Iniciativa científica de:

GECP lung cancer research

Lung Cancer UPDATES

ESMO HIGHLIGHTS

23 - 27 OCTUBRE 2026

Madrid, Spain



#ESMOUPDATES

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Cáncer de pulmón no microcítico avanzado con mutación *driver*

Dr. Alejandro Olivares Hernández
Servicio de Oncología Médica
Hospital Universitario de Salamanca

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6. Investigación traslacional con ctDNA: ALK y *EGFR*

* La presentación actual se ha realizado mediante el material científico-docente del congreso ESMO 2026

ESTUDIO KRAScendo-1

ESTUDIO KRAScendo-1

Inhibidor de *KRAS* G12C (divarasib) frente a sotorasib/adagrasib en 2L de CPNM avanzado

MADRID 2026 ESMO congress

Krascendo 1: efficacy and safety of divarasib versus sotorasib or adagrasib in previously treated locally advanced or metastatic *KRAS* G12C-mutated NSCLC

Ferdinandos Skoulidis,[1] Myung-Ju Ahn,[2] Shirish Gadgeel,[3] Sanjay Popat,[4] Niels Reinmuth,[5] Bianca van Veggel,[6] Farastuk Bozorgmehr,[7] Victoria J Bray,[8] Michael Duruisseaux,[9] Wade T Iams,[10] Julien Mazieres,[11] Laura Bonanno,[12] Lore Decoster,[13] Bo Wang,[14] Youyou Hu,[15] Sarah M Shagan,[16] Verena Lam,[16] Uyi Osaghae,[16] Tien Hoang,[16] Benjamin J Solomon[17]

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- *KRAS* G12C mutado (9-10%)
- Entre 1-3 líneas de tratamiento
- No permitido entrecruzamiento

Objetivo primario: PFS por BICR

Krascendo 1: a phase III, global, randomised, open-label trial in previously treated *KRAS* G12C-mutated advanced or metastatic NSCLC

Key eligibility criteria

- KRAS G12C inhibitor naive
- ECOG PS 0–1
- Advanced *KRAS* G12C-mutated NSCLC
- ≥1 but ≤3 lines of prior systemic therapy, including platinum-based chemo and a PD-(L)1 inhibitor*
- Measurable disease per RECIST v1.1
- Patients with active, untreated CNS metastases were excluded

R 1:1

N=338

- **Divarasib (400 mg QD)**
- Treatment crossover not permitted
- **Sotorasib (960 mg QD) OR adagrasib (600 mg BID)†**

Treatment until disease progression, unacceptable toxicity, death, withdrawal of consent, or study termination

Treatment beyond progression was permitted

Stratification factors
- History of CNS metastases
- Intended choice of control arm KRAS G12C inhibitor†
- *KEAP1* mutation status‡

Primary endpoint
- PFS per BICR

Key secondary endpoints
- OS
- Confirmed ORR per BICR
- TTCD

Other secondary endpoints
- DoR per BICR
- Safety
- Other PROs

Patient demographics and baseline characteristics

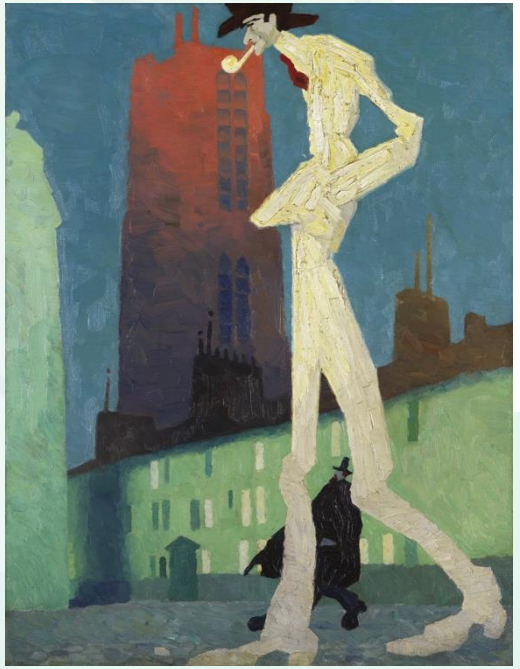
Characteristic, n (%), unless otherwise specified		**Divarasib (n=167)**	**Control (n=171)**
Median age, years (range)		66.0 (42–87)	66.0 (39–89)
Sex	Female	72 (**43**)	75 (**44**)
	Male	95 (**57**)	96 (**56**)
ECOG PS	0	65 (**39**)	68 (**40**)
	1	102 (**61**)	103 (**60**)
Smoking status	Never smoked	4 (**2**)	6 (**4**)
	Current/former smoker	163 (**98**)	165 (**96**)
Race	White	127 (**76**)	135 (**79**)
	Asian	17 (**10**)	23 (**13**)
	Black or African American	5 (**3**)	1 (**1**)
	Other	18 (**11**)	12 (**7**)
Geographic region	Europe	106 (**63**)	103 (**60**)
	North America	28 (**17**)	27 (**16**)
	Rest of World	33 (**20**)	41 (**24**)
Prior lines of therapy	1	121 (**72**)	118 (**69**)
	2	41 (**25**)	51 (**30**)
	3	5 (**3**)	2 (**1**)

Characteristic, n (%), unless otherwise specified		**Divarasib (n=167)**	**Control (n=171)**
Histology	Adenocarcinoma	158 (**95**)	164 (**96**)
	Squamous	2 (**1**)	4 (**2**)
	Other	7 (**4**)	3 (**2**)
KEAP1 mutation status*	Detected	35 (**21**)	37 (**22**)
	Not detected or unknown†	132 (**79**)	134 (**78**)
PD-L1 status‡	<1%	57 (**34**)	70 (**41**)
	1–<50%	62 (**37**)	65 (**38**)
	≥50%	43 (**26**)	32 (**19**)
History of CNS metastases*§	Yes	48 (**29**)	51 (**30**)
	No	119 (**71**)	120 (**70**)
Intended KRAS G12C inhibitor in control arm*	Sotorasib	153 (**92**)	156 (**91**)
	Adagrasib	14 (**8**)	15 (**9**)
Prior CPI status	Prior CPI¶	165 (**99**)	171 (**100**)
	No prior CPI	2 (**1**)	0

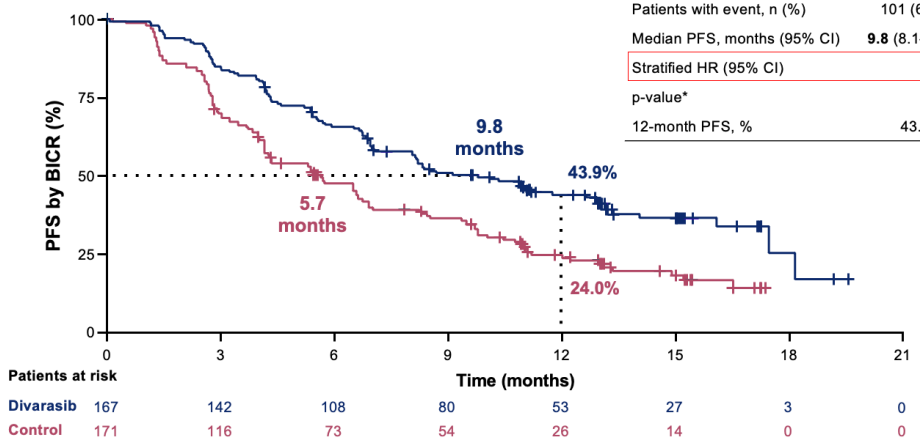








Progression-free survival by BICR



	Divarasib (n=167)	Control (n=171)
Patients with event, n (%)	101 (60.5)	128 (74.9)
Median PFS, months (95% CI)	**9.8** (8.1–12.9)	**5.7** (4.2–6.8)
Stratified HR (95% CI)	**0.58** (0.44–0.76)	
p-value*	<0.0001	
12-month PFS, %	43.9	24.0

SUPERVIVENCIA LIBRE DE PROGRESIÓN

9,8m frente 5,7m

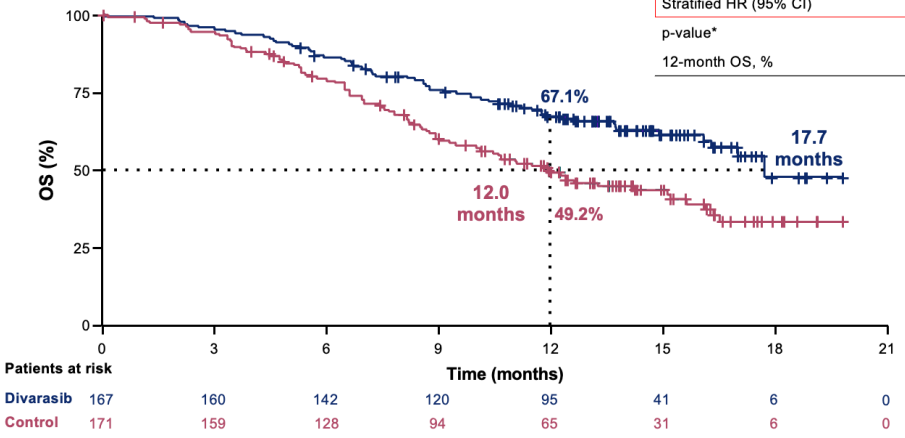
HR=0,58 (-0,76)

Overall survival

SUPERVIVENCIA GLOBAL

17,7m frente 12,0m

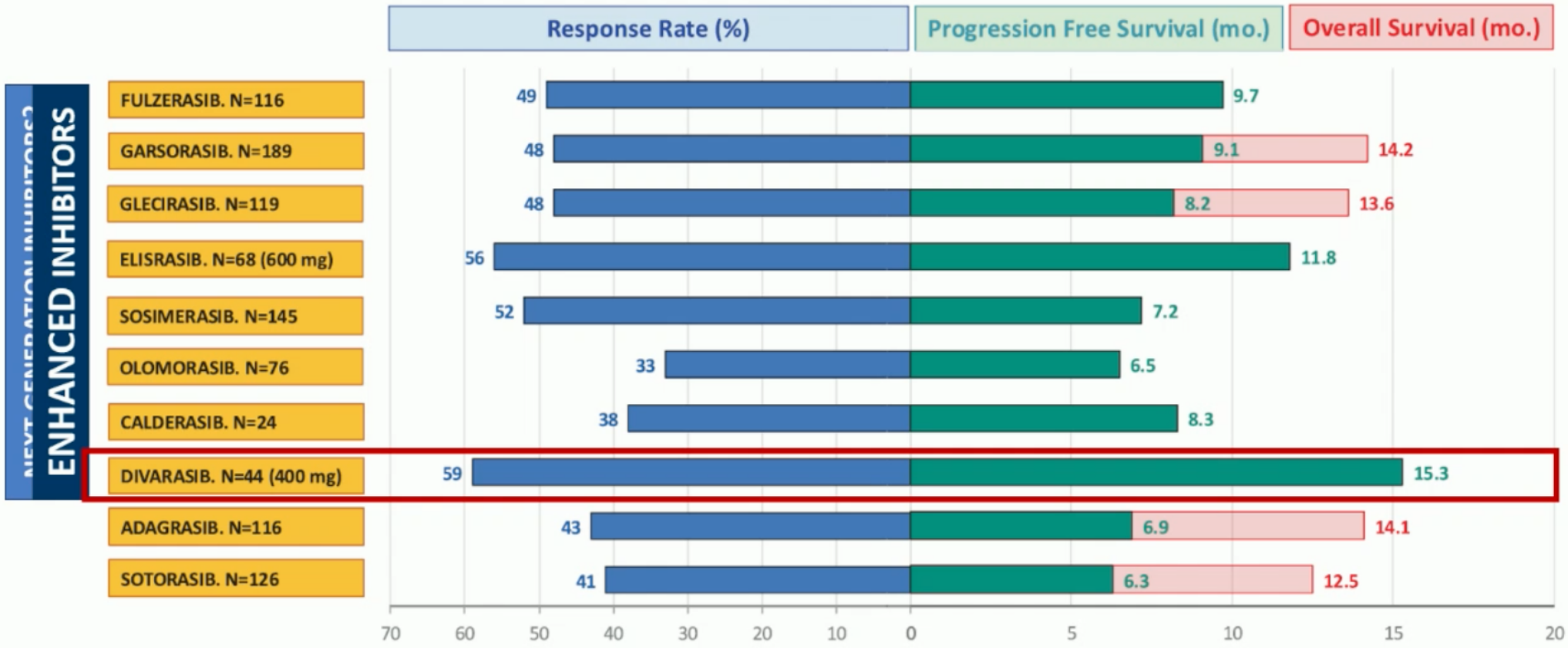
HR=0,56 (-0,78)



	Divarasib (n=167)	Control (n=171)
Patients with event, n (%)	63 (37.7)	93 (54.4)
Median OS, months (95% CI)	**17.7** (16.3–NE)	**12.0** (10.0–15.2)
Stratified HR (95% CI)	**0.56** (0.41–0.78)	
p-value*	0.0005	
12-month OS, %	67.1	49.2

KEAP1 mutation status*	Detected	72	35	8.7	37	7.0	0.63 (0.36–1.10)
	Not detected/unknown†	266	132	NE	134	14.2	0.53 (0.35–0.78)
History of CNS metastases*	Yes	99	48	16.1	51	12.0	0.78 (0.44–1.39)
	No	239	119	17.7	120	11.9	0.50 (0.34–0.74)

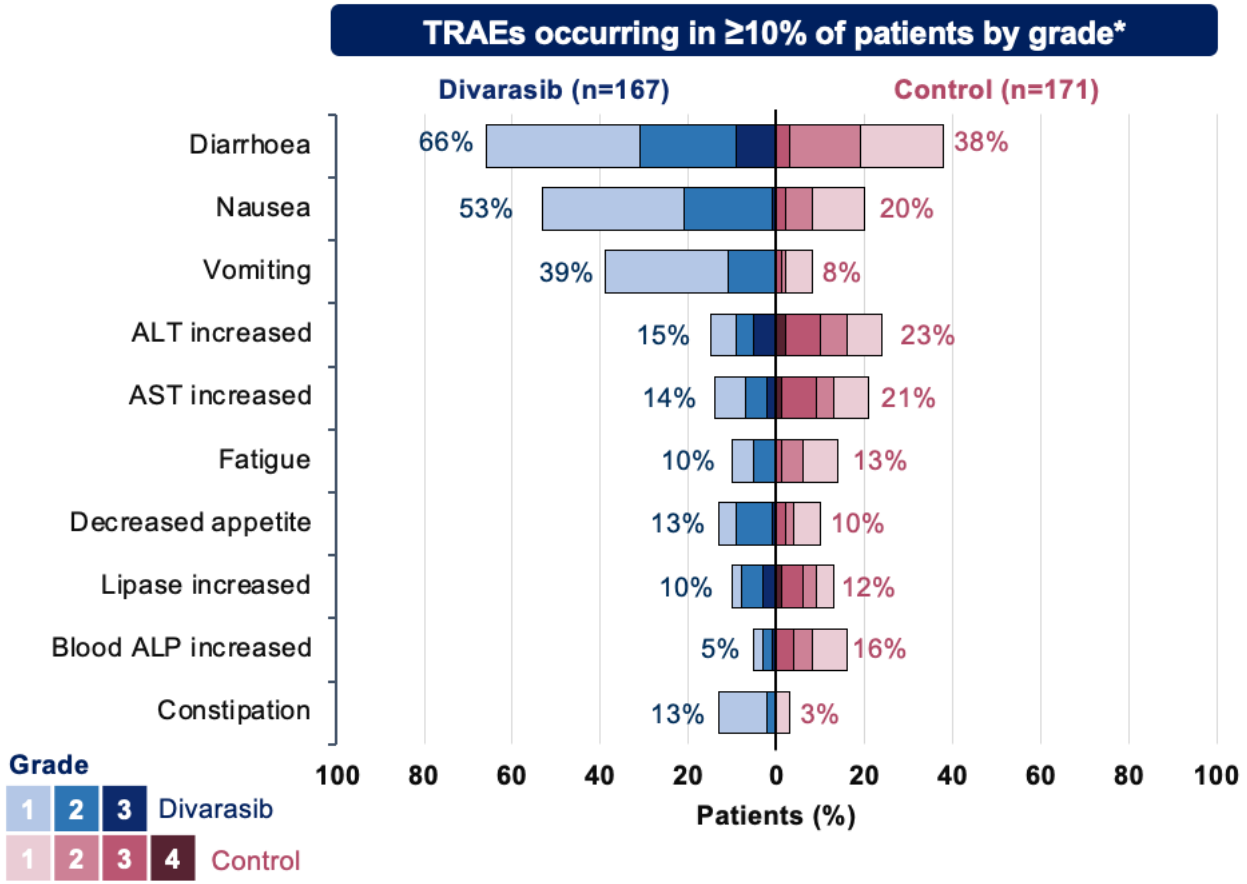
KRASi (OFF): highly competitive field (Phase I/II data)



KRAS: de inhibir G12C(OFF) a bloquear RAS(ON) — ¿el siguiente cambio de paradigma?

Toxicidad

Most common TRAEs



- Toxicidad similar a los inhibidores de la misma clase.
- Principales eventos adversos los gastrointestinales (diarrea, náuseas y vómitos). Mayor tasa de toxicidad digestiva frente a brazo control (grado 1-2).
- Menor tasa de hepatotoxicidad.

Discusión del estudio





Beneficio consistente en SG y SLP

Perfil de toxicidad similar

Fase 3 de comparador brazo estándar

Beneficio dudoso en ECOG 2 o mts cerebrales

Persiste la toxicidad de los iRAS G12C (OFF)

Probable terapia puente a iRAS ON

ESTUDIO SAFFRON

ESTUDIO SAFFRON

Inhibición dual de *EGFR* y *MET* (oral) en progresión a 1L con osimertinib (CPNM avanzado *EGFR*mut)

MADRID 2026 ESMO congress

Osimertinib plus savolitinib versus platinum–pemetrexed in patients with *EGFR*m advanced NSCLC with MET overexpression and/or amplification after progression on osimertinib: SAFFRON Phase III primary results

Shun Lu,[1] Nicolas Girard, Yusuke Okuma, Frances A. Shepherd, Xiaorong Dong, Surein Arulananda, Giulio Metro, Hidetoshi Hayashi, Laura Bonanno, Yu Jung Kim, Zhe Liu, Myung-Ju Ahn, Yuping Sun, Thuong Vu Le, Silvia Novello, Rafal Lolo, Balakumar Swaminathan, Laura Roncoroni, Francis Maria Esposito, James Chih-Hsin Yang

[1]Department of Medical Oncology, Shanghai Chest Hospital, Shanghai Jiao Tong University, School of Medicine, Shanghai, China

25 October 2026



- ***MET* sobreexpresado (IHQ >90%) o amplificado (FISH10+)**
- Brazo comparador: platino + pemetrexed
- Objetivo primario: PFS BICR

Key inclusion criteria

- Aged ≥18 years (≥19 years in the Republic of Korea)
- *EGFR*m (Ex19del, L858R, and/or T790M) locally advanced or metastatic NSCLC
- MET overexpression (IHC3+≥90%) and/or amplification (FISH10+)* centrally confirmed with post-progression tissue biopsy
- Progression on 1L or 2L osimertinib as the most recent anticancer treatment†
- ECOG PS 0 / 1
- Stable CNS metastases were allowed

N = 324

R 1:1

Stratified by:

Race (Asian / non-Asian)

Baseline brain metastases (Yes / no)

Line of treatment (2L / 3L)

Osimertinib 80 mg QD + savolitinib 300 mg BID‡

Treatment beyond PD allowed per investigator discretion¶

Pemetrexed 500 mg/m² + carboplatin AUC5 / cisplatin 75 mg/m² (Q3W for 4 cycles)

Maintenance pemetrexed 500 mg/m² (Q3W)‡

Investigator-assessed PD (per RECIST v1.1) or discontinuation

Endpoints

Primary: PFS by BICR per RECIST v1.1 in the FAS

Key secondary / exploratory:

- PFS by investigator, OS, ORR, DoR, DCR and tumour shrinkage in the FAS
- PFS in patients in the MET-overexpressed (IHC3+≥90%) or *MET*-amplified (FISH10+) analysis sets
- Safety in the SAF

Characteristic	Osimertinib + savolitinib (n = 171)	Platinum–pemetrexed (n = 167)
Sex: male / female, %	36 / 64	35 / 65
Median age (range), years	64 (34–86)	64 (37–86)
Race: Asian / non-Asian,* %	63 / 36	66 / 28
ECOG PS: 0 / 1, %	35 / 65	29 / 71
Extent of disease: locally advanced / metastatic, %	5 / 95	5 / 95
Brain metastases at baseline, yes / no, %	31 / 69	31 / 69
Prior line of therapy: 1 / ≥2, %	84 / 16	84 / 16
MET status: IHC3+≥90% / FISH10+ / both, %	73 / 75 / 49	71 / 69 / 41
EGFR mutation at randomisation:† Ex19del / L858R / other,‡ %	65 / 35 / 1	69 / 30 / 1
Smoking status: never / current / former, %	68 / 2 / 30	68 / 4 / 28

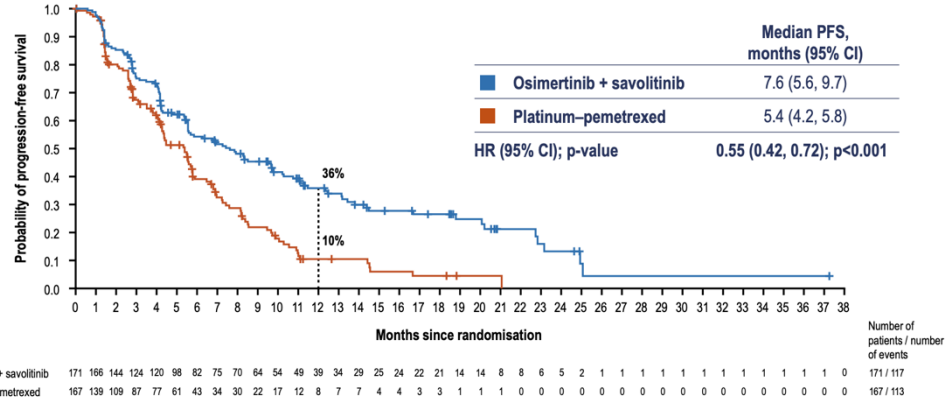






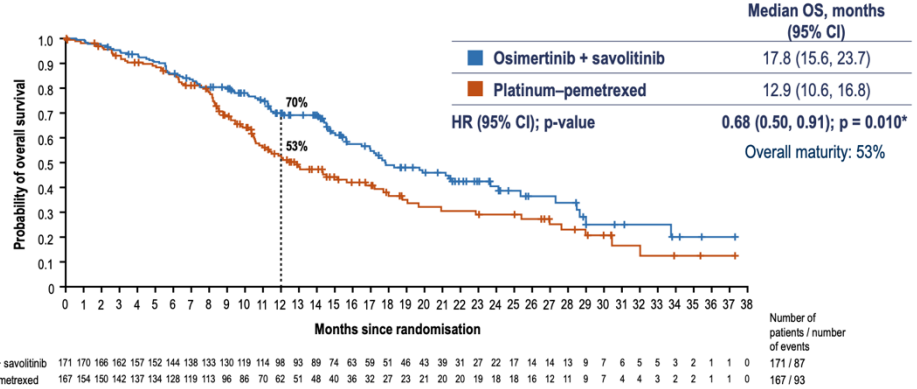


Progression-free survival by BICR



Osimertinib plus savolitinib provided a statistically significant 45% reduction in the risk of disease progression or death compared with platinum–pemetrexed

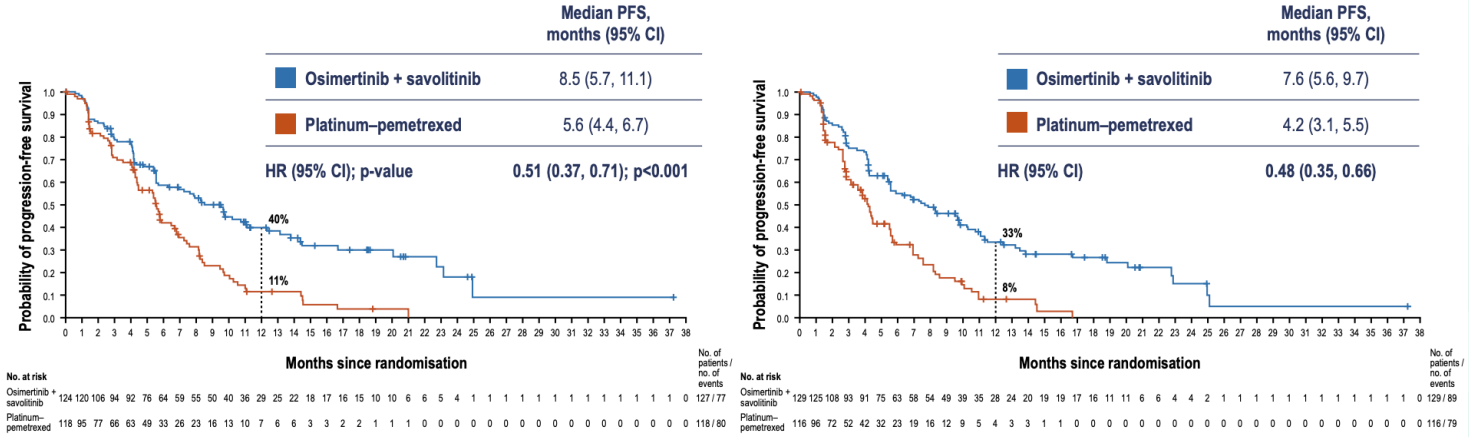
Overall survival



Osimertinib plus savolitinib provided a statistically significant 32% reduction in the risk of death, with a 4.9 month improvement in median OS, compared with platinum–pemetrexed

Progression-free survival by BICR: MET subgroups

MET-overexpressed analysis set

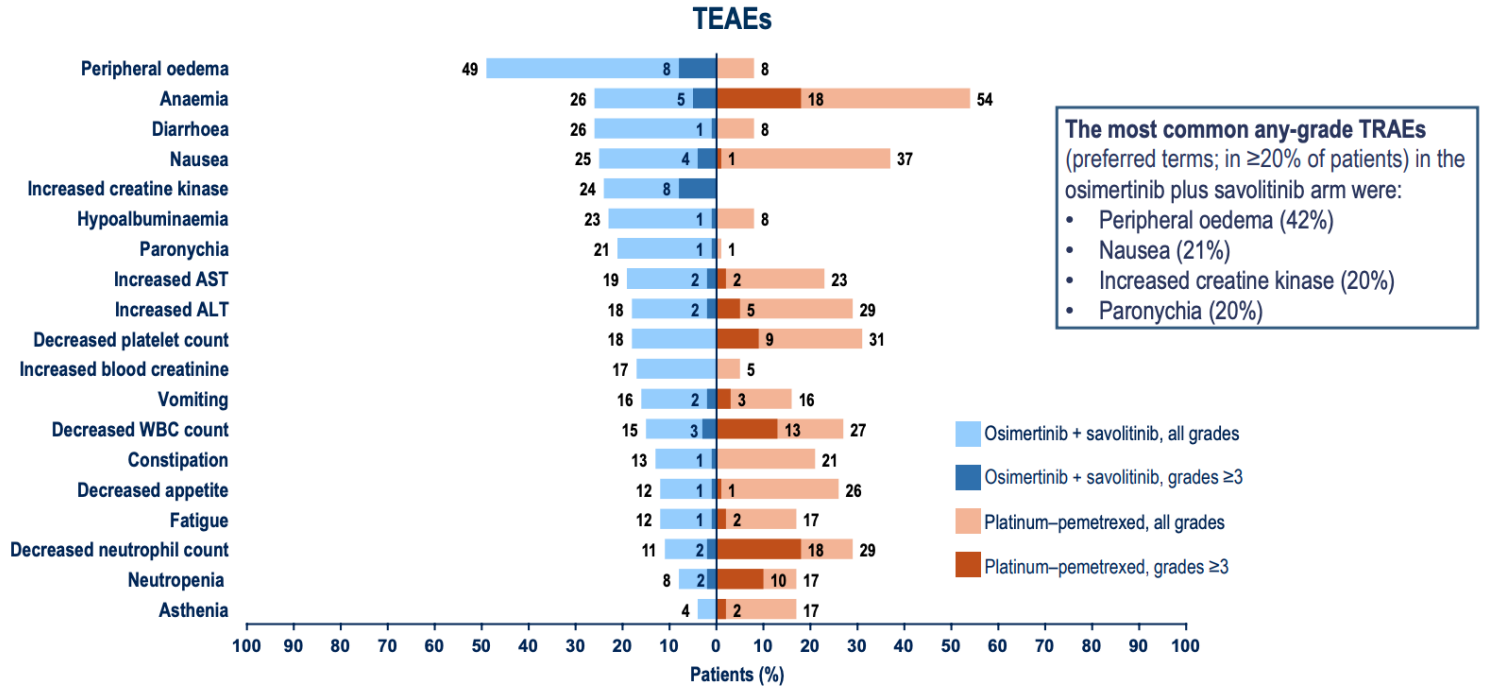


MET-amplified analysis set*

PFS by BICR in the MET-overexpressed and *MET*-amplified analysis sets was generally consistent with the full analysis set

Beneficio consistente en el análisis de subgrupos

All-causality adverse events in ≥15% patients*

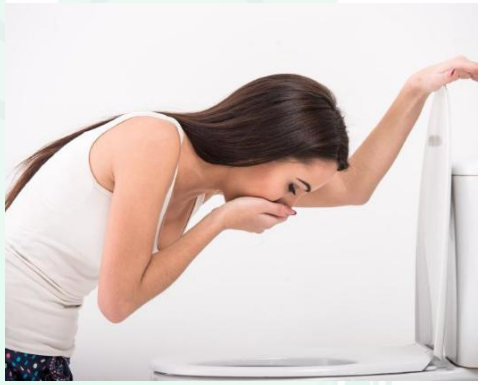


The most common any-grade TRAEs (preferred terms; in ≥20% of patients) in the osimertinib plus savolitinib arm were:

- Peripheral oedema (42%)
- Nausea (21%)
- Increased creatine kinase (20%)
- Paronychia (20%)

The most common TEAEs in the osimertinib plus savolitinib arm were peripheral oedema, anaemia and diarrhoea







- **Perfil de toxicidad para tener en cuenta.**
- Toxicidad grado ≥3: 64% frente 64%
- **Interrupción del tratamiento: 14% frente 8%**

Discusión del estudio





Beneficio consistente en SG y SLP

Alternativa oral frente a QT

Selección de pacientes (*MET+*)

Brazo comparador clásico

Diferencia en SLP modesta

Perfil de toxicidad dudoso

ESTUDIO OptiTROP-Lung04

ESTUDIO OptiTROP-Lung04

Resultados finales de SG con sacituzumab-tiromutecan en CPNM 2L *EGFR*mut frente a QT



- **Nueva posibilidad de secuencialidad de tratamiento**
- Objetivo primario: PFS BICR

OptiTROP-Lung04 Study Design

Randomized, multicenter, open-label, phase 3 trial (NCT05870319)

Key Eligibility

- ECOG score 0 or 1
- Nsq-NSCLC (stage IIIB/IIIC or stage IV)
- *EGFR*-sensitive mutations
- Progression after 3rd gen TKI therapy or progression after 1st or 2nd gen TKIs with negative T790M

R 1:1

N=376

Sac-TMT
5 mg/kg IV, Q2W
(n=188)

- **Pemetrexed 500 mg/m² + Carboplatin AUC 5 or Cisplatin 75 mg/m² Q3W for up to 4 cycles**
- **Pemetrexed 500 mg/m² maintenance, Q3W**

(n=188)

Primary endpoints*

- PFS assessed by BICR

Secondary endpoints*

- **OS (key secondary endpoint)**
- PFS assessed by investigator
- ORR, DCR, DOR, etc.
- Safety

Treatment until disease progression, intolerable toxicity, or patient request to discontinue treatment.

Stratification factors:

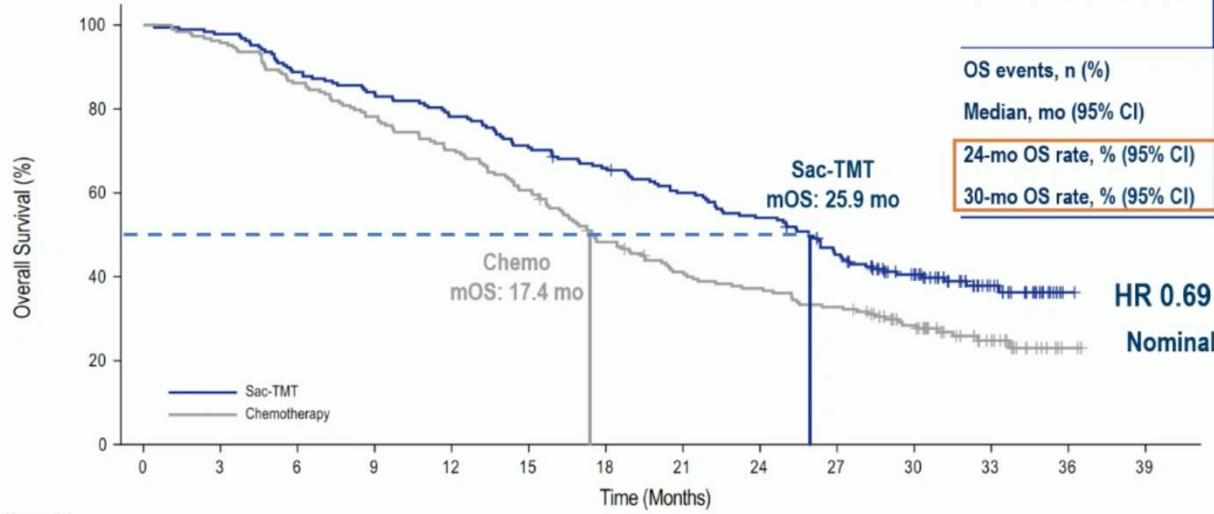
1. Prior EGFR-TKI therapy
   (**3rd gen TKI in 1st line** *vs* **in 2nd line** *vs* **no 3rd gen TKI**)
2. Brain metastases (**yes** *vs* **no**)

Pre-specified final analysis for OS:

- approximately 250 OS events have occurred

Overall Survival

Sac-TMT demonstrated a sustained OS benefit over chemotherapy, with a 31.0% reduction in the risk of death, corresponding to an 8.5-month difference in median OS between the two groups.



	Sac-TMT (n = 188)	Chemo (n = 188)
OS events, n (%)	114 (60.6)	137 (72.9)
Median, mo (95% CI)	25.9 (22.2, 27.5)	17.4 (15.7, 20.3)
24-mo OS rate, % (95% CI)	54.1 (46.6, 60.9)	37.2 (30.3, 44.2)
30-mo OS rate, % (95% CI)	40.5 (33.4, 47.5)	28.4 (22.0, 35.2)

HR 0.69 (95% CI: 0.54–0.89)

Nominal one-sided *P* = 0.0021

Median follow-up: 32.3 months

Beneficio en pacientes que progresan a 1L.

Beneficio en L858R

Beneficio en mts multinivel

MAL PRONÓSTICO

SUPERVIVENCIA GLOBAL

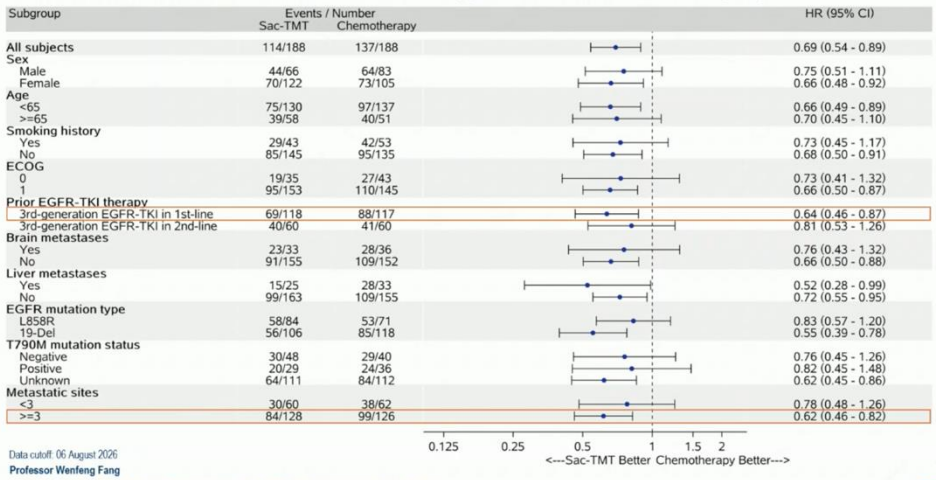
25,9 meses frente 17,4 meses

2 años: 54% frente 37%

30 meses: 40,5% frente 28,4%

Overall Survival: Subgroup Analyses

A consistent OS benefit of sac-TMT over chemotherapy was observed across all subgroups.

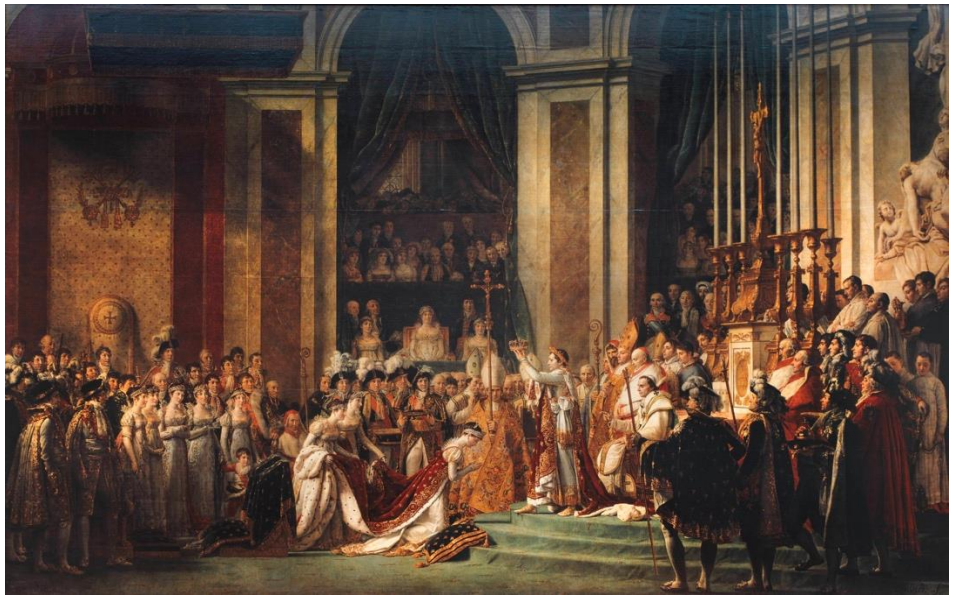


Data cutoff: 06 August 2026

Professor Wenfeng Fang

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Discusión del estudio





Confirma datos en SG

Perfil de toxicidad manejable

Baja tasa de eventos adversos G≥3

Brazo comparador clásico ¿QT-amivantamab?

¿Solo pacientes de mal pronóstico?

Necesidad de biomarcadores

ESTUDIO PANKU-Lung01 y HERON-Lung01

PANKU-Lung01 Study Design

PANKU-Lung01 study is an open-label, multicenter, randomized, phase III study

Key eligibility criteria

- Locally advanced or metastatic non-squamous NSCLC
- Documented *EGFR* sensitizing mutant (exon 19 deletion or L858R)
- Progressed on prior 3rd-generation EGFR TKI therapy (up to 2 prior lines of EGFR TKI)
- Treated/stable CNS metastasis allowed
- Measurable lesion per RECIST v1.1
- ECOG PS 0-1

R 1:1
N=432

- **Iza-bren** 2.5 mg/kg D1D8 Q3W
- **PBC** Pemetrexed + Cisplatin/Carboplatin Q3W

Stratified by

- ECOG PS (0 vs 1)
- CNS metastases (Yes vs No)
- Prior anti-angiogenic therapy (Yes vs No)

Treatment until

Disease progression per RECIST v1.1 or intolerable toxicity

Primary endpoint: PFS (BICR)
Key secondary endpoint: OS

H_{PFS} α=0.025 → H_{OS} α=0

Other secondary endpoints

- PFS (INV)
- ORR (BICR & INV)
- DCR (BICR & INV)
- DOR (BICR & INV)
- Safety, PK, Immunogenicity

Note: Iza-bren was dosed with 10% compensation per protocol; pemetrexed 500 mg/m² plus cisplatin 75 mg/m² or pemetrexed 500 mg/m² plus carboplatin (AUC 5) Q3W.

ECOG PS, Eastern Cooperative Oncology Group performance status; **Q3W**, every 3 weeks; **AUC**, area under the curve; **CNS**, central nervous system
PFS, progression-free survival; **BICR**, blinded independent central review; **OS**, overall survival; **INV**, investigator assessed; **ORR**, objective response rate (confirmed); **DCR**, disease control rate; **DOR**, duration of response.

Izalontamab brengitecan

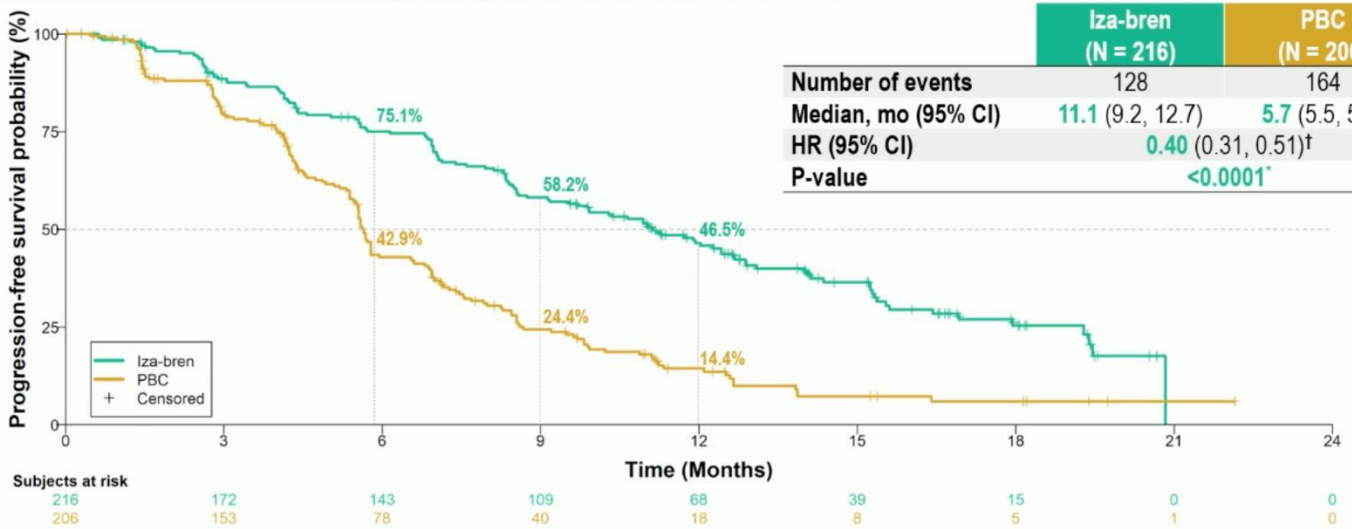
ADC biespecífico *EGFR/HER3*

- SLP 11,1m frente 5,7m, HR=0,40
- SG inmadura (NA frente 19,4m)
- **Toxicidad G≥3: 78,7% frente 62,1%**

>>> Principal toxicidad mieloide

PFS by BICR: Primary Endpoint

Iza-bren showed statistically significant and clinically meaningful improvement vs PBC with doubling of mPFS.



	Iza-bren (N = 216)	PBC (N = 206)
Number of events	128	164
Median, mo (95% CI)	11.1 (9.2, 12.7)	5.7 (5.5, 5.9)
HR (95% CI)	0.40 (0.31, 0.51)†	
P-value	<0.0001*	

PFS (BICR) was evaluated in the mITT population. The median duration of follow-up for PFS was 14.6 months.
Similar results were observed for PFS (BICR) in the ITT population, with 129 events in the iza-bren arm and 164 events in the PBC arm. The hazard ratio (95% CI) was 0.40 (0.31, 0.51).
†Based on a stratified Cox regression model. *One-sided, based on a stratified log-rank test.

Li Zhang

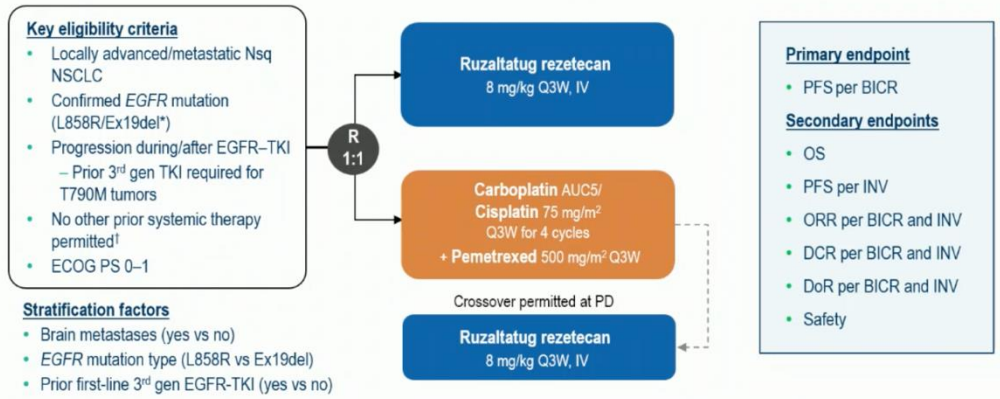
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Study Design

❖ HERON-Lung01: an open-label, randomized, phase 3 trial (NCT06671379)

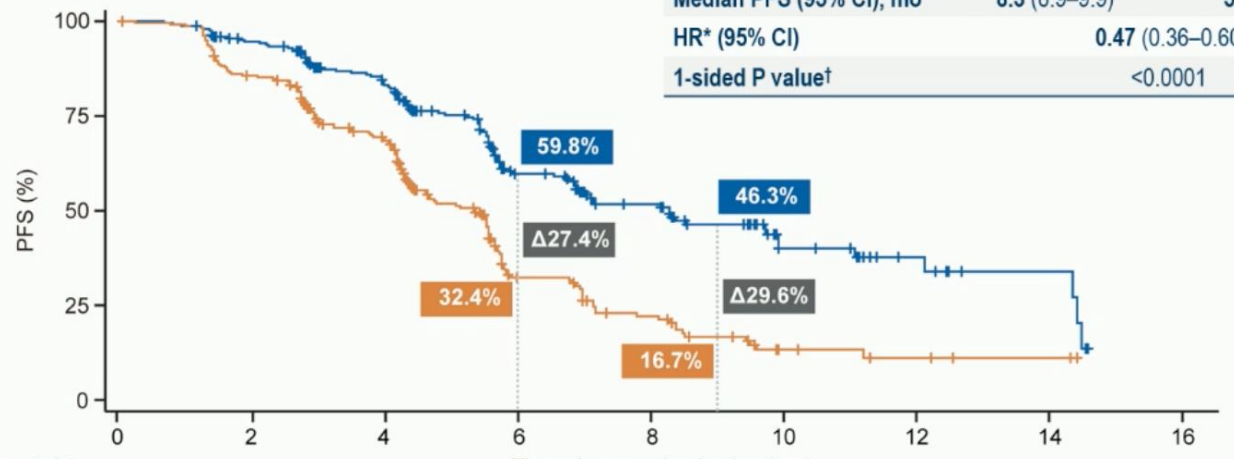


Ruzaltatug rezetecan

ADC *HER3*

PFS per BICR

	Ruzaltatug rezetecan (n = 250)	Chemo (n = 248)
Event, n (%)	108 (43.2)	156 (62.9)
Median PFS (95% CI), mo	**8.3** (6.9–9.9)	**5.4** (4.4–5.6)
HR* (95% CI)	**0.47** (0.36–0.60)	
1-sided P value†	<0.0001	



- Consistent PFS benefit per investigator assessment: HR **0.46** (95% CI 0.36–0.60)

- **SLP 8,3m frente 5,4m, HR=0,47**
- SG inmadura (HR=0,70, p>0,05)

>>> Entrecruzamiento permitido

- Toxicidad G≥3: 40,7% frente 38,9%

>>> Perfil de toxicidad más favorable

Investigación traslacional con ctDNA: ALK y EGFR

Papel del ctDNA en la aparición de resistencia a lorlatinib (CPNM *ALK*+ 1L)

Aparición de resistencias mediante las vías de angiogénesis, RAS e inflamación

Resistance mechanism	Genes[c]	Patients with altered genes, n (%)[d]
DNA damage repair	*ATM, BRCA2, CHEK2, XRCC2*	6/17 (35%)
Inflammatory-response	*AXL, IL1R1, LYN, MET, MYC, and ROS1*	6/17 (35%)
RTK/RAS/MAPK	*FLT1/VEGFR1, KRAS, MAP2K1, MET, NTRK1*	6/17 (35%)
Oncogene amplification	*MET, MYC, NOTCH2*	5/17 (29%)
Tumor suppressor	*BRCA2, FAT1, LRP5, RB1*	5/17 (29%)
Angiogenesis	*FLT1/VEGFR1*	3/17 (18%)
Epigenetic regulation	*HDAC6*	2/17 (12%)

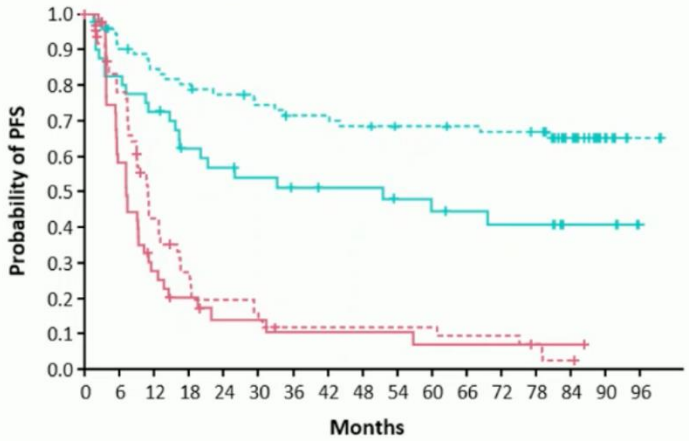
MAYOR HETEROGENEIDAD TUMORAL
→ RESISTENCIA PRECOZ

***TP53*wt**

Respuesta prolongada que no se correlaciona en crizotinib

7-year PFS by baseline *TP53* mutation status

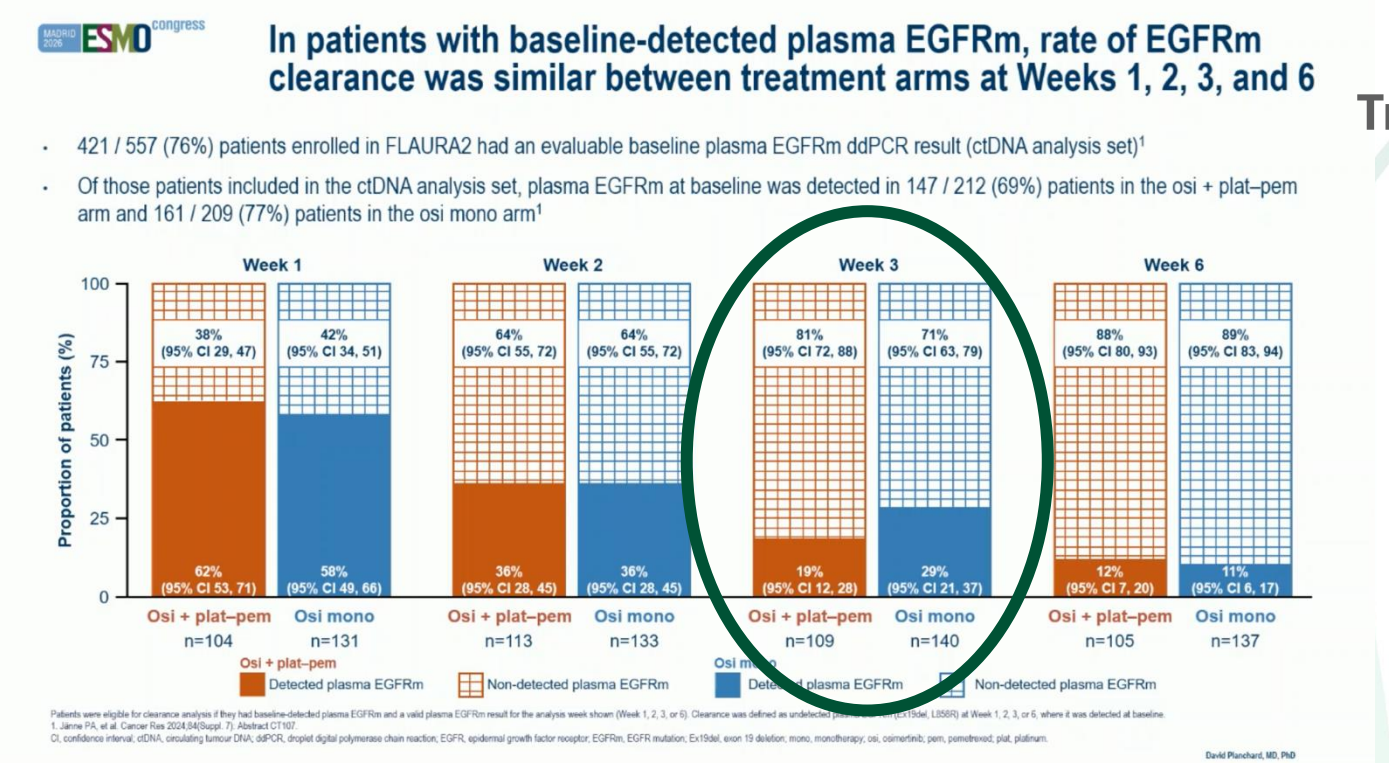
- Lorlatinib showed prolonged PFS vs crizotinib regardless of baseline *TP53* mutation status
- In the lorlatinib group, longer PFS was observed in the *TP53* mutation–negative vs mutation–positive subgroup; in the crizotinib group, PFS was short regardless of baseline *TP53* status



	Lorlatinib		Crizotinib	
	TP53 mut (–) n=73	*TP53* mut (+) n=43	*TP53* mut (–) n=65	*TP53* mut (+) n=44
PFS, median (95% CI), months	NR (NR-NR)	51.6 (16.4-NR)	10.9 (9.0-12.8)	7.2 (5.6-9.3)
7-year PFS (95% CI), %	65.0 (54.5-77.4)	40.8 (27.3-61.1)	2.3 (0.3-15.7)	6.9 (1.9-24.5)
HR (95% CI), mut (–) vs mut (+)	0.48 (0.268-0.857)		0.78 (0.513-1.186)	

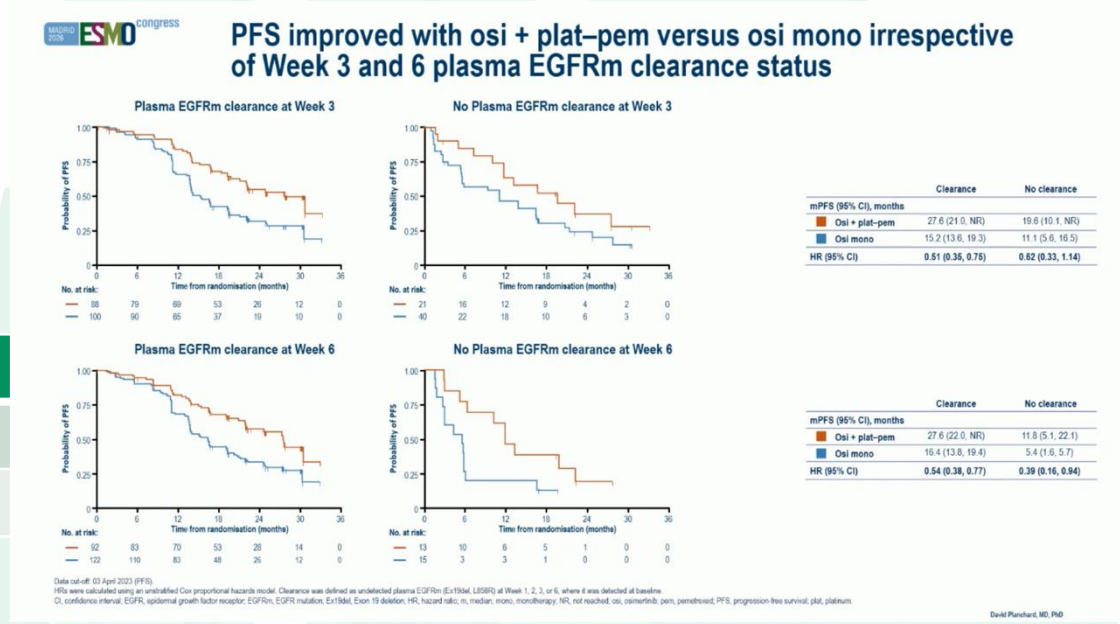
Aclaramiento del ctDNA en la respuesta a QT-osimertinib (CPNM *EGFR*mut 1L)

El aclaramiento de ctDNA en las tres primeras semanas es biomarcador pronóstico (no predictivo)



Tras seis semanas de tratamiento alrededor del 90% de los pacientes aclara el ctDNA.

→ La diferencia de aclaramiento es durante las tres primeras semanas.



ctDNA (3 semanas)	Osimertinib + QT	Osimertinib
Aclarado	27,6 meses	15,2 meses
Persistente	19,6 meses	11,1 meses

Conclusiones

Conclusiones

***KRAS* G12C:** Los resultados de KRAScendo-1 muestran una mejoría significativa en supervivencia con divarasib frente a los inhibidores disponibles, consolidando la inhibición de KRAS como una estrategia terapéutica en evolución.

EGFRmut: La inhibición dual *EGFR/MET* y los nuevos ADC han demostrado resultados relevantes tras la progresión a osimertinib. Sin embargo, la selección molecular, los perfiles de toxicidad y la secuenciación óptima de estas estrategias continúan siendo cuestiones importantes por resolver.

Investigación traslacional: Los análisis de ctDNA en *EGFR* y *ALK* permiten profundizar en los mecanismos de resistencia y en la identificación de pacientes con diferente pronóstico.

Gracias