	36	32	27	20	18	16	14	13	9	3	0

PD-L1 TPS <1%

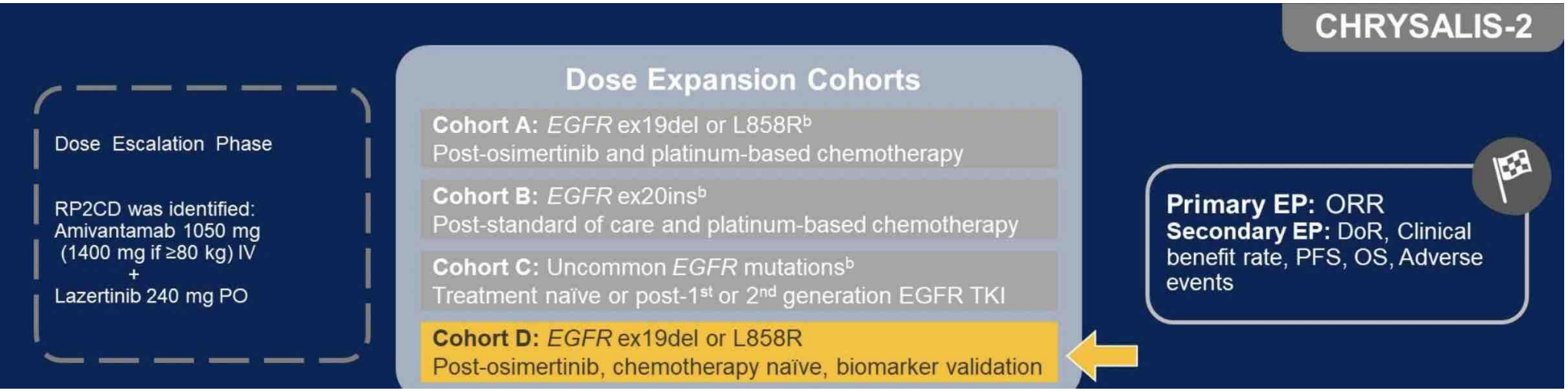
	Events, n (%)	HR (95% CI)
Pembrolizumab + chemo	115 (90.6)	0.91 (0.70–1.19)
Placebo + chemo	104 (92.0)	

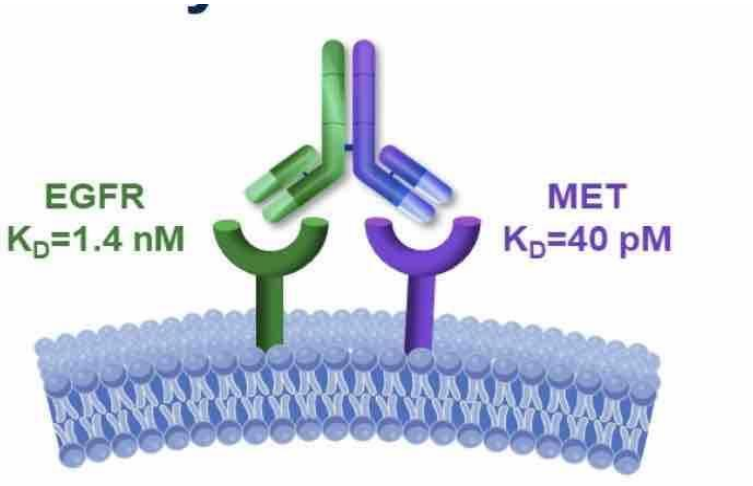


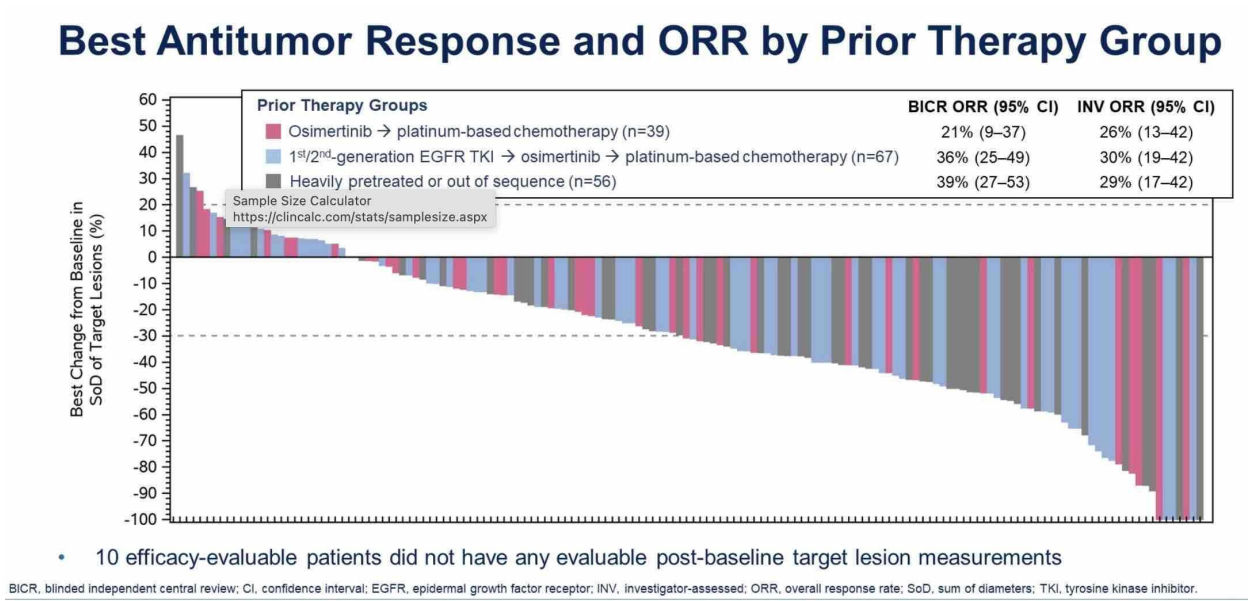
No. at risk

127	122	114	96	79	66	55	47	34	29	23	18	12	8	3	2	1	0
113	108	102	81	69	55	44	36	29	25	20	11	6	4	3	1	0	0

Chrysalis 2 (Phase 1B), Amivantanab (BITE) and Lazertinib (3rd gen. EGFR TKI) post Osimertinib in mEGFR NSCLC
ASCO 2022, C. Shu, #9006



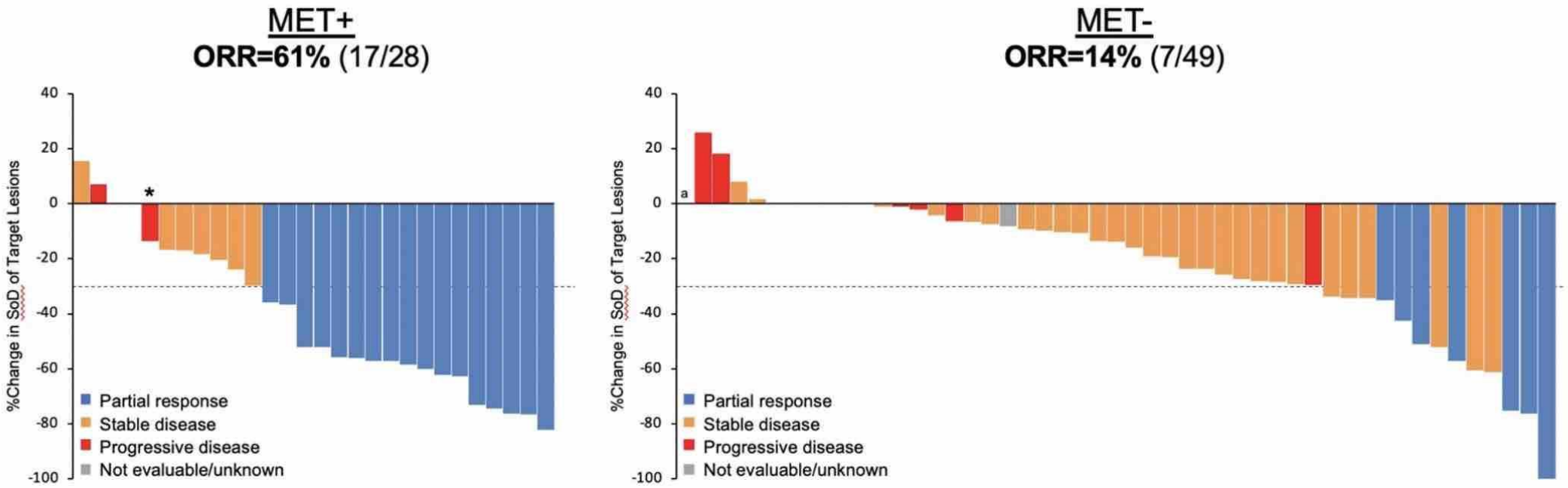




MET IHC Expression 3+ a predictive biomarker?
B. Besse, ASCO 2023

CHRYSALIS-2

MET+ by IHC Enriched Response to Amivantamab Plus Lazertinib



- A total of 28 of 77 (36%) patients[b] had MET 3+ staining on ≥25% of tumor cells (MET+)
- *MET* amplification was detected by NGS of ctDNA in 1 patient (see asterisk)

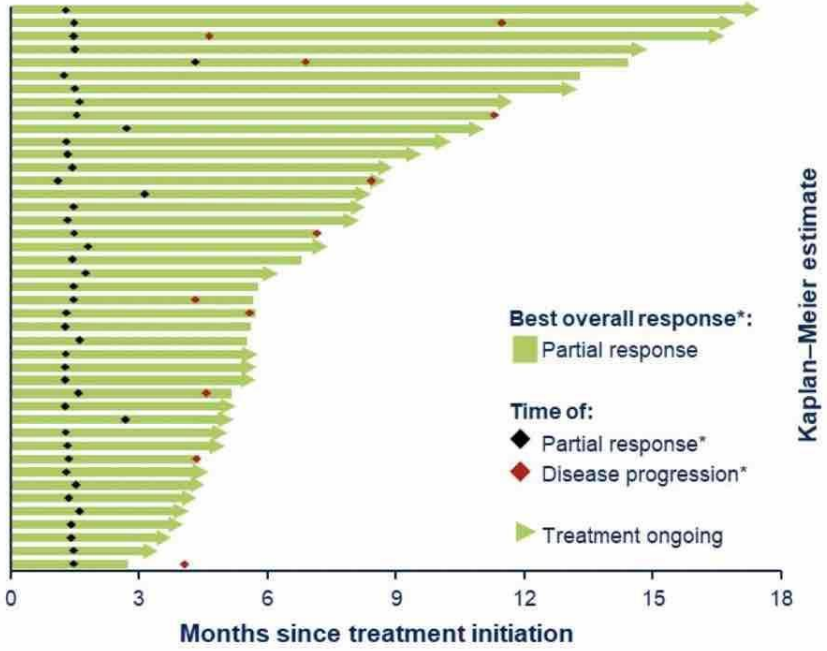
INSIGHT 2

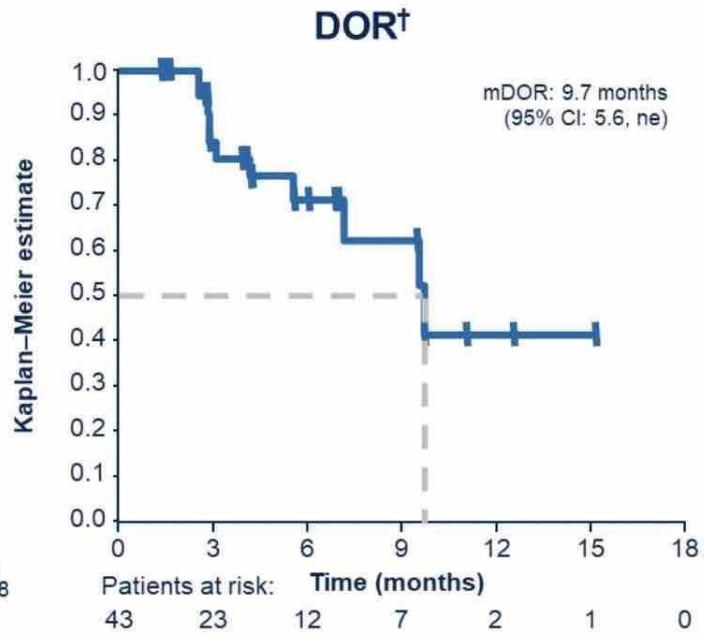
Tepotinib and OSI for FISH amplified MET mEGFR

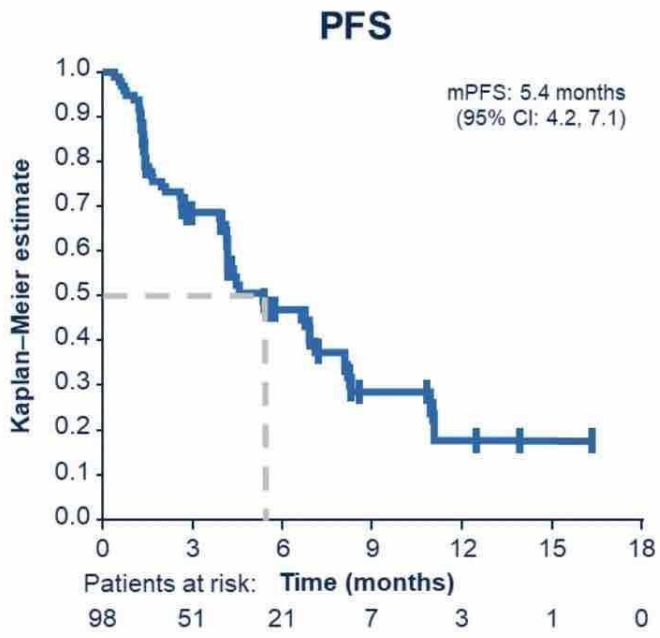
D. Tan, ASCO 2023

INSIGHT 2: Efficacy TBx FISH+

- Responses mostly occurred within 6 weeks, median DOR was 9.7 months (95% CI: 5.6, ne) and median PFS was 5.4 months (95% CI: 4.2, 7.1)
- Treatment was ongoing in 42 patients







EGFR Exon 20 Insertion

→

New players

Sunvocertinib (WUKONG Study)

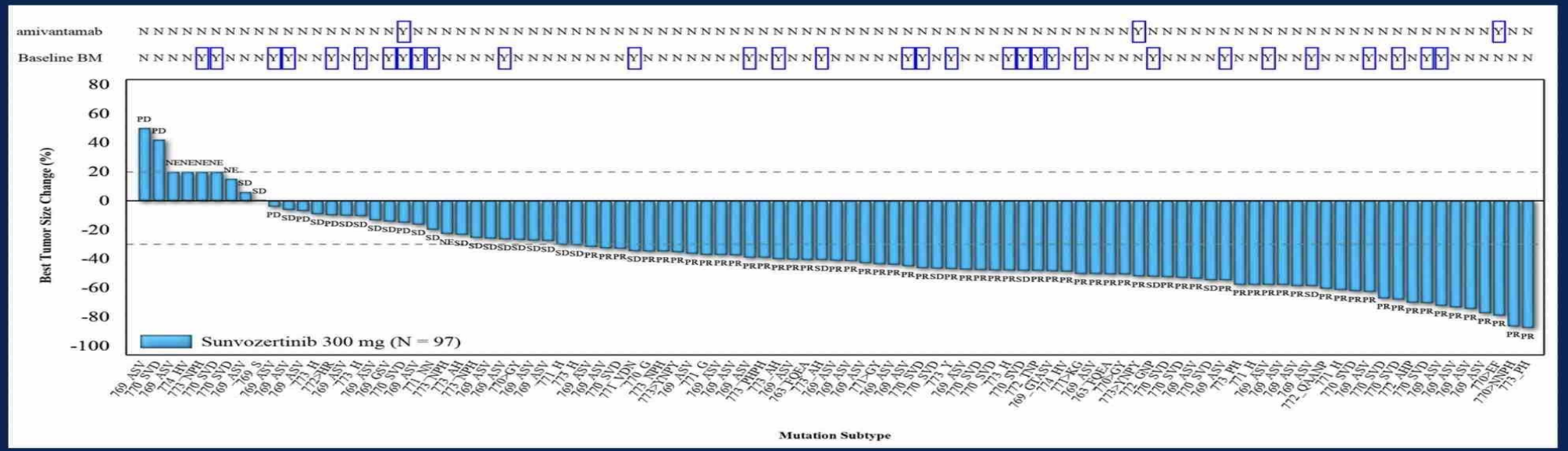
and YK-029A

2023 ASCO ANNUAL MEETING

Sunvozertinib for the Treatment of NSCLC with *EGFR* Exon20 Insertion Mutations: the First Pivotal Study Results

Mengzhao Wang[1], Yun Fan[2], Meili Sun[3], Yongsheng Wang[4], Yanqiu Zhao[5], Bo Jin[6] Ying Hu[7], Zhigang Han[8], Xia Song[9], Anwen Liu[10], Kejing Tang[11], Cuimin Ding[12], Li Liang[13], Lin Wu[14], Junzhen Gao[15], Jianghong Wang[16], Ying Cheng[17], Jianying Zhou[18] Yong He[19], Li Zheng[20]

Target Tumor Size Change per IRC Assessment



BM, brain metastasis

- Tumor shrinkage was observed in > 90% of subjects with sunvozertinib treatment.
- Tumor response was observed in patients with baseline brain metastasis or post amivantamab treatment.

Efficacy

	Mobocertinib[1] (N=114)	Amivantamab[2] (N=81)	Sunvozertinib (DZD9008) (N=97) WUKONG6[3]
Investigator assessed			
ORR, %	35%	36%	46.4%
Disease control rate, %	78%	73%	
Duration of response, mos	11.2 mo	-	
IRC assessed (95% CI)			
ORR, % (95% CI)	28% (20-37%)	40% (29-51%)	60.8% (50.4-70.6%)
Disease control rate, %	78%	74%	87.6%
Duration of response, months	17.5 mo	11.1 mo	64.4% responding at median fup of 5.6 mo.
PFS, months	7.3 mo	8.3 mo	-
Brain Mets, ORR (N=)	-	-	44% (N=25)[4]

1. Zhou C. et al. *JAMA Oncol.* 2021 Oct 14;e214761. 2. Park K, et al. *J Clin Oncol.* 2021;39:3391-3404. 3. M. Wang et al ASCO 2023. ABS7 9002. 4. L. Bazhenoza et al NACLC 2022.

Safety

EGFR Exon 20 Tx	Trial	Diarrhea	Rash	Other Major Notable
Amivantamab	CHRYSALIS[2]	12% (2% G3+)	86% (4% G3+)	Infusion-related reaction 66% (8% G3+), Paronychia
Mobocertinib	EXCLAIM[1]	93% (16% G3+)	45% (0% G3+)	lipase, amylase, other GI, lipase, amylase elevation
Sunvozertinib	WUKONG6[4]	67.3% (7.7% G3+)	53.8% (1% G3+)	CPK Elevation (57.7%, 17.3% G3+)

*WUKONG 1,2,6 pooled at 300 mg dose [5]

Other EGFR Exon 20 ins TKI with Putative CNS Penetration in Development

- TAS6417 (CLN-081)
- Blu-451
- Oric-114
- Furmonertinib

New Exon 20 ins Inhibitors

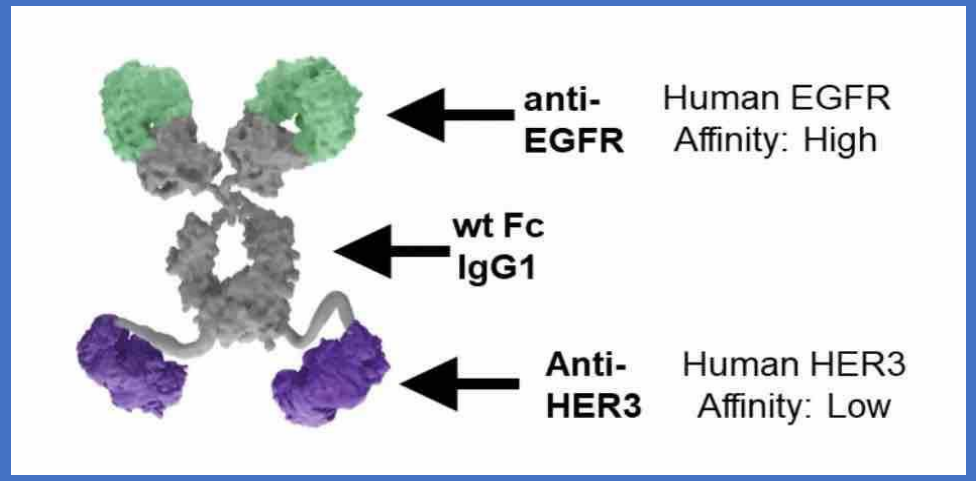
	Mobocertinib	Amivantamab	Sunvozertinib DZD9008	Zipalertinib CLN081/TAS6417	YK-029A
	FDA accelerated approval 2021	FDA accelerated approval 2021	FDA BTD	FDA BTD	
		Phase 3 PAPLLON	1L phase 3 trial ongoing	1L phase 3 trial ongoing	1L phase 3 trial ongoing (vs. chemo)
ORR	28% (post-chemo)	40% (post-chemo)	61% (2L) 78% (treatment-naïve)	41%	**73% (Treatment-Naïve)**
PFS (months)	7.3	8.3	NR	12	9.3
DoR (months)	17.5	11.1	NR	NR	7.5
CNS activities	No	No	Not known	Not known	Not known
Common Toxicities All grade (G3+)	Diarrhea, 91% (21%) Rash, 45% (0%)	Diarrhea 11% (2%) Rash 86% (4%)	Diarrhea, 59% (6.5%) Rash 39% (1%)	Diarrhea, 30% (3%) Rash, 80% (1%)	Diarrhea 46% (14.6%) Rash 32%(0%) Mucositis (4.9%)
	QTc prolongation (Black Box)	Infusion reaction (66%)	CPK elevation 31%		
Dose discontinuation	17%	10%	NR	5%	4.9%
Dose reduction	25%	13%	NR	13%	22%

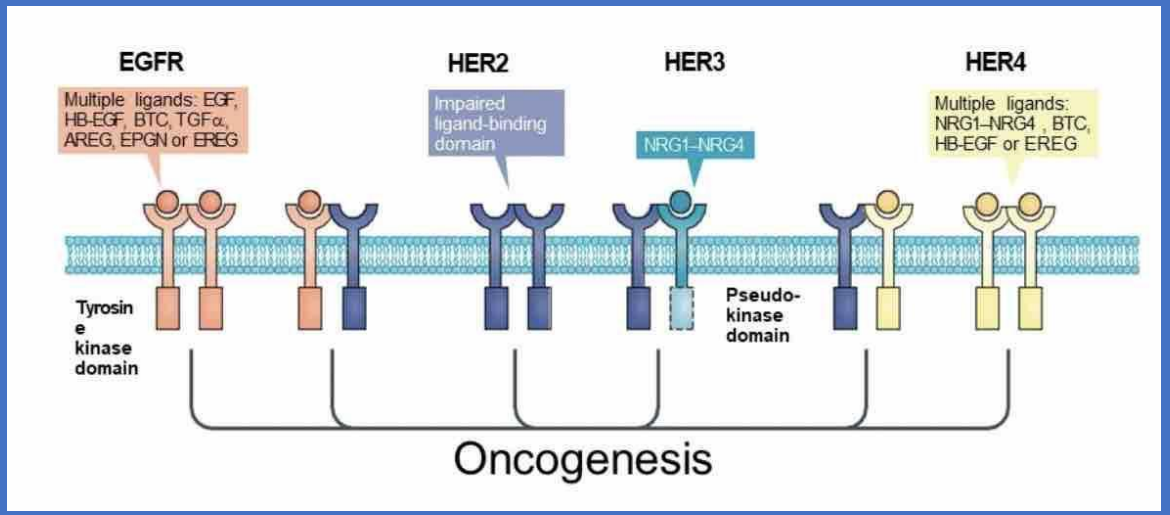
Blu-451, ORIC-114, BAYER7088, PLB004, furmonertinib, and many others in clinical development

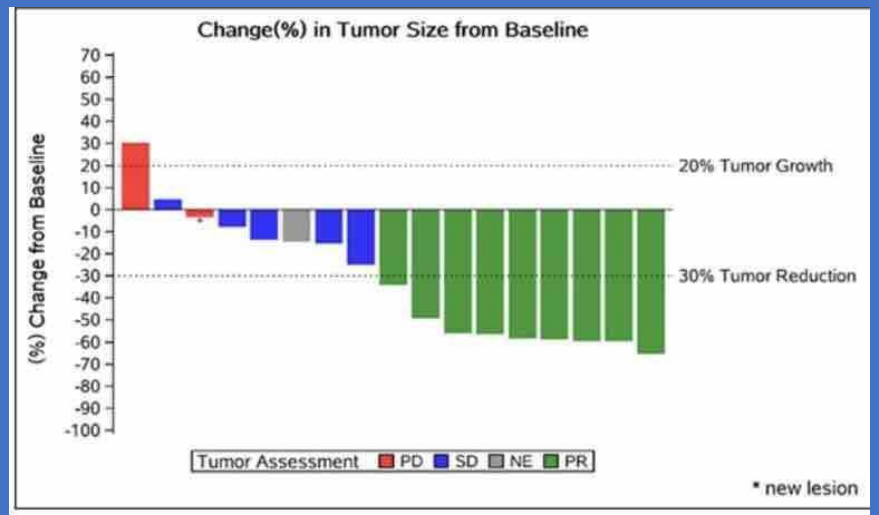
Zhou C et al JAMA Oncol 2021, Park K et al JCO 2021,* Bazhenova LA *et al NALC 2022, Yu H et al ASCO 2022, Wang et al ASCO 2023, Xu et al ASCO 2023, Duan J et al ASCO 2023

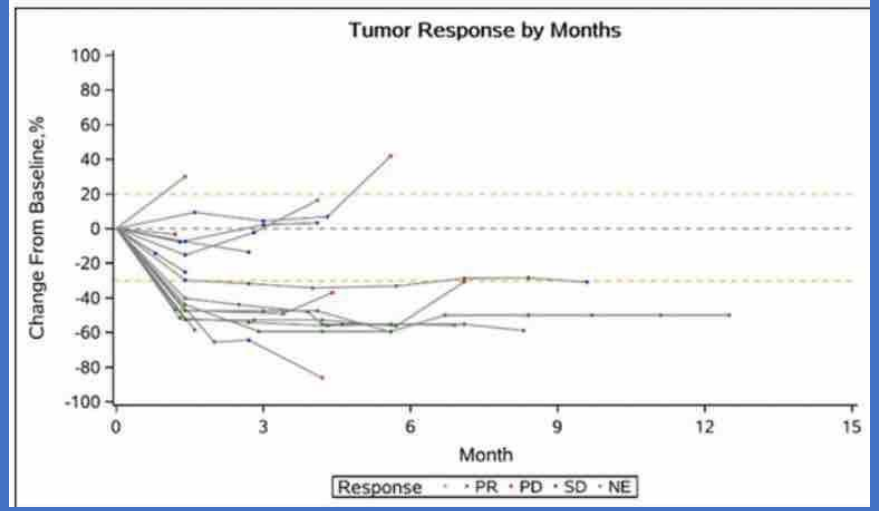
Targeting EGFR Wild Type

SI-B001: A new BITE EGFR/HER3 AB in wt EGFR/ALK ASCO 2023, Zhao , #2025









Targeting ALK

New ALK inhibitors

	Ceritinib	Alectinib	Brigatinib	Ensartinib	Lorlatinib (3G)	APG-2449
ORR	72%	81-91%	74%	74%	77%	**78% (Treatment-Naïve)**
PFS (months)	16.6	25.7-34.1	24.0	25.8	NR (f/u 37m)	NR
CNS ORR	73%	81-94%	78%	64%	83%	Promising
Common Toxicities All grade (G3+)	Nausea 66-83% ALT/AST 20-45%	Nausea 10-21% ALT/AST 12-28% Constipation 24-33%	Nausea 40-55% ALT/AST 15%	Nausea 22% ALT/AST 48% Rash 68% Elevated creatinine 14%	Nausea 15% ALT/AST 17% Edema 55% Hyperlipidemia 70% Cognitive 21%	Nausea 27% (0.7%) ALT/AST 40% (3%) Elevated Creatine (46%)
Dose discontinuation	5% (80% dose interruption)	11-13%	8-13%	9%	7%	NR

NVL-655 and other ALK inhibitors in clinical development

FAK inhibitor defactinib (VS6063), BI 853520, GSK2256098 in clinical development with combination strategies.

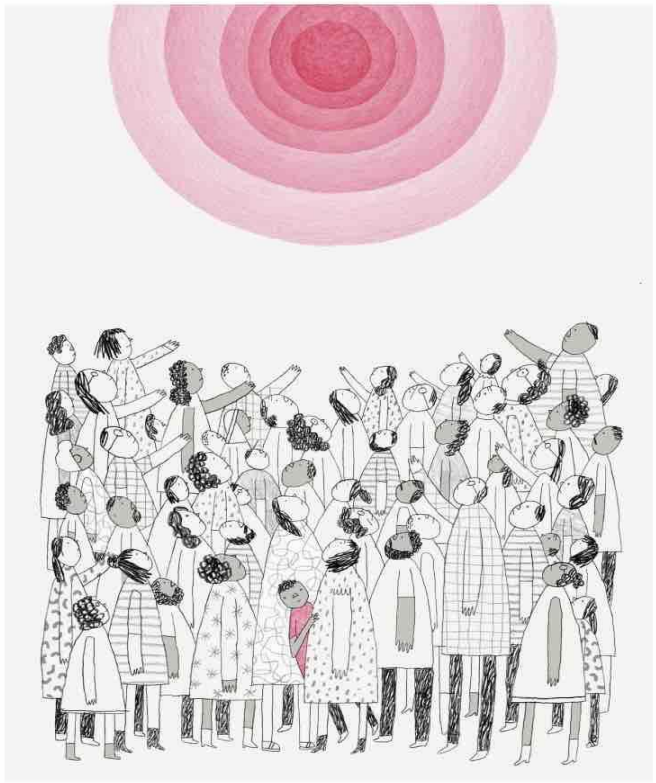
Soria JC et al Lancet 2017, Hida T et al Lancet 2017, Peters S. e t al NEJM 2017, Mok T e t al Ann Oncol 2020, Zhou et al Lancet Respir Med. 2019, Camidge DR, et al. J Thorac Oncol, Camidge DR, et al. J Clin Oncol. 2020, Horn L, et al. JAMA Oncol. 2021, Shaw AT, et al. NEJM 2020, Solomon BJ, et al. Lancet Resp Med. 2022, Ou SH et al unpublished data

Antibody Drug Conjugates (ADC) in Lung Cancer

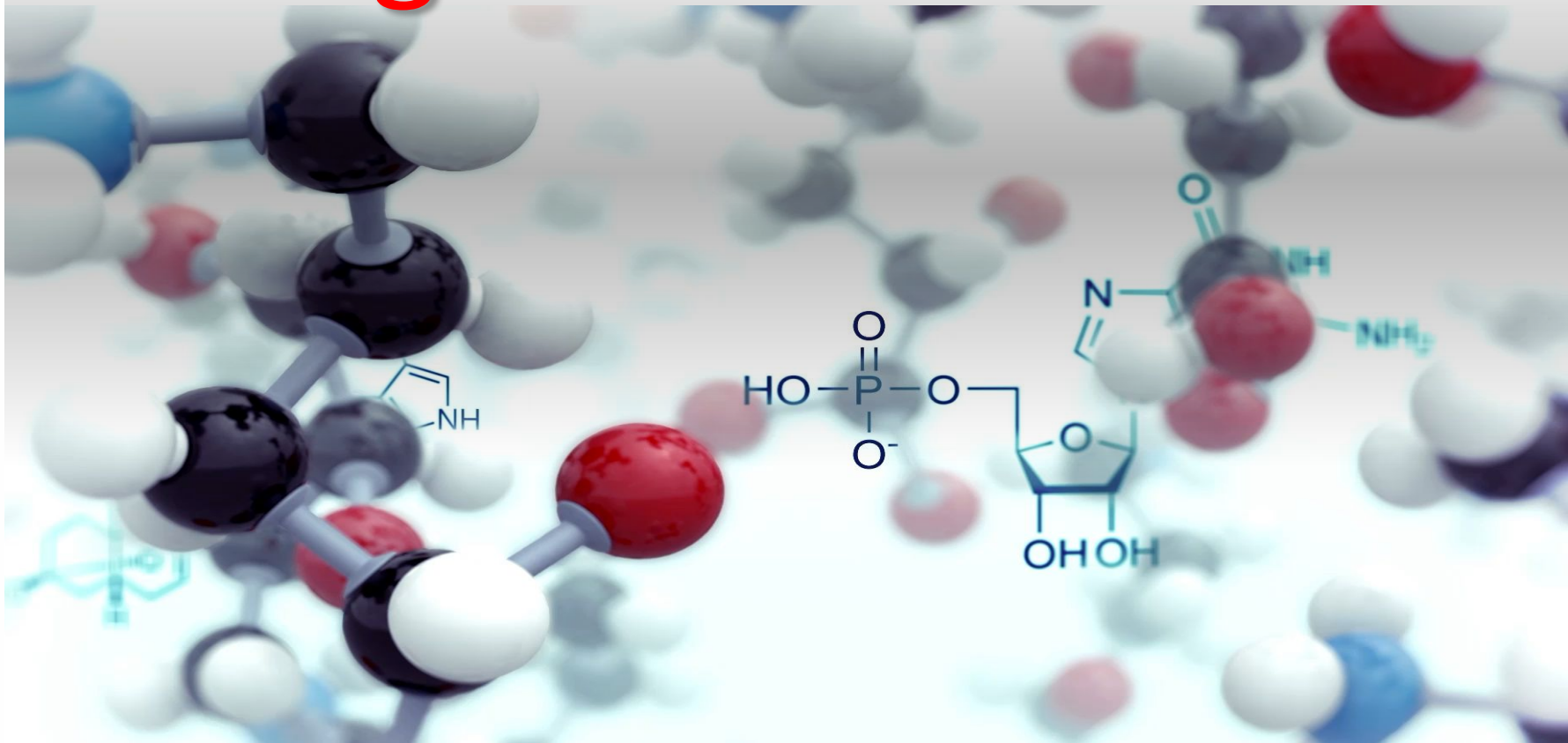
OPINION
GUEST ESSAY

Is a Revolution in Cancer Treatment Within Reach?

June 16, 2023



Brian Rea



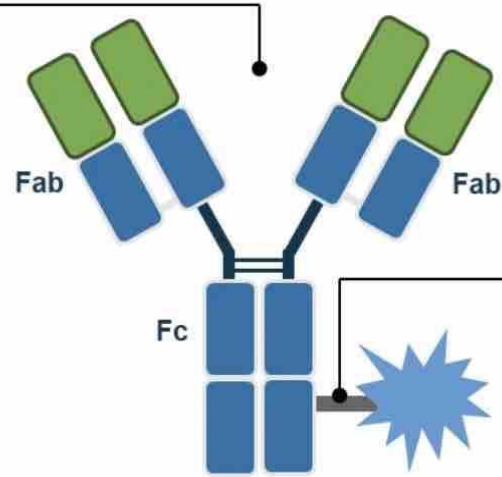
Antibody-Drug Conjugates: New kids on the block

Important Properties of the ADC Components and Target Antigen

Antigen
- High homogeneous expression on tumor
- Low or no expression on healthy tissues
- High affinity and avidity for antibody recognition

Antibody
- High affinity and avidity for tumor antigen
- Chimeric or humanized to decrease immunogenicity
- Long half-life and high molecular weight

Cytotoxic Payload
- Highly potent agents:
  - Calicheamicin
  - Maytansine derivative (DM1 or DM4)
  - Auristatin (MMAE or MMAF)
  - SN-38
  - DXd topoisomerase I inhibitor
- Optimal DAR (range: 2 to 8)



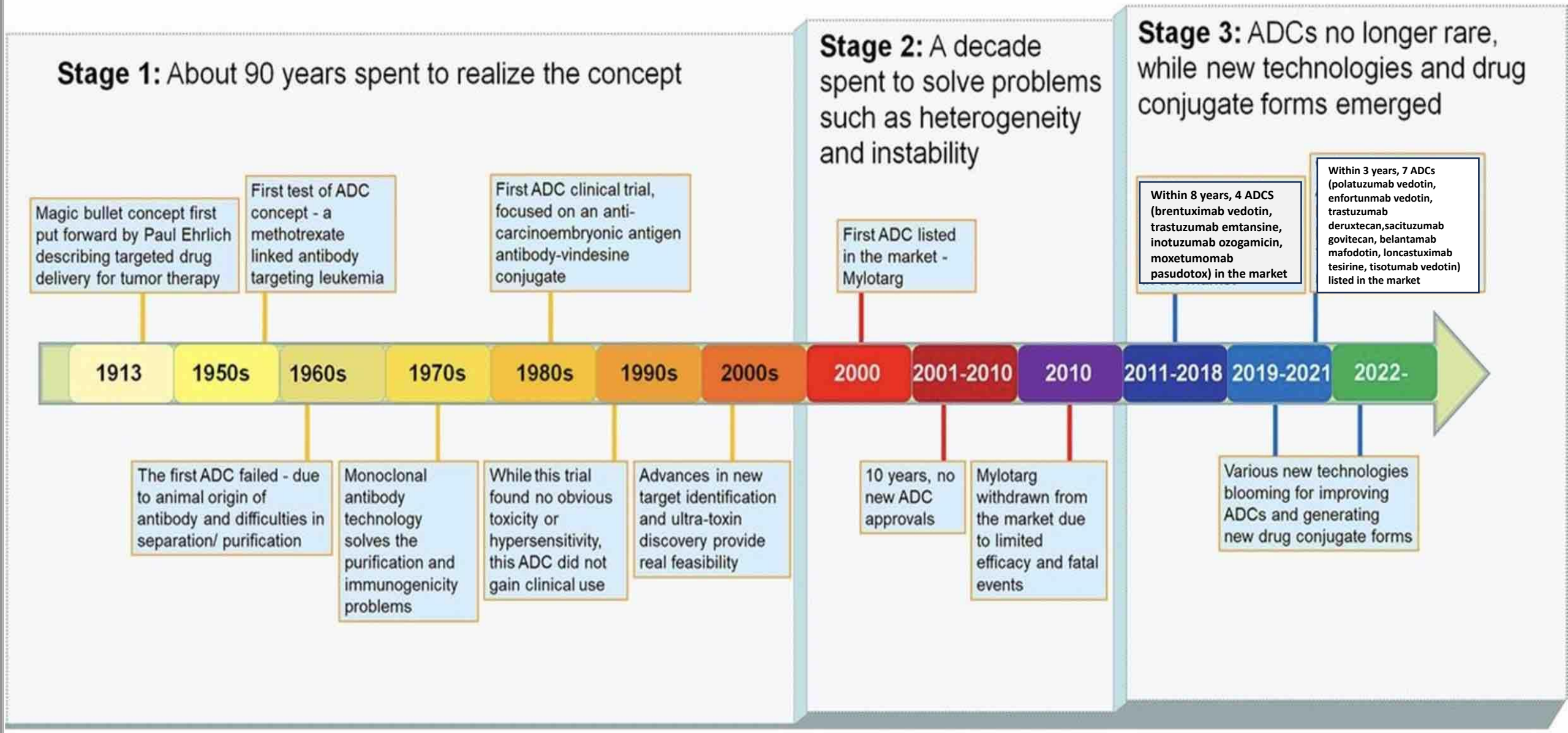
Linker
- Stable in circulation
- Efficient release of payload at target site
- Prevents premature release of payload at nontarget tissue
- Efficient linker technology **(cleavable vs noncleavable)**
- Site of conjugation
- DAR affects drug distribution and pharmacokinetics

Cleavable Linkers
Depend on physiological conditions: pH, proteolysis, or high intracellular glutathione

Noncleavable Linkers
Depend on lysosomal degradation

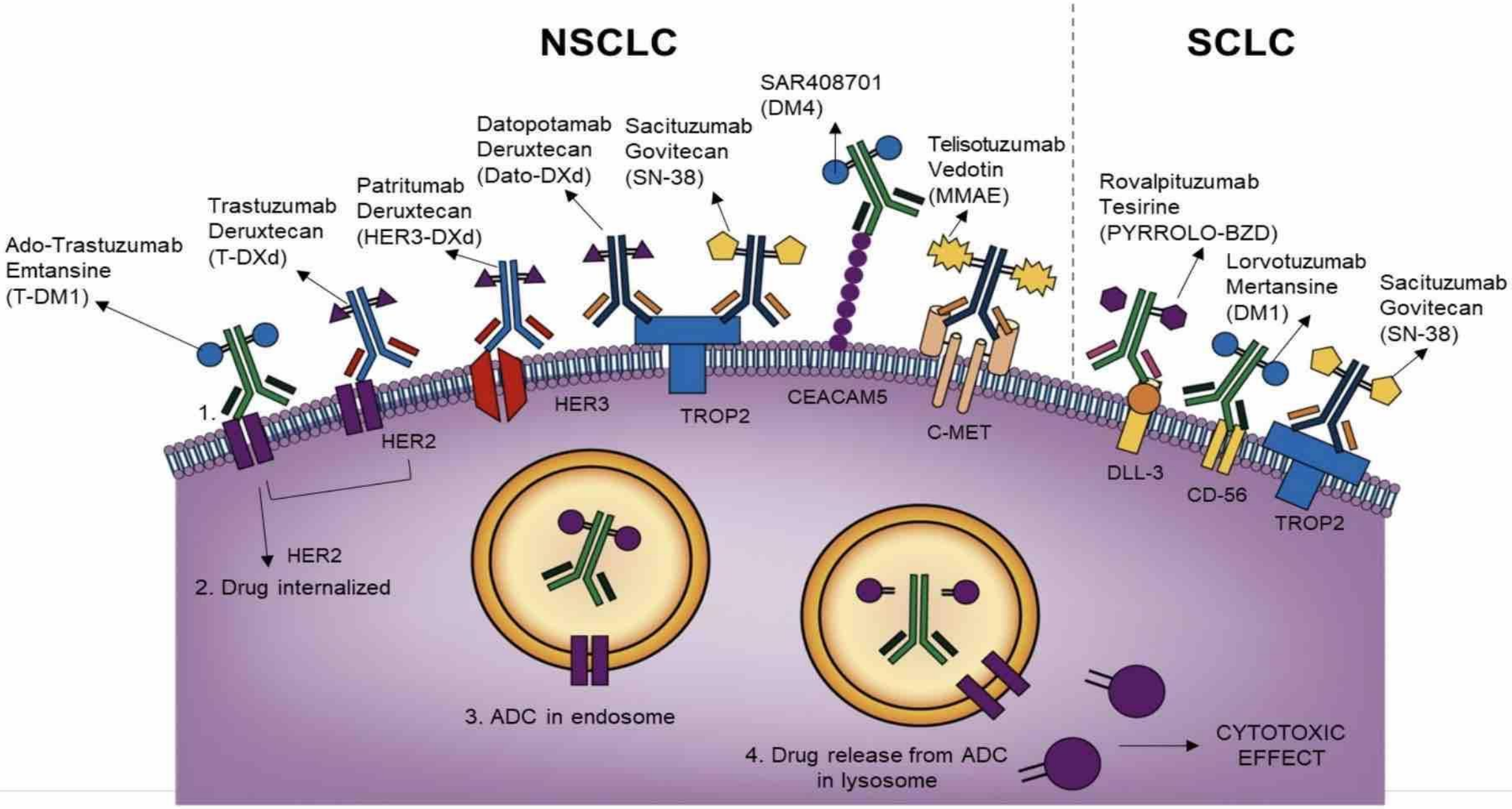
Chau CH, et al. Lancet. 2019;394:793-804.

History, progress and research stages of drug conjugates



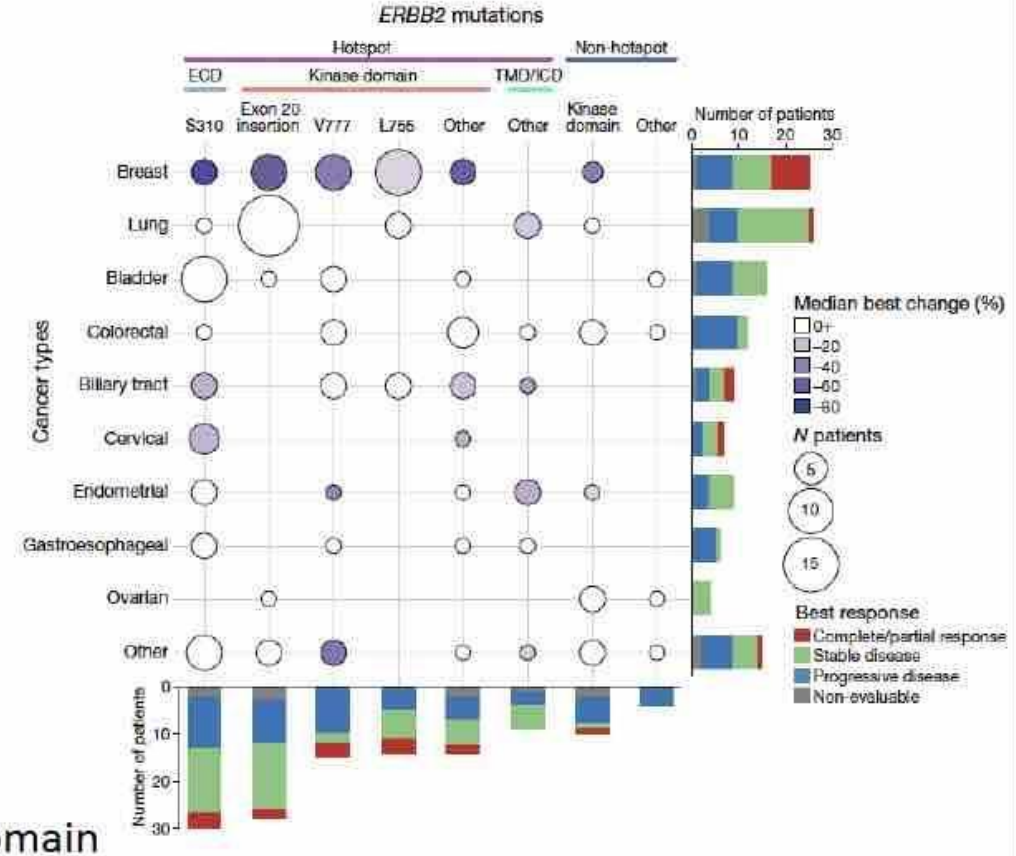
Jin et al. Pharmacological Reviews July 2022

Desai, Lung Cancer, 2022



What Is "HER2+" Lung Cancer

HER2 in NSCLC	Frequency
Protein overexpression	59%
Overexpression (IHC 2+ and 3+)	15-30%
Overexpression (IHC 3+ only)	2-6%
Amplification (FISH) GCN to CEP 17 >=2	2-6%
Mutations	1-5%



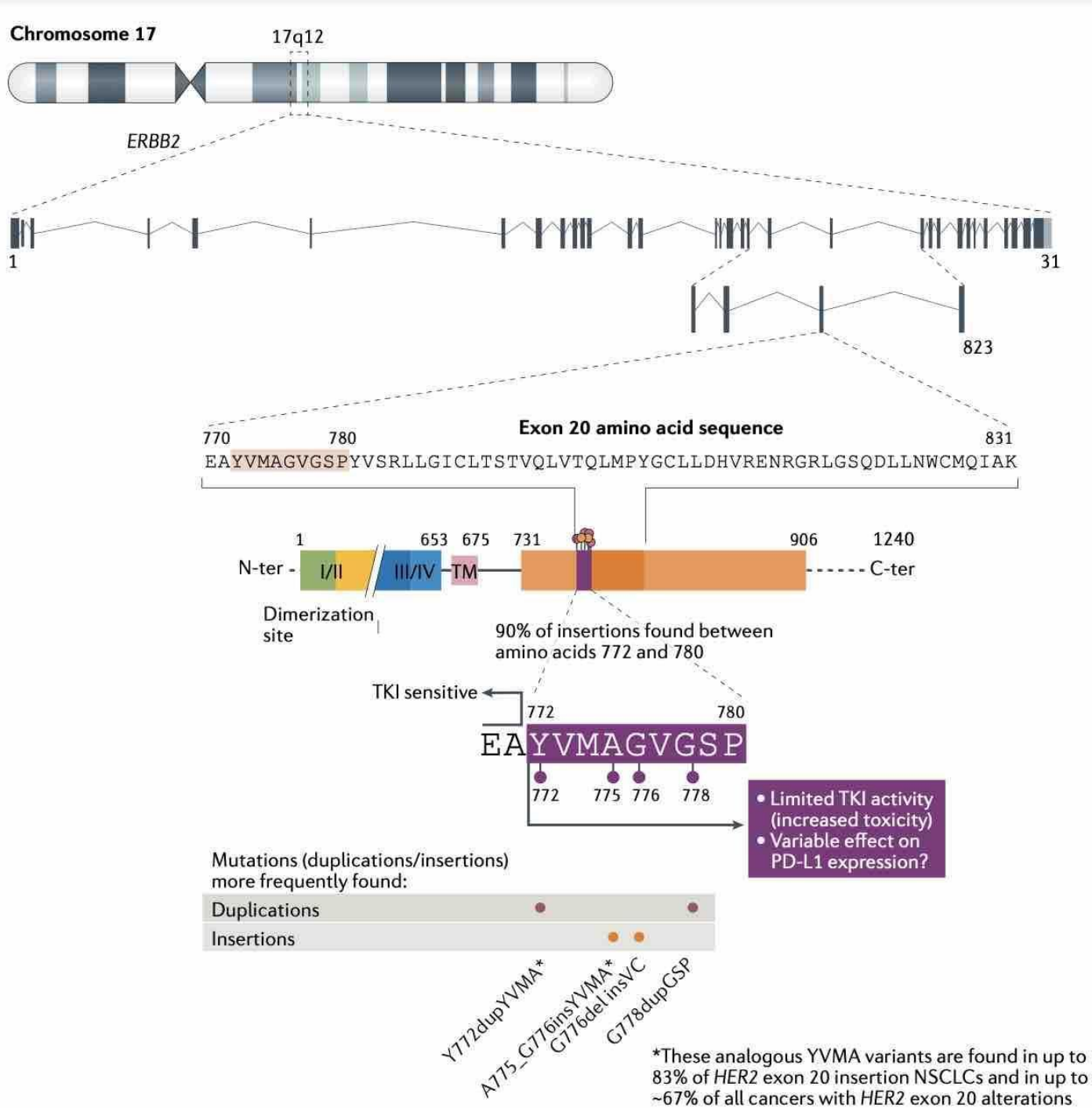
- Most common HER 2 mutations in lung cancer are in tyrosine kinase domain
  - exon 20 insertions
- Less common are in
  - Furin-like domain (S310F)
  - Transmembrane domain (V659E)
- Contrary to breast and gastric cancer, overexpression does not always co-occur with amplification

slide credit: Dr. Bazhenova

Pillai RN et al. *Cancer*. 2017;123(21)4099-4105. Liu S et al. *J Clin Oncol*. 2010;28(25):4006-4012. Nakamura H et al. *Cancer*. 2005;92(6):1033-1037. Bansal P et al. *Front Oncol*. 2016;6:112. Heinmoller P et al. *Clin Cancer Res*. 2003;9:5238-5244. Menard S et al. *Ann Oncol*. 2001;12(suppl 1):S15-S19. Peters S et al. *Transl Lung Cancer Res*. 2014;3(2):84-88. Rothschild SI et al. *Cancer*. 2015;7(2):930-949. Hyman DM et al. *Nature*. 2018;554:189-194.

HER2 Ex20Ins

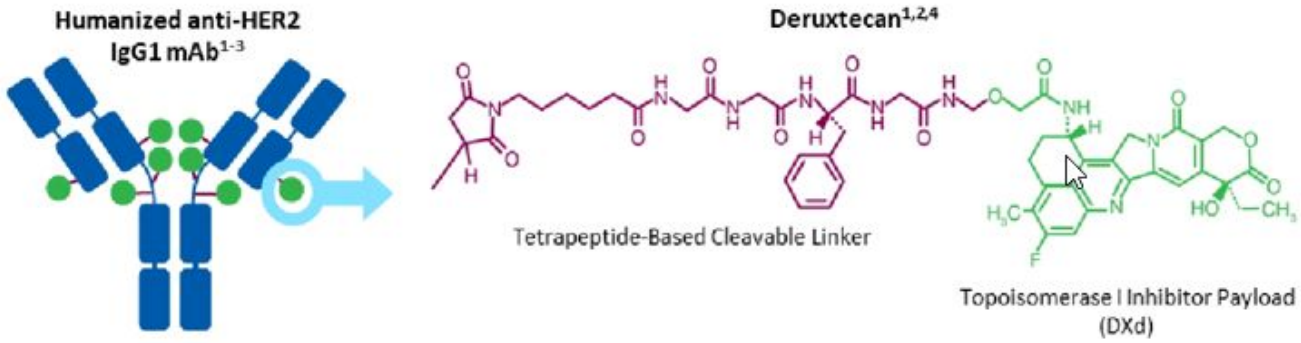
A Friedlaender, Nat Rev 1/2022



HER 2 **Mutation** Target in Lung Cancer Trastuzumab Deruxtecan (T-Dxd)

T-DXd is an ADC with 3 components:

- A humanized anti-HER2 IgG1 mAb with the same amino acid sequence as trastuzumab
- A topoisomerase I inhibitor payload, an exatecan derivative
- A tetrapeptide-based cleavable linker



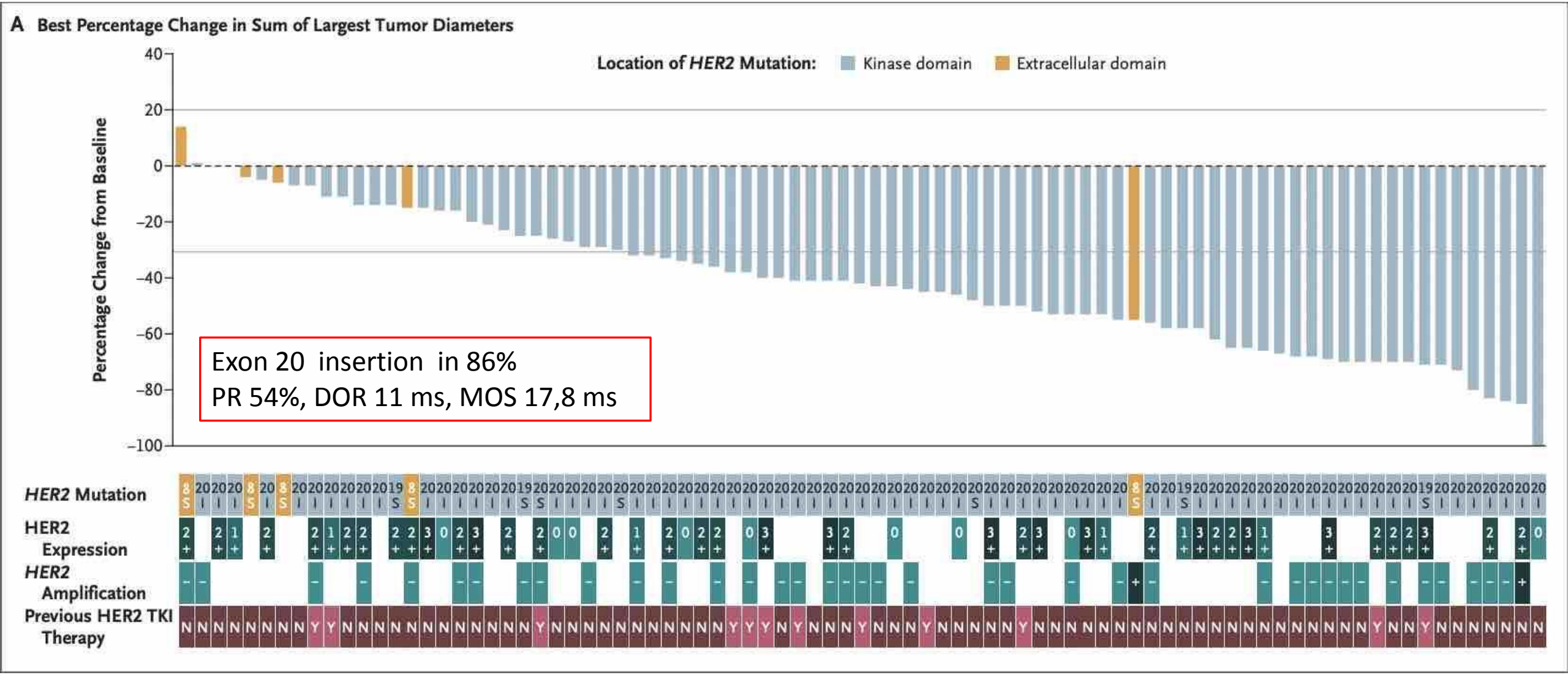
Key Design Attributes:

Antibody Drug Conjugate (ADC)
Payload mechanism of action: topoisomerase I inhibitor [1,2]
High potency of payload [1,2]
High drug-to-antibody ratio, ≈ 81,[2]
Payload with short systemic half-life [1,2]
Stable linker-payload [1,2]
Tumor-selective cleavable linker [1,2]
Membrane-permeable payload [1,4]

1. Nakada T et al. *Chem Pharm Bull*(Tokyo). 2019;67(3):173-185. 2. Ogitani Y et al. *Clin Cancer Res*. 2016;22(20):5097-5108. 3. Trail PA et al. *Pharmacol Ther*. 2018;181:126-142. 4. Ogitani Y et al. *Cancer Sci*. 2016;107(7):1039-1046.

Trastuzumab Deruxtecan in mutated HER2

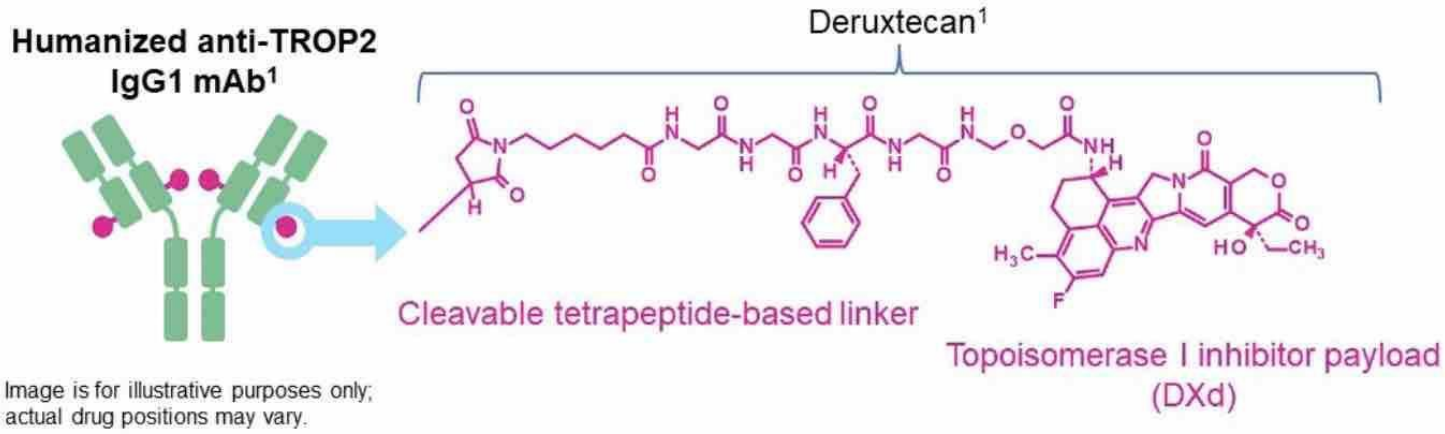
Destiny Trial, Li BT, NEJM, 2021



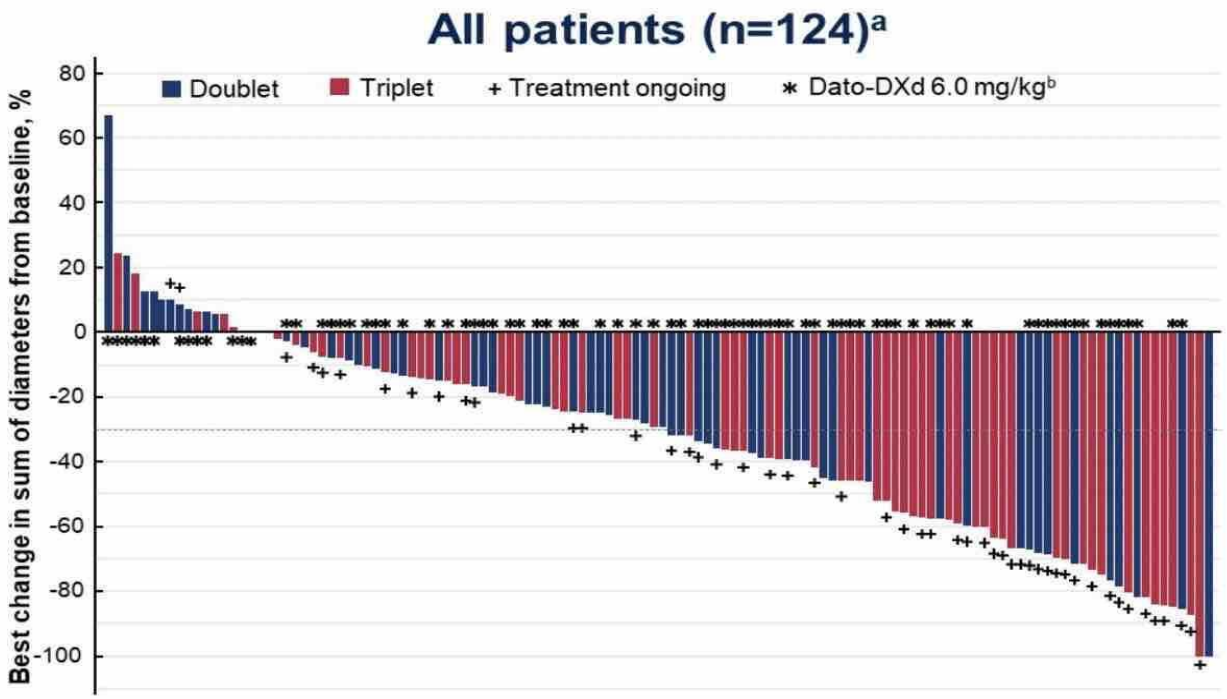
2023 ASCO ANNUAL MEETING

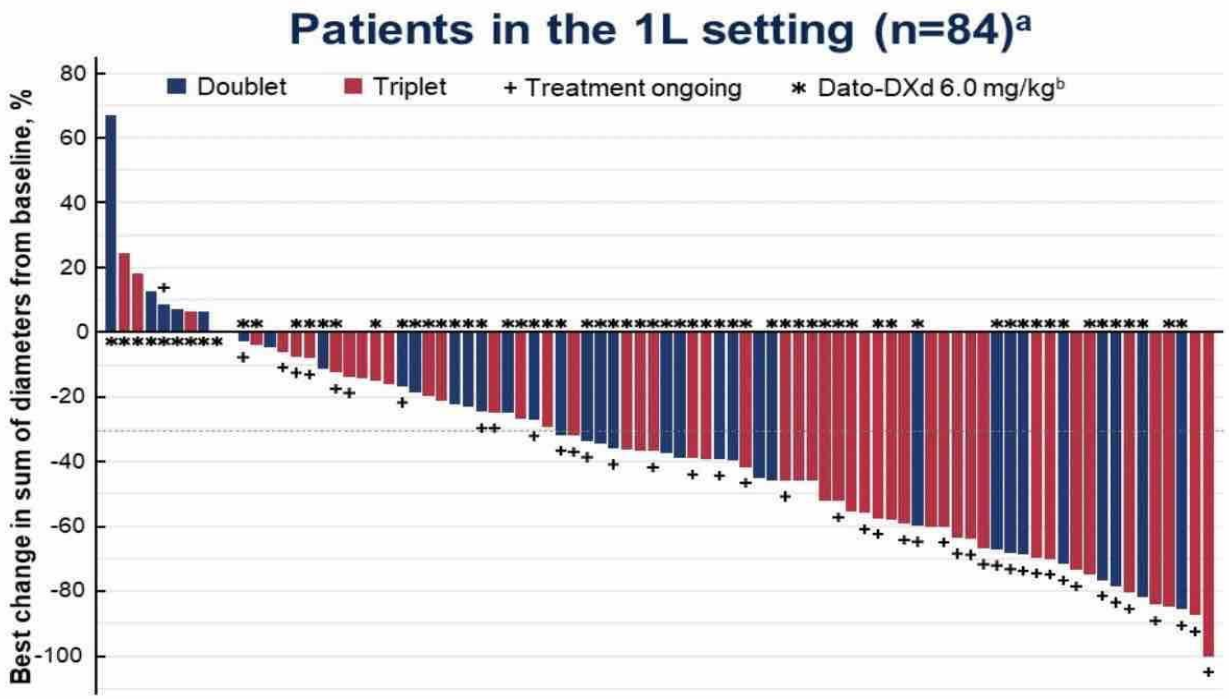
TROPION-Lung02: Datopotamab Deruxtecan (Dato-DXd) Plus Pembrolizumab With or Without Platinum Chemotherapy in Advanced Non-Small Cell Lung Cancer

Yasushi Goto, MD, PhD,[1] Wu Chou Su, MD,[2] Benjamin Levy, MD,[3] Olivier Rixe, MD, PhD,[4,5] Tsung Ying Yang, MD, PhD,[6] Anthony Tolcher, MD,[7] Yanyan Lou, MD, PhD,[8] Yoshitaka Zenke, MD, PhD,[9] Panayiotis Savvides, MD,[10] Enriqueta Felip, MD, PhD,[11] Manuel Domine, MD, PhD,[12] Konstantinos Leventakos, MD, PhD,[13] Mariano Provencio Pulla, MD, PhD,[14] Atsushi Horiike, MD, PhD,[15] Edward Pan, MD,[5] Daisy Lin, PhD,[5] Jessie Gu, PhD, MS,[5] Priyanka Basak, MD, MBE,[5] Michael Chisamore, PhD,[16] Luis Paz-Ares, MD, PhD[17]



Tropion Lung 02: Anti Trop2 AB + I/O +/- Chemo





AESI, n (%)[a,b]	Doublet (n=64)		Triplet (n=72)	
	All grades	Grade ≥3	All grades	Grade ≥3
Oral mucositis/stomatitis	37 (58)	5 (8)	31 (43)	4 (6)
ILD/pneumonitis adjudicated as drug related[c]	11 (17)	2 (3)	16 (22)	2 (3)
Ocular surface toxicity[d]	10 (16)	1 (2)	17 (24)	2 (3)
IRR[e]	15 (23)	0	10 (14)	0

TROP 2 Trials in 1st Line

TROPION-Lung02	IB	Dato-DXd + pembro + pembro + platinum	1L/2L	NCT04526691
TROPION-Lung04	IB	Dato-DXd + durva + durva + platinum	1L/2L	NCT04612751
TROPION-Lung07	III	Dato-DXd + pembro + platinum Dato-DXd + pembro Pembro + pemetrexed + platinum	1L PDL1 < 50%	NCT05555732
TROPION-Lung08	III	Dato-DXd + pembro Pembro	1L PDL1 ≥ 50%	NCT05215340
AVANZAR	III	Dato-DXd + durva + carboplatin Pembro + histology-specific platinum doublet	1L	NCT05687266
EVOKE-03	III	Sacituzumab + pembro Pembro	1L PDL1 ≥ 50%	NCT05609968
EVOKE-02	II	Sacituzumab + pembro Sacituzumab + pembro + platinum	1L	NCT05186974

Encorafenib and Binitemib in mBRAF V600E NSCLC PHAROS, ASCO 2023, G Riely, #9018

- The combination of encorafenib (BRAF inhibitor) plus binimetinib (MEK inhibitor) has demonstrated clinical efficacy with an acceptable safety profile in patients with metastatic BRAF V600E/K–mutant melanoma[1]
- For patients with metastatic BRAF V600E–mutant NSCLC the combination of dabrafenib and trametinib was approved by the US FDA and is a current standard of care [2]
  - This approval was based on the results of a single-arm, phase 2 study that showed meaningful antitumor activity and a manageable safety profile[3,4]
    - In treatment-naïve and previously treated patients, the ORR by IRR was 64% and 63%, respectively
    - The median DOR by IRR was 15.2 months and 9.0 months, respectively
- Given the observed efficacy and safety profile of encorafenib plus binimetinib in patients with BRAF V600E/K–mutant metastatic melanoma, this combination therapy was assessed in patients with metastatic BRAF V600E–mutant NSCLC

Key eligibility criteria

- Metastatic BRAF V600E–mutant NSCLC
- ECOG performance status 0 or 1
- No *EGFR* mutation, *ALK* fusion, or *ROS1* rearrangement
- No more than 1 prior line of treatment in the advanced setting
- No prior treatment with BRAF or MEK inhibitor
- No symptomatic brain metastases

***BRAF* mutation testing**

- Determined locally by PCR- or NGS-based assay; sent to central laboratory[a]
- Pleural fluid, fresh and archived tissue, and fine needle aspiration were acceptable

Patients enrolled

- Treatment naïve n=59
- Previously treated n=39

Encorafenib: 450 mg QD

Binimetinib: 45 mg BID

28-day cycles

Treat until progression or unacceptable toxicity

Primary endpoint

- ORR[b] by IRR

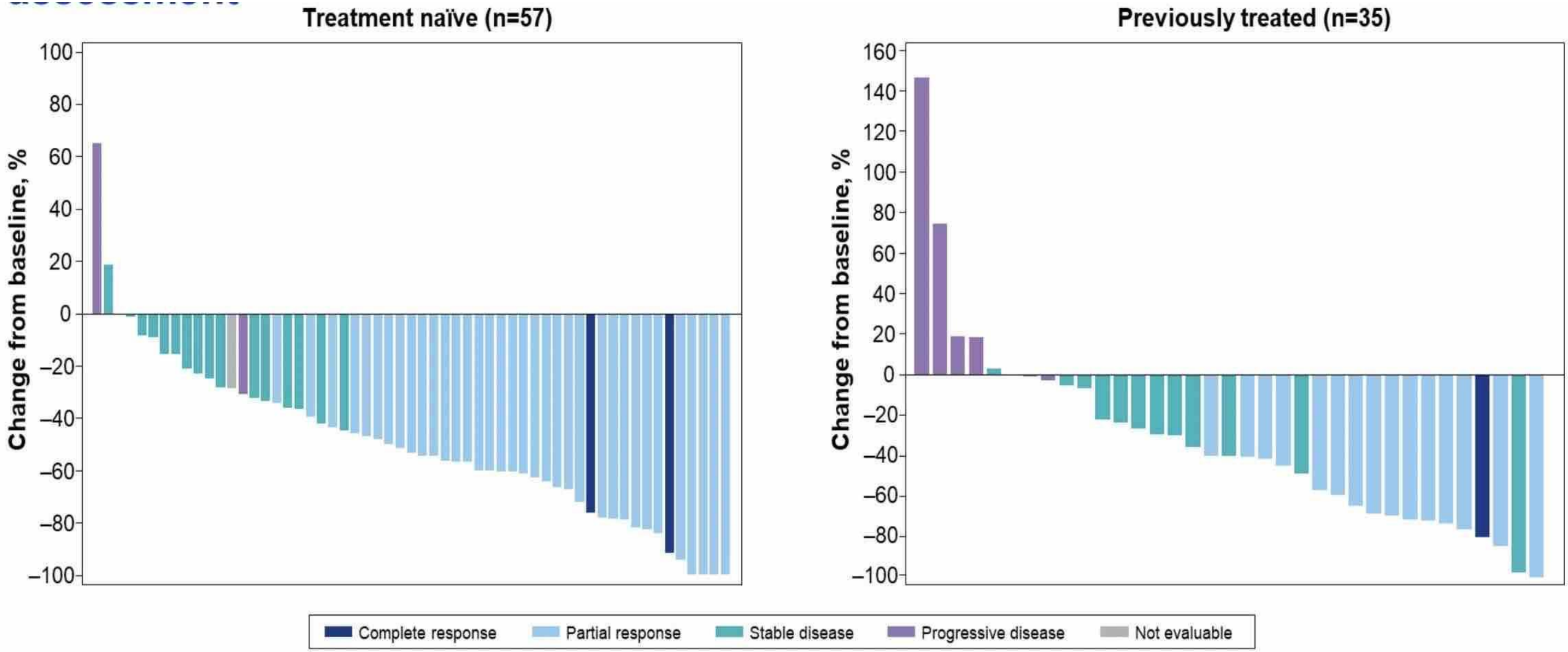
Secondary endpoints

- ORR by investigator
- DOR, DCR, PFS, and TTR (all by IRR and investigator)
- OS
- Safety

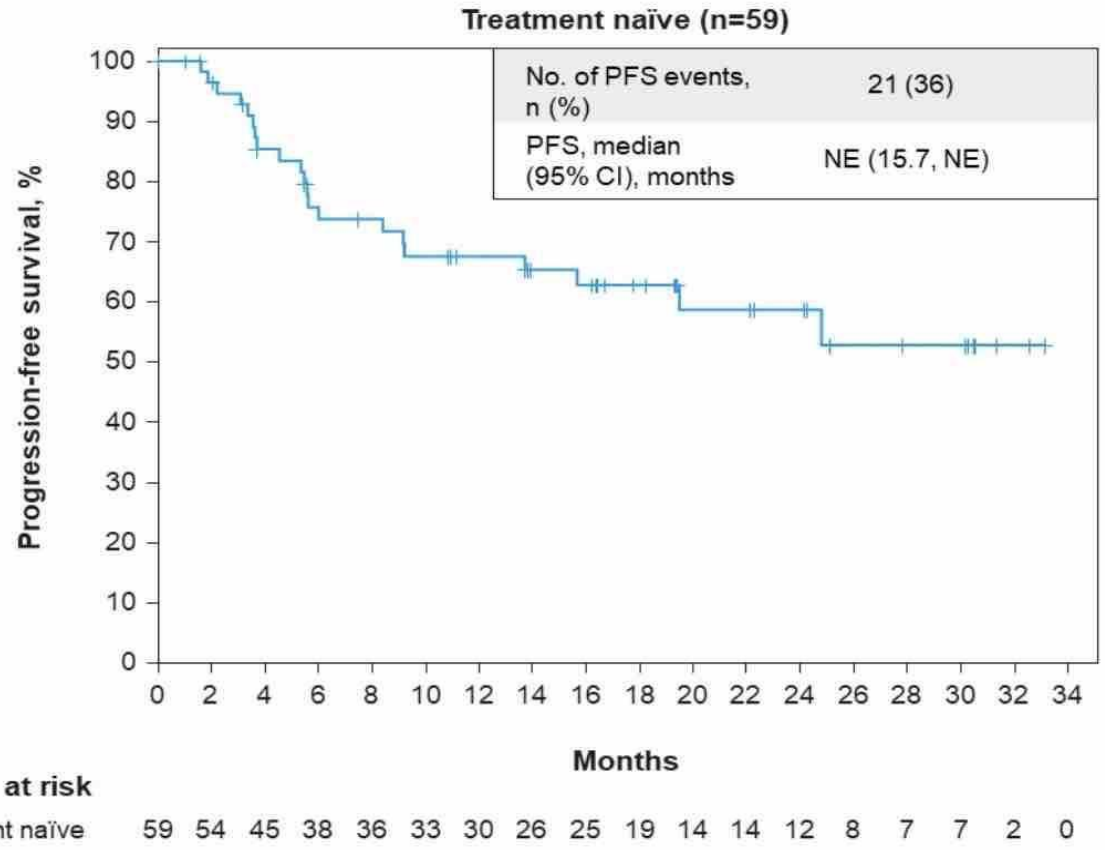
Exploratory endpoints

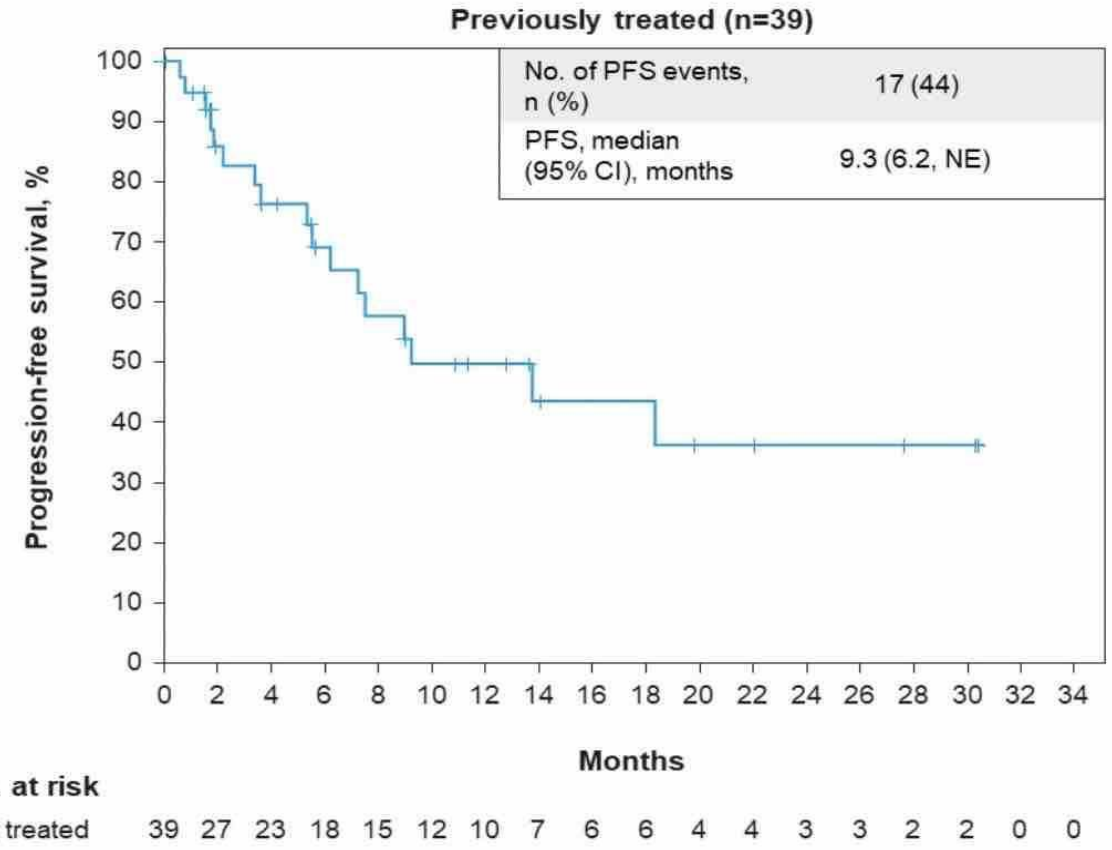
- Biomarker and pharmacokinetic analyses

Encorafenib and Binitemib in mBRAF V600E NSCLC PHAROS, ASCO 2023, G Riely, #9018



Encorafenib and Binitemib in mBRAF V600E NSCLC PHAROS, ASCO 2023, G Riely, #9018





- The median duration of follow-up for PFS by IRR was 18.2 months (95% CI, 16.4, 22.3 months) in treatment-naïve patients and 12.8 months (95% CI, 9.0, 19.8 months) in previously treated patients

KRAS G12C Inhibititor (Sotorasib) + Chemotherapy

SCARLET: study schema

Key inclusion criteria

- Advanced non-Sq, NSCLC
- With KRAS G12C
- Naïve for Cytotoxic chemotherapy and KRAS inhibitor
- With measurable lesion
- ECOG PS 0-1
- Asymptomatic CNS mets allowed

Induction phase

Sotorasib 960mg
+ CBDCA (AUC5)/ PEM 500 mg/m ^{2}
[q3W, 4 cycles]
(n = 30)

Maintenance phase

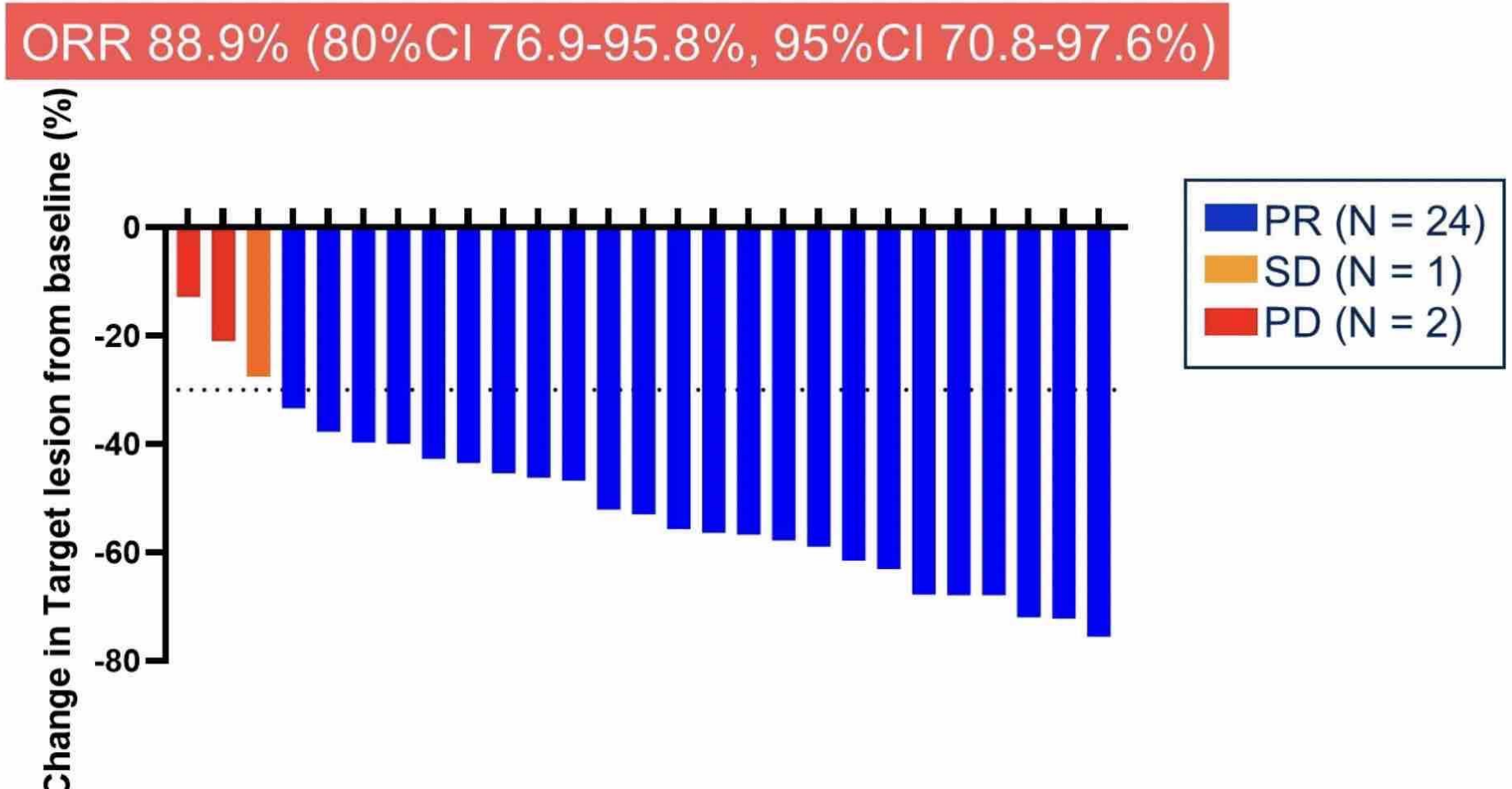
Sotorasib + PEM
[q3W, until PD]

- Primary endpoint; ORR by blinded independent central review (BICR)
- Secondary endpoints; DCR, PFS, DOR, OS and AEs
- Translational research; NGS analysis (tissue and plasma [at baseline, 3 wks, and PD])

Trial identifier: jRCT2051210086

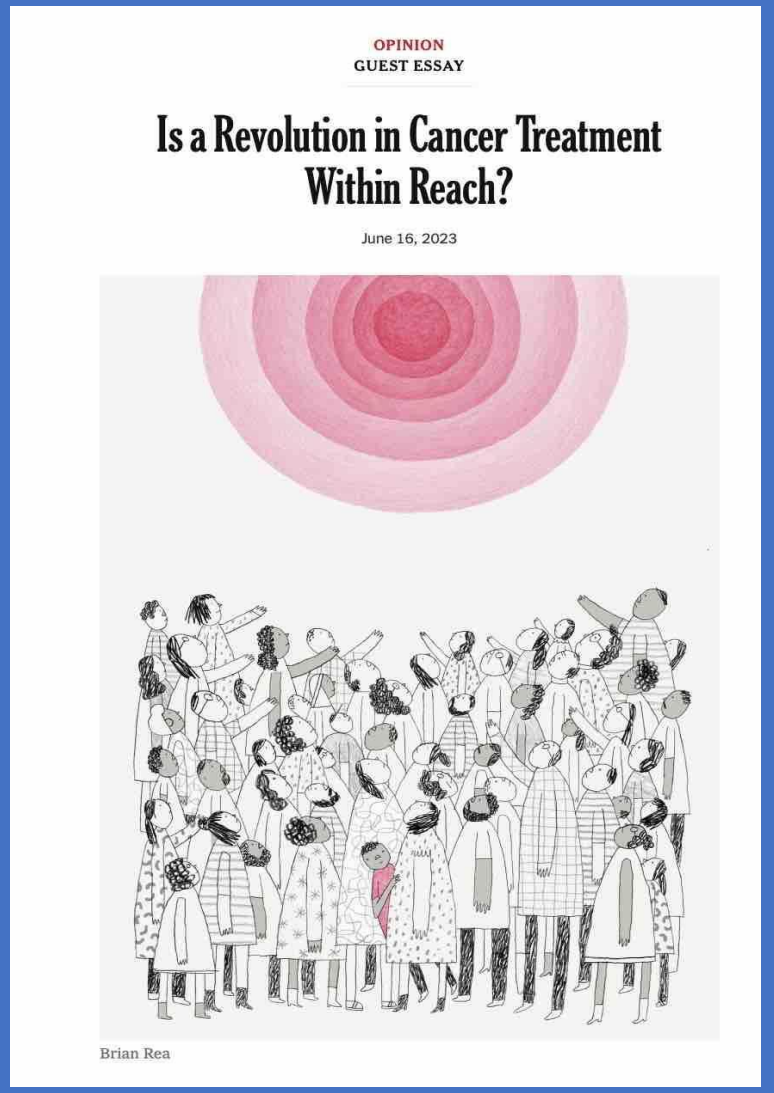
Scarlet: KRAS G12C + Chemotherapy

Primary endpoint: ORR by BICR



MY TAKE HOME FROM ASCO 2023

- Increasing molecular targets in NSCLCs
- mEGFR TKI resistance mechanisms better understood
- Chemo/TKI not synergistic in mEGFR NSCLC
- Chemo/IO not effective in overcoming resistance
- More targeted therapies available and coming soon
- EGFR Exon 20, Her 2 mutation, EGFR WT, BRAF, KRAS G12C
- ADCs: a "Revolution Within Reach"?



OPINION
GUEST ESSAY

Is a Revolution in Cancer Treatment Within Reach?

June 16, 2023

Brian Rea