

Redefining the Treatment Landscape of Small-Cell Lung Cancer

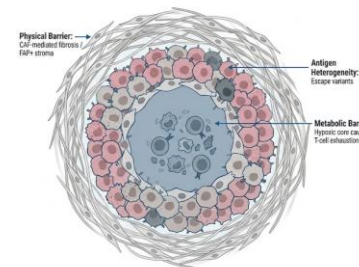
From four decades of platinum–etoposide to a DLL3-anchored, subtype-directed era

Sun-Ha Choi, MD

Kyungpook National University

THE PROBLEM

Small-Cell Lung Cancer — a Hard Disease to Treat



~15%

of all lung cancers

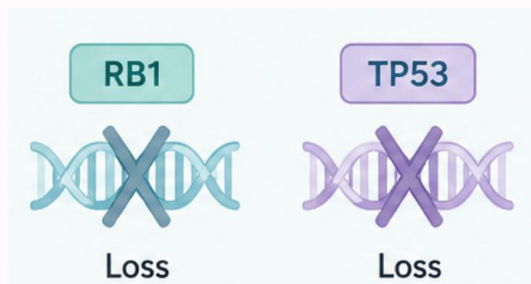
~70%

extensive-stage at diagnosis

~3%

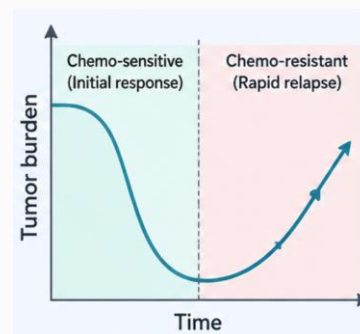
5-yr survival (extensive-stage)

Biology



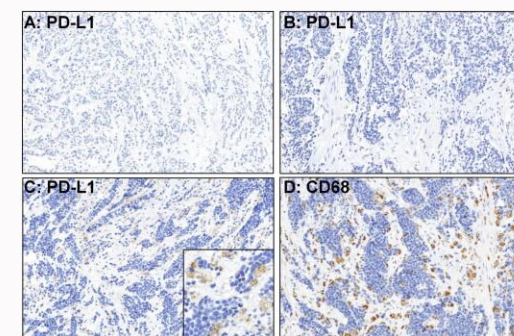
Defined by biallelic loss of RB1 and TP53 — with no targetable driver mutation.

Kinetics



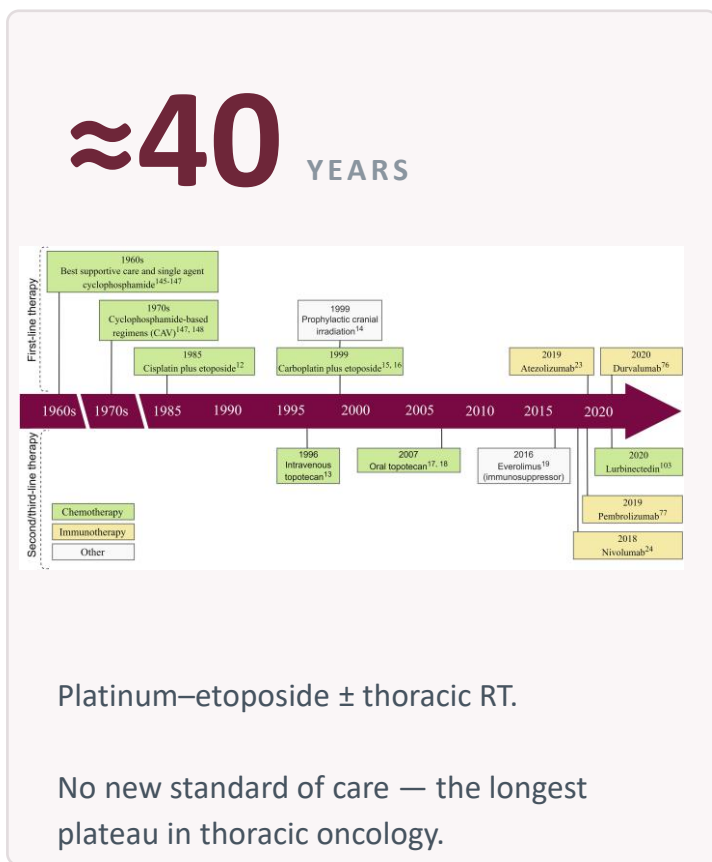
Highly chemo-sensitive up front, but rapid relapse with chemo-resistant disease.

Immunology



Neuroendocrine and often immunologically “cold” — limited benefit from ICIs alone.

Four Decades of Silence — Then Three Positive Phase 3 Trials



ALREADY CHANGED · POSITIVE PHASE 3

LIMITED-STAGE

ADRIATIC

Consolidation durvalumab

ES · 1L MAINTENANCE

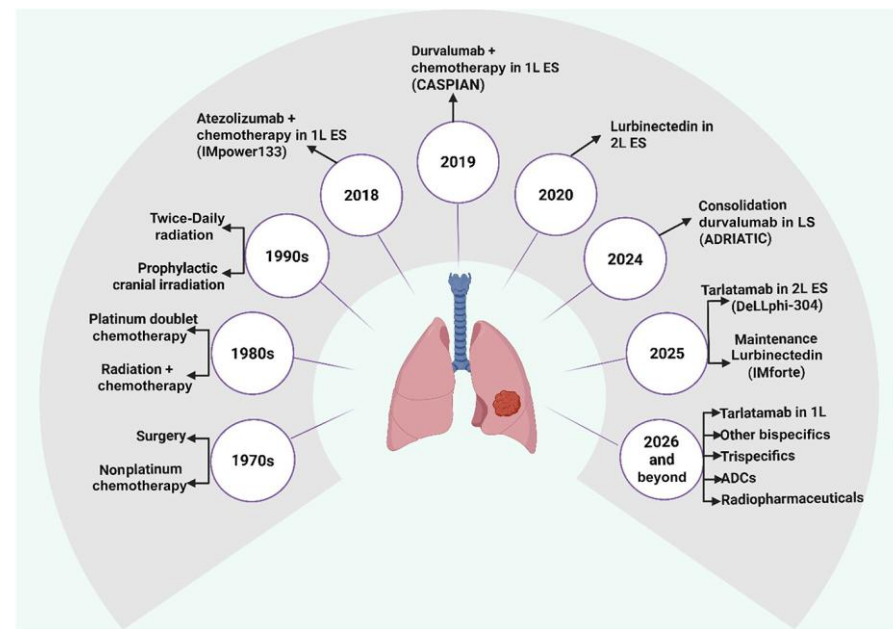
IMforte

Lurbinectedin + atezolizumab

ES · 2L / RELAPSE

DeLLphi-304

Tarlatamab — DLL3 × CD3 BiTE



WHAT IS COMING

New targets & modalities → The contest for first line → Selection by biology

The Standard of Care Before ADRIATIC

Concurrent CRT

4× platinum–etoposide with thoracic RT



± PCI

Prophylactic cranial irradiation



Observation

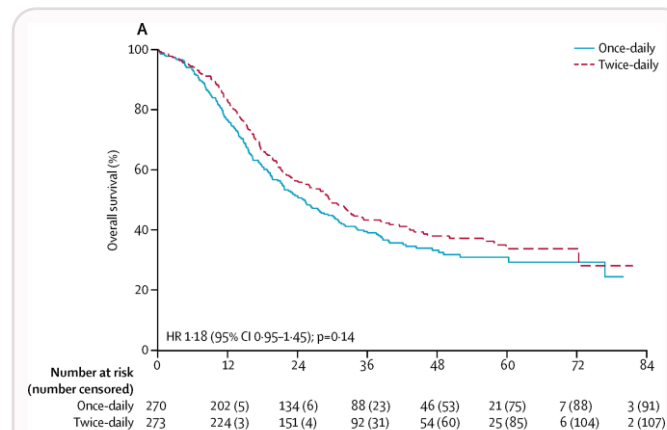
...then watchful waiting

29–34%

5-year overall survival

< 2 yrs

most relapses occur within



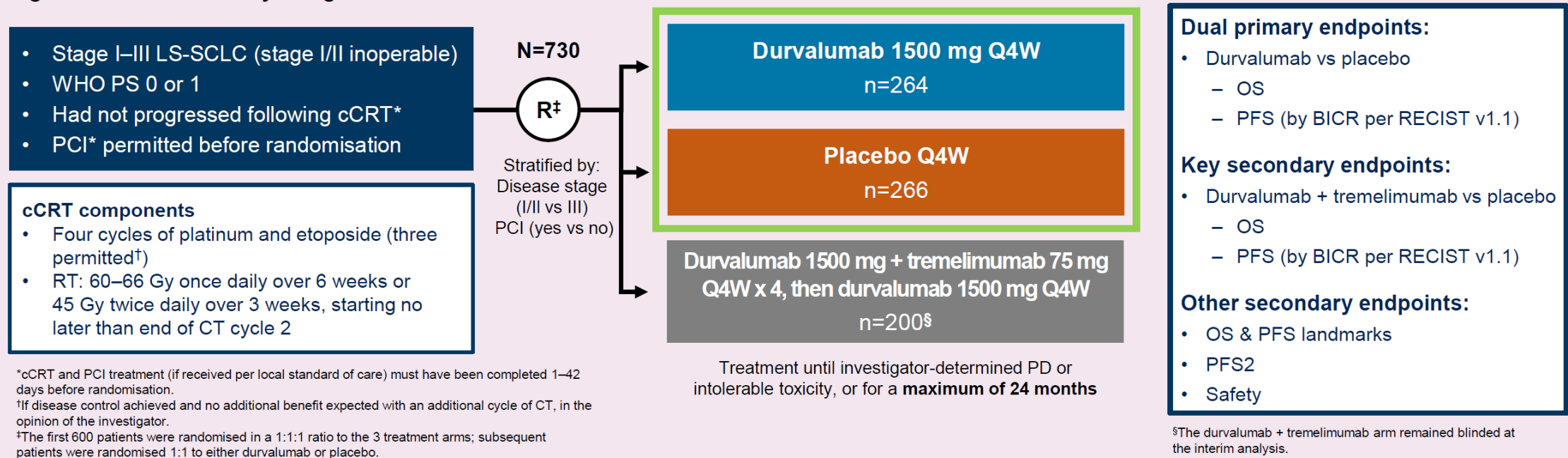
No new option for decades — a clear ceiling on cure.

ADRIATIC

The effects of CRT may have a priming effect on tumors for response to anti PD-1/PD-L1 therapy

The phase III ADRIATIC trial is evaluating durvalumab ± tremelimumab in patients with Stage I-III LS-SCLC who have not progressed following cCRT.

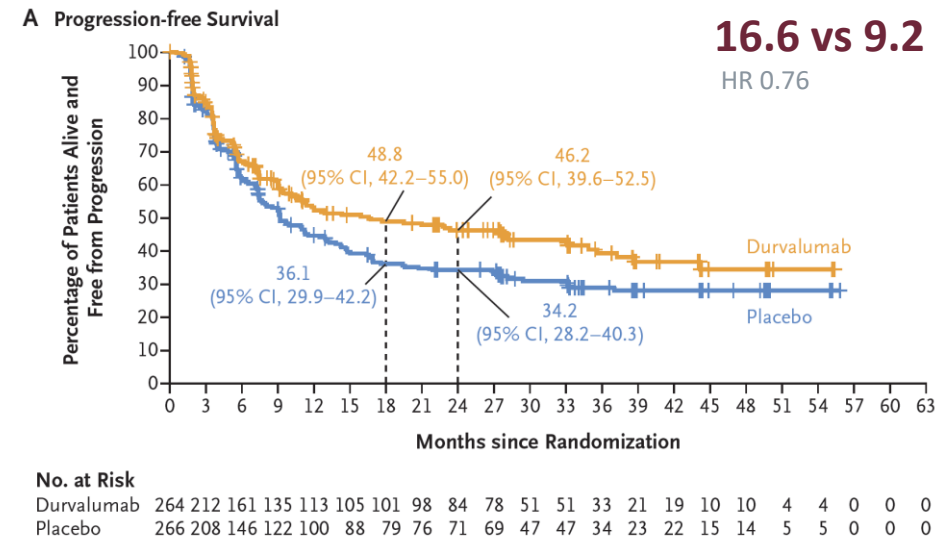
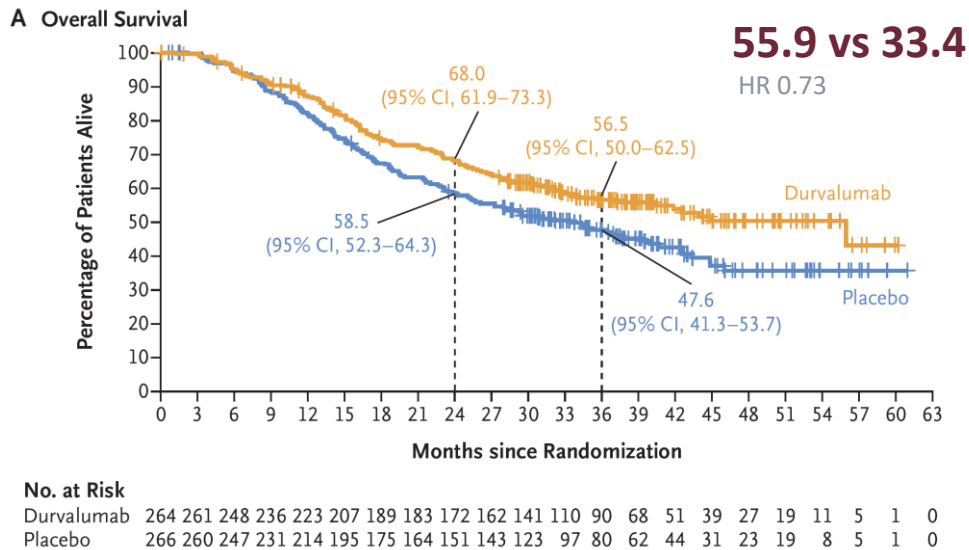
Figure 1. ADRIATIC study design



Sequential design — durvalumab starts after chemoradiotherapy is complete.

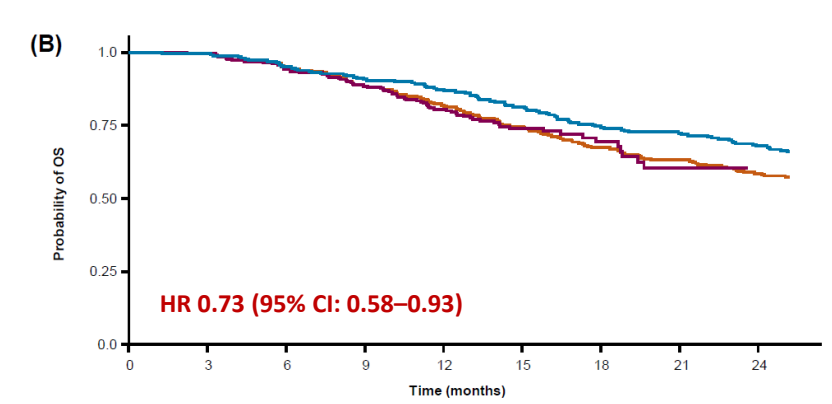
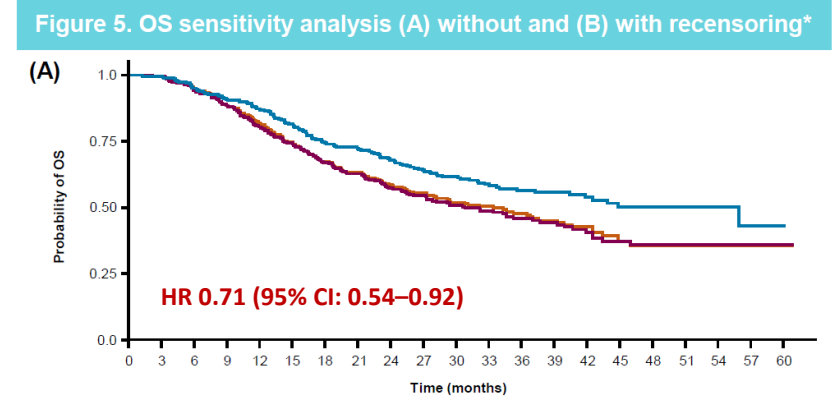
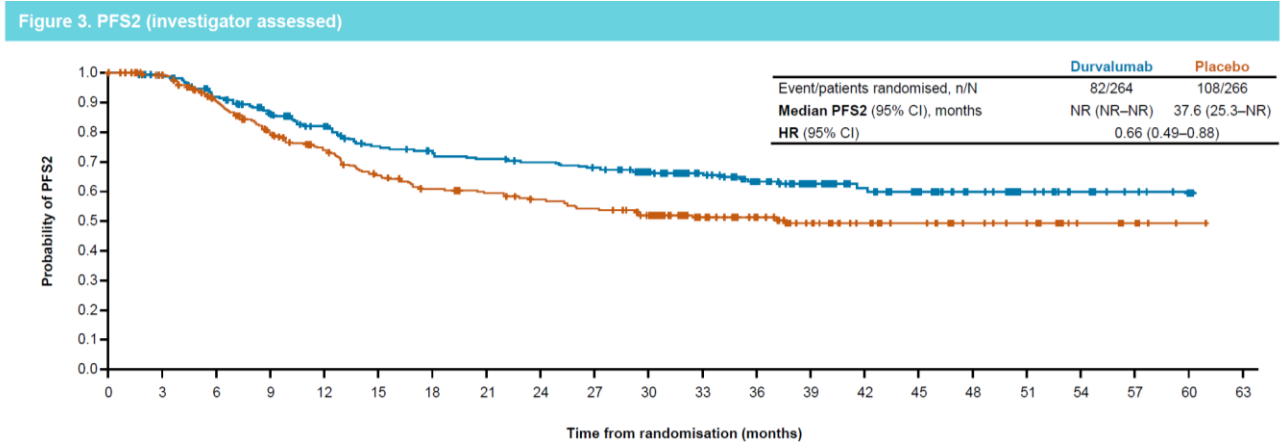
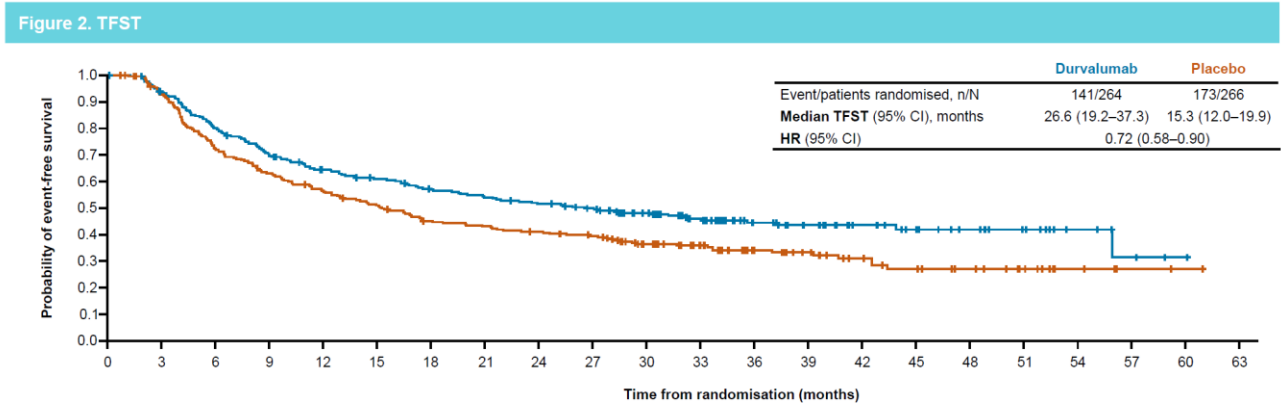
NRG-LU005 later showed that giving atezolizumab concurrently with cCRT did not improve survival, which retrospectively supports this choice.

Consolidation Durvalumab Improves Survival



- The PFS benefit remained significant in patients without PCI and was generally consistent across most subgroups.
- Durvalumab consolidation for up to 2 years was well tolerated, with no new safety signals identified after cCRT.
- By platinum — OS HR 0.56 with carboplatin vs 0.82 with cisplatin; by RT schedule 0.72 (QD) vs 0.68 (BID). No significant treatment-by-subgroup interaction, and the subgroups differed in baseline composition. — hypothesis-generating, not a selection rule.

Subsequent treatment patterns and impact (ELCC 2026)



What the Pathway Looks Like Now



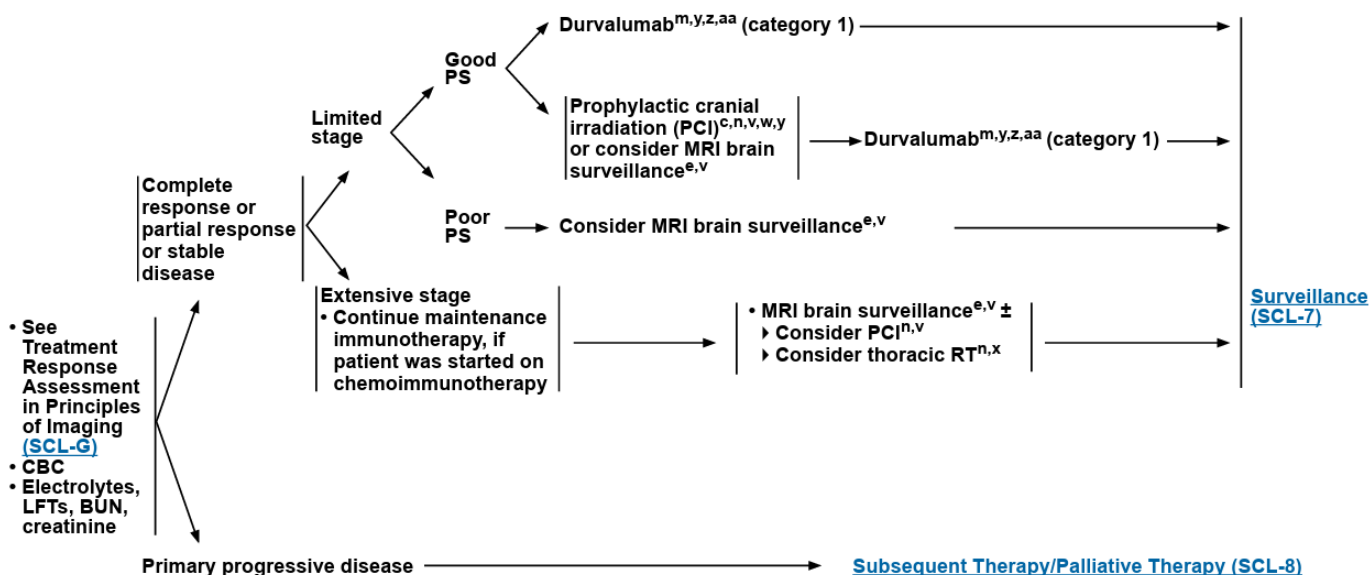
National
Comprehensive
Cancer
Network®

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NCCN Guidelines Version 1.2027
Small Cell Lung Cancer

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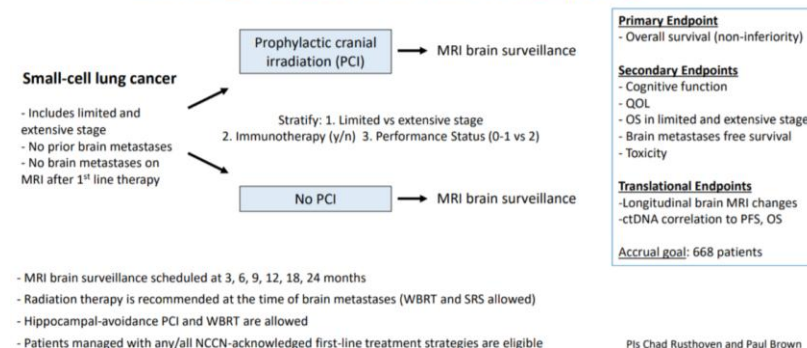
RESPONSE ASSESSMENT FOLLOWING
PRIMARY TREATMENT

ADJUVANT THERAPY



PCI — or MRI surveillance

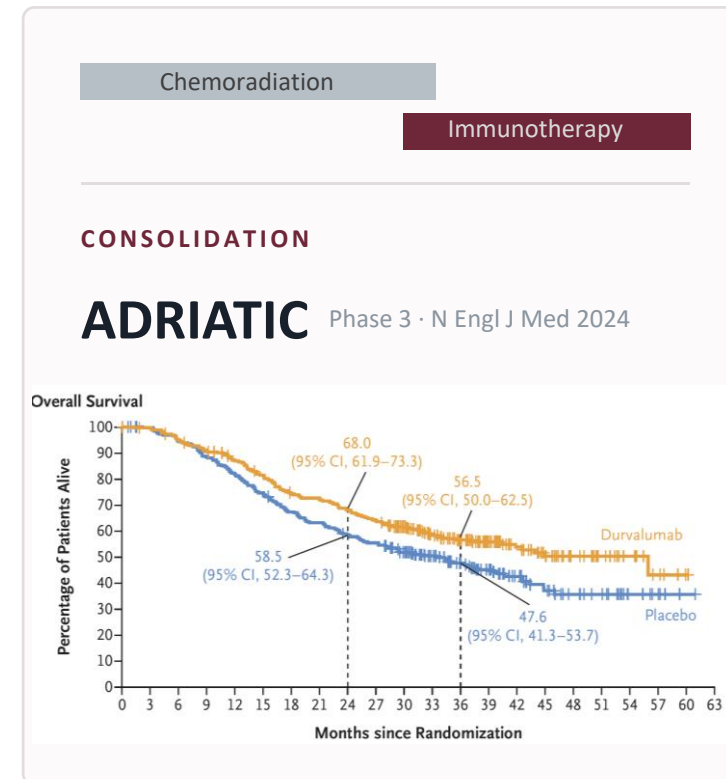
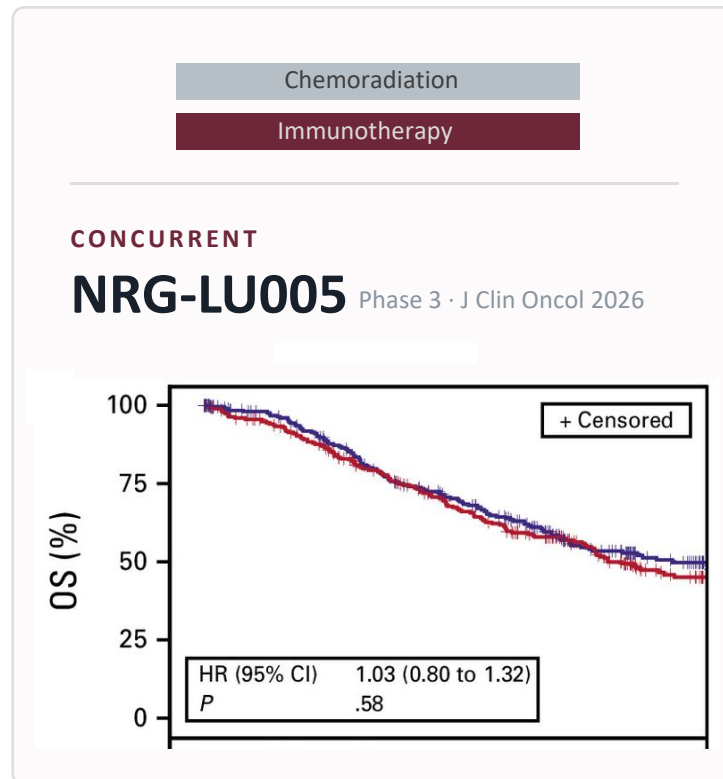
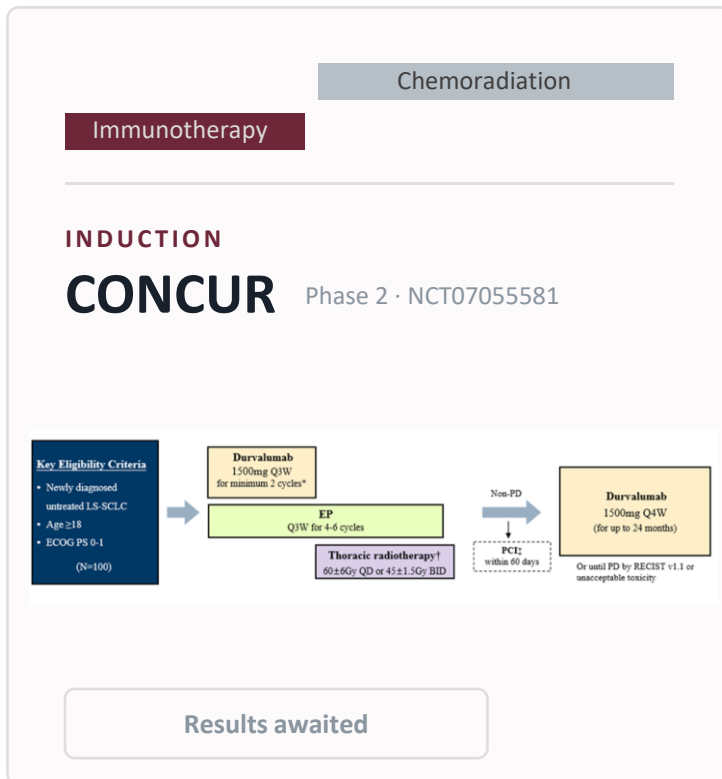
MAVERICK (SWOG 1827): MRI Brain Surveillance Alone Versus MRI Surveillance and Prophylactic Cranial Irradiation: A Randomized Phase III Trial in Small-Cell Lung Cancer



PCI is not recommended in patients with poor PS or impaired neurocognitive function. Increased **cognitive decline after PCI** has been observed in older adults (≥60 years) in prospective trials; the risks and benefits of PCI versus close brain surveillance, MRI (preferred) or CT with contrast, should be carefully discussed with these patients.

Still unsettled — whether PCI can be replaced by MRI surveillance (MAVERICK, SWOG 1827), and whether immunotherapy should start earlier (CONCUR).

When Should Immunotherapy Enter?



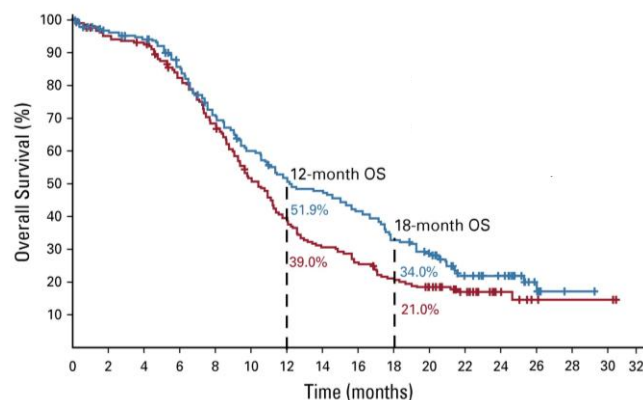
Why sequencing may matter — thoracic radiotherapy irradiates mediastinal lymphoid tissue and depletes the circulating lymphocytes that checkpoint blockade depends on.

Three Trials, One Conclusion — and One Near Miss

IMpower133

Atezolizumab + carboplatin–etoposide

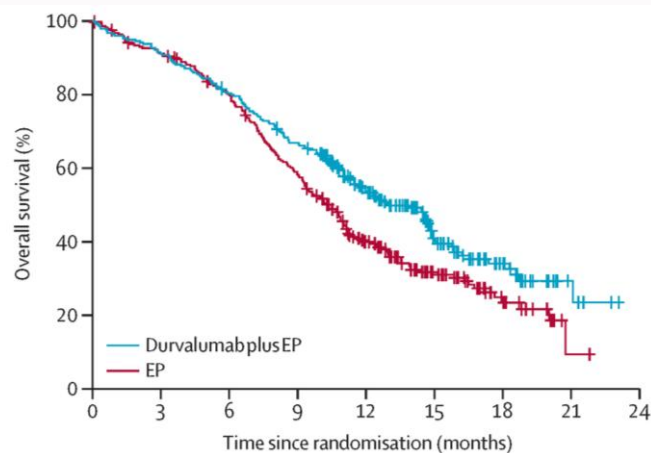
HR 0.70 $p = .007$ · $n = 403$ **12.3 vs 10.3**



CASPIAN

Durvalumab + platinum–etoposide

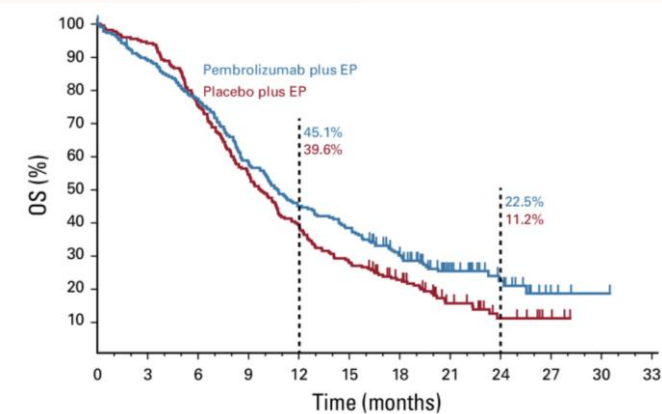
HR 0.73 $p = .0047$ · $n = 805$ **13.0 vs 10.3**



KEYNOTE-604

Pembrolizumab + platinum–etoposide

HR 0.80 $p = .0164$ vs boundary .0128 **10.8 vs 9.7**

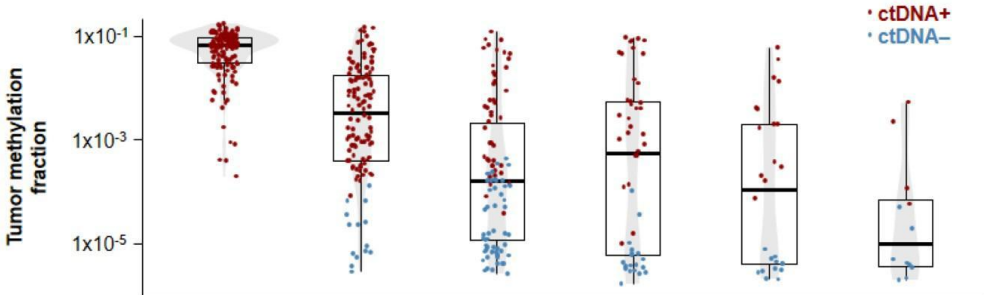
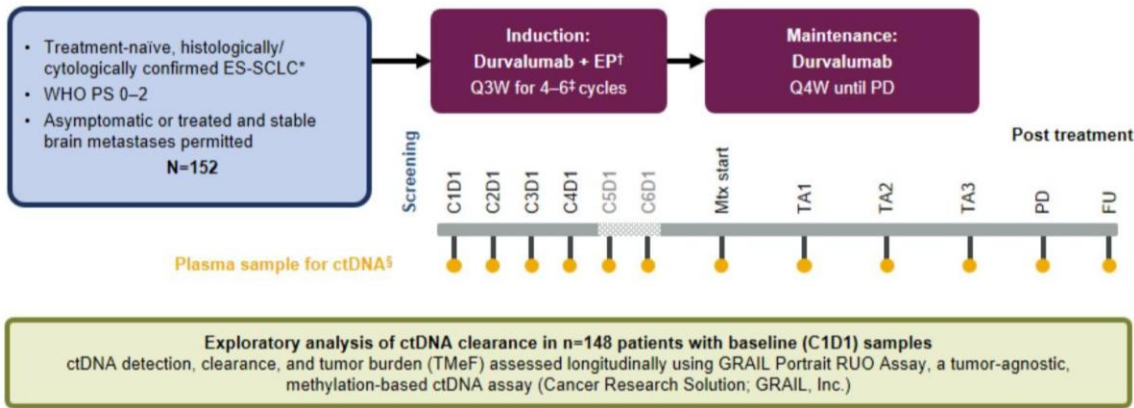


The hazard ratios sit between 0.70 and 0.80 with overlapping confidence intervals — the same modest benefit, observed three times. What separated them was alpha allocation, not biology.

Which leaves the harder question — does a benefit this size hold up outside a trial population?

LUMINANCE

Phase 3b, single-arm, multicenter, international study (NCT04774380)



	C1 (baseline)	C2	Mtx start	TA1	TA2	TA3
Total, n	148	126	98*	50	26	12
ctDNA+, n	148	112	45	31	14	4
ctDNA-, n	0	14	53*	19	12	8
% Detected	100%	89%	46%	62%	54%	33%

13.1 mo

median OS (95% CI 10.1–NE)

55.6%

12-month OS

65.8%

confirmed ORR

5.1mo

median DoR (95% CI 4.9–5.3)

ctDNA — an exploratory read-out

Methylation-based, tumour-agnostic assay in the 148 patients with baseline samples. Detection fell from 100% at baseline to 46% by the start of maintenance, and 54% of patients cleared ctDNA over that interval — tumour methylation fraction fell in parallel.
Median follow-up was 8.6 months, so the survival figures are still immature.

Timepoints include only patients free of progression — attrition bias.

TAXIO — Can Etoposide Come Out of the Regimen?

THE HYPOTHESIS

Etoposide may blunt lymphocyte activation — so the chemotherapy backbone could be working against the immunotherapy it is paired with.

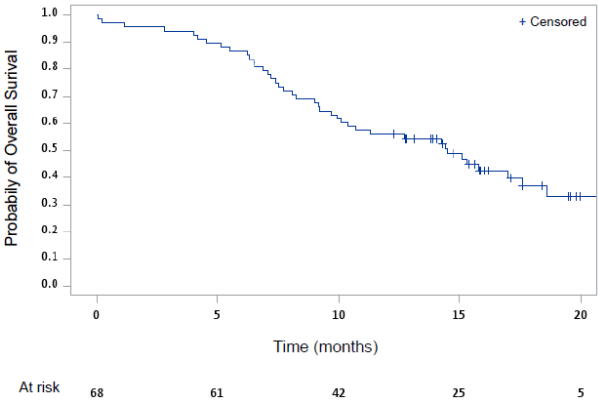
THE REGIMEN · PCD

Paclitaxel 200 mg/m² + carboplatin AUC6 + durvalumab, q3w × 4 induction cycles → durvalumab q4w until progression. One-day administration.

Overall response rate assessed by investigator (ORR)

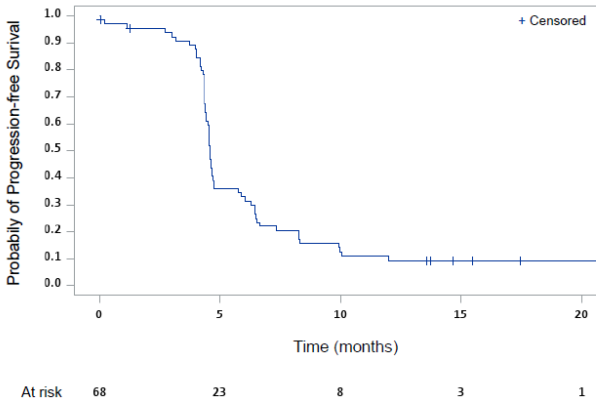
	N = 68
Complete Response	2 (2.9%) [0.0 - 7.0]
Partial Response	54 (79.4%) [69.8 - 89.0]
Objective Response	56 (82.4%) [73.3 - 91.4]
Stable Disease	5 (7.4%) [1.1 - 13.6]
Disease Control	61 (89.7%) [82.5 - 96.9]
Progressive Disease	0
Not evaluable	7 (10.3%) [3.1 - 17.5]

Overall Survival (OS)



	N = 68
Event: N (%)	41 (60.3)
Median OS: months [95% CI]	14.5 [9.9-17.6]

Progression-Free Survival assessed by investigator (PFS)



	N = 68
Event: N (%)	58 (85.3)
Median PFS: months [95% CI]	4.6 [4.4-4.7]
6-month PFS: % [95% CI]	32.9 [21.8-44.4]
12-month PFS: % [95% CI]	9.4 [3.8-18.0]

Clearly active — 82% response, no primary progression. And simpler to give: one day per cycle.

Single-arm, 68 patients — and CASPIAN's 12-month OS was 54%. Despite 82% response, median PFS is 4.6 months: deep, not durable. Toxicity shifts rather than falls

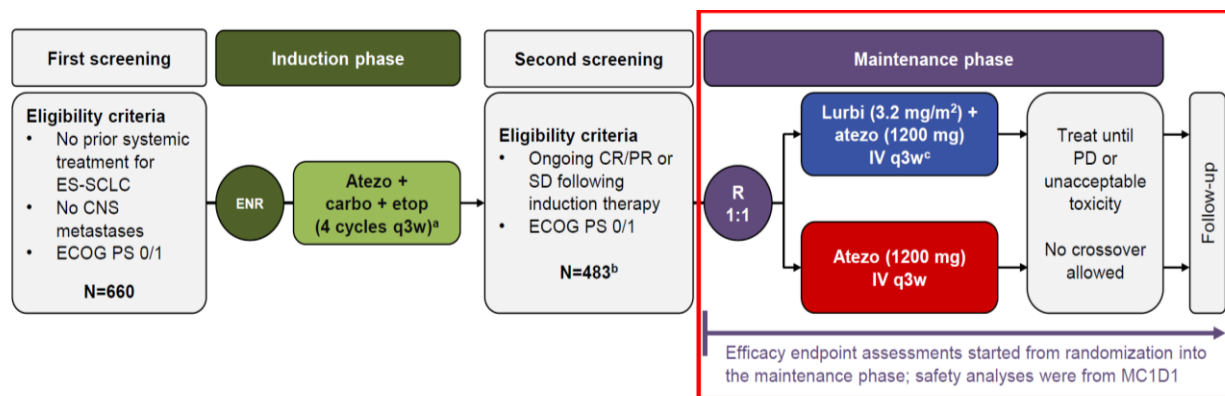
IMforte — the First Positive Maintenance Trial in SCLC

660 enrolled

> 4 cycles Atezo + carboplatin/etoposide induction

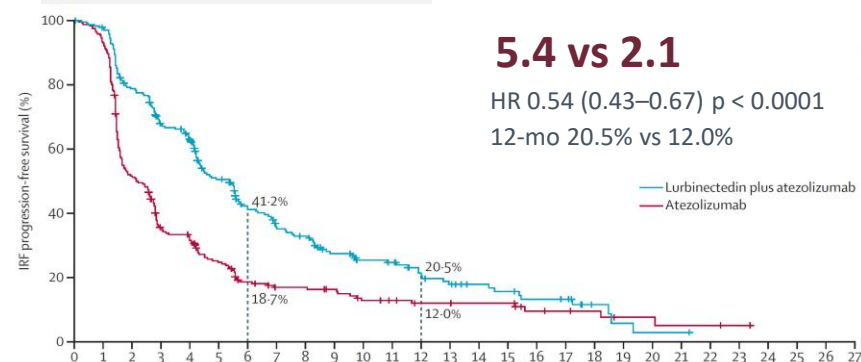
> 483 randomised 1:1
— only those without progression

> No crossover permitted

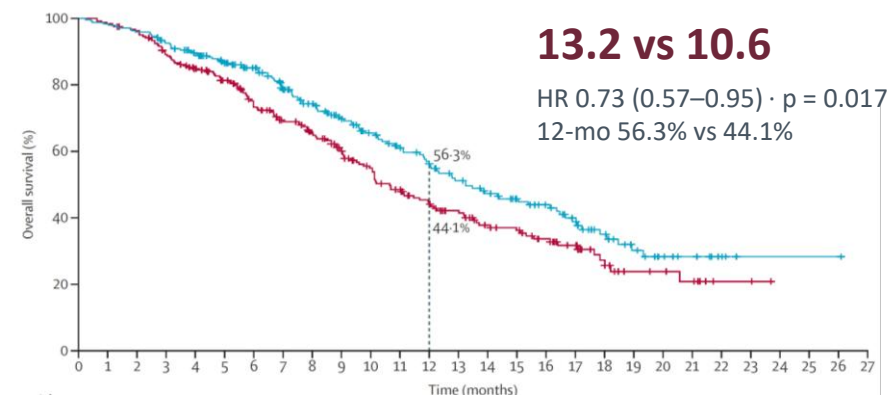


- Lurbinectedin added to atezolizumab maintenance significantly improved both IRF-PFS and OS, with benefit consistent across most prespecified subgroups. The first maintenance strategy in SCLC to move survival.
- **Grade ≥3 adverse events rose to 38% from 22% — higher, but with no new or unexpected safety signal for either drug.**
- **Only induction responders reached randomisation — 483 of 660.** Patients with CNS metastases were excluded. In clinic, a meaningful share never arrives at this decision point.

PROGRESSION-FREE SURVIVAL



OVERALL SURVIVAL



The Same Two Drugs, a Different Setting



MC1923 Phase II Clinical Trial of Durvalumab Plus Lurbinectedin in Platinum-Resistant Relapsed Extensive-Stage Small Cell Lung Cancer After Prior Chemoimmunotherapy (Cohort B)

Konstantinos Leventakos, MD, PhD¹, Anastasios Dimou, MD¹, Nathan R Foster, MS¹, Brandy L Jaszewski¹, Aaron S Mansfield, MD¹, Kaushal Parikh, MBBS¹, Janet L Lensing, CCRP¹, Lacey T Berning¹, Bobbi Jebens¹, Lynn M Flickinger, MA¹, Katherine Emilie Rhoades Smith, MD¹, Deepti Behl, MD¹, Mohamed Shanshal, MD¹, Antonious Z Hazim, MD², Anna J Schwecke, APRN, CNP, MS¹, Ailsa G Luce, PA-C¹, J Nicole Moffett, APRN, CNP, MSN¹, Ashley L Potter, PharmD, RPh, BCOP¹, Abdullah Al-Ajmi, PharmD, RPh, BCPS¹, Kadi M Dick, RN¹, Dena L Weinmann, RN¹, Julian R Molina, MD, PhD¹, Alex A Adjei MD, PhD³
¹Mayo Clinic, Rochester, MN; ²Mayo Clinic, Scottsdale, AZ; ³Cleveland Clinic, Cleveland, OH

Platinum-resistant relapse progression $\leq$ 3 months after last chemotherapy, all after chemo-immunotherapy

Durvalumab + lurbinectedin 1500 mg + 3.2 mg/m² q3w until progression

Phase 2, Simon optimal two-stage · N = 22 · ECOG 1 in 91% · 96% prior atezolizumab · median 4 cycles · median follow-up 21.1 months

PRIMARY ENDPOINT · 6-MONTH PFS

4 / 22

achieved · 18.2%

$\geq 7 / 22$

required · 32%

The prespecified efficacy threshold was not met.

FIGURE 1: PFS

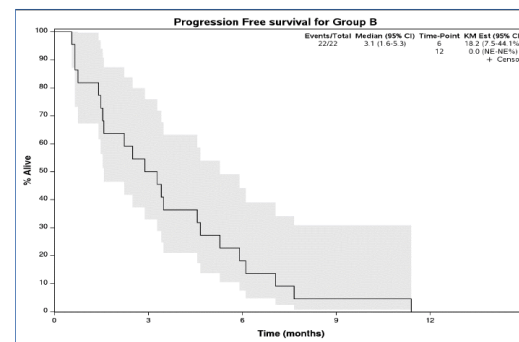
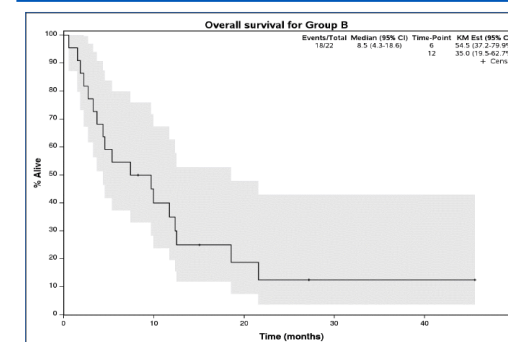


FIGURE 2: OS

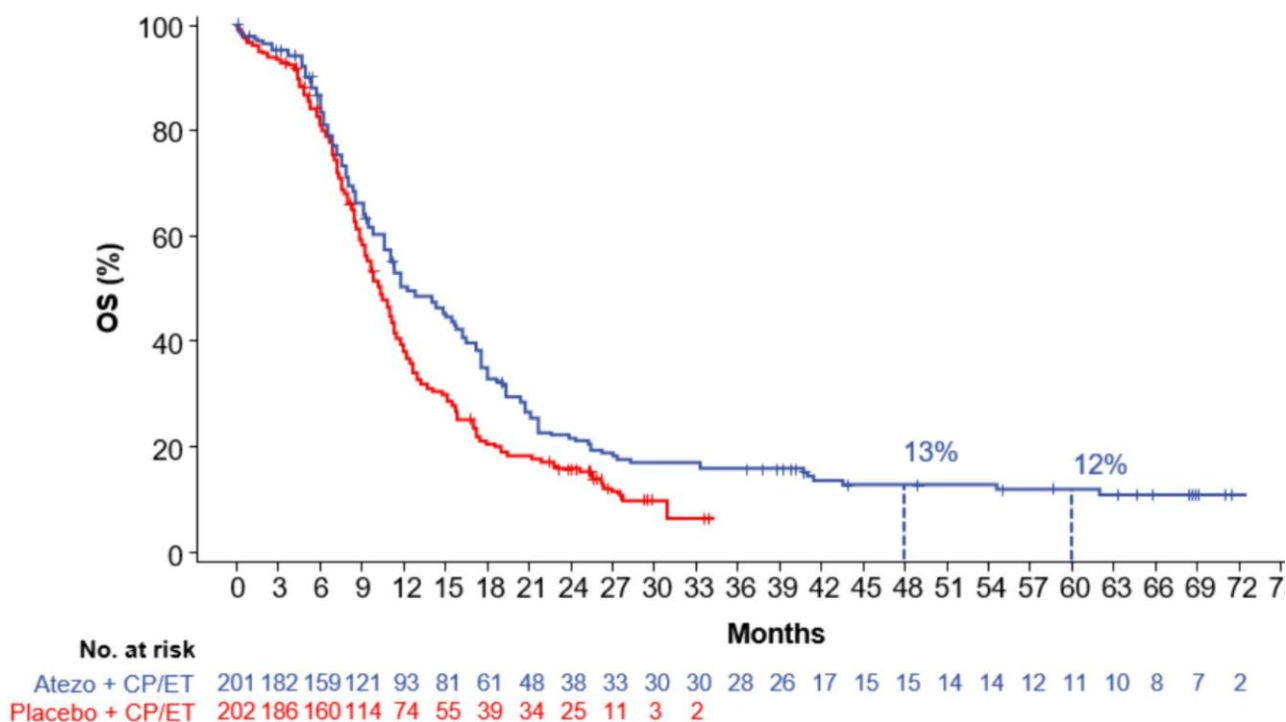


Maintenance, not salvage. The identical pairing was positive in IMforte — given before relapse, in patients still responding.

■ HOW MUCH HAS ACTUALLY CHANGED

The Plateau Is Broken — the Tail Is Still Thin

PhIII IMpower133, PhIII IMbrella A extension study



Long-term survivors exist now

IMbrella A extension: 3-, 4- and 5-year OS of 16%, 13% and 12%. CASPIAN 3-year OS 18%. Before 2019 this tail was essentially absent.

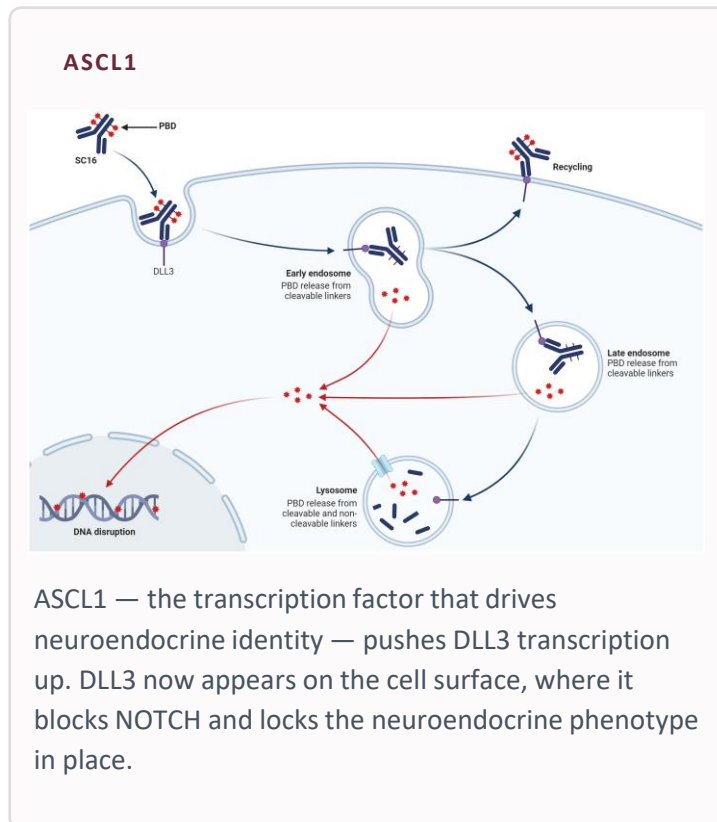
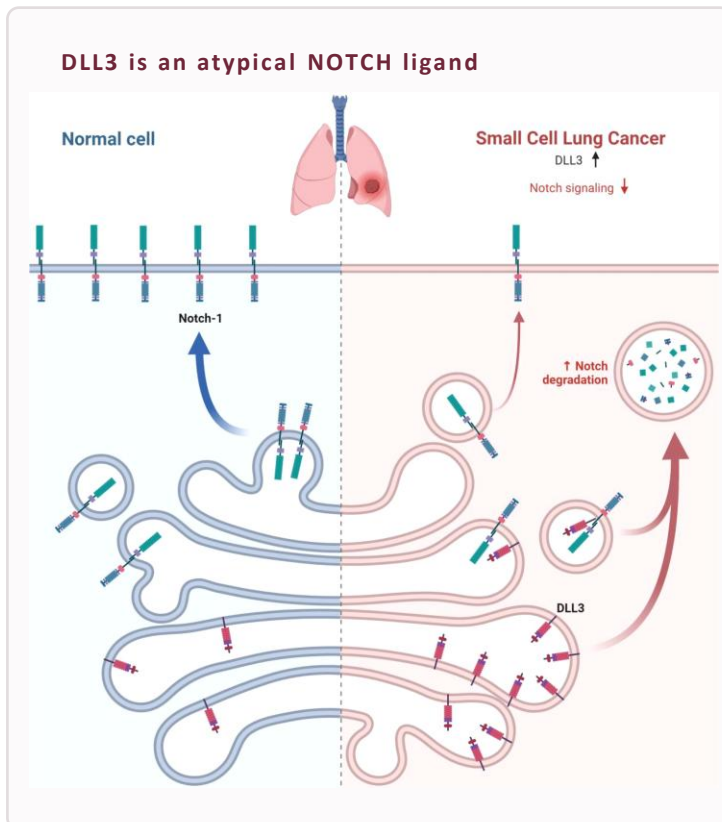
But the gains are narrow

IMforte: grade ≥ 3 adverse events 38% vs 22%, and patients with brain metastases were excluded — limiting who the benefit applies to.

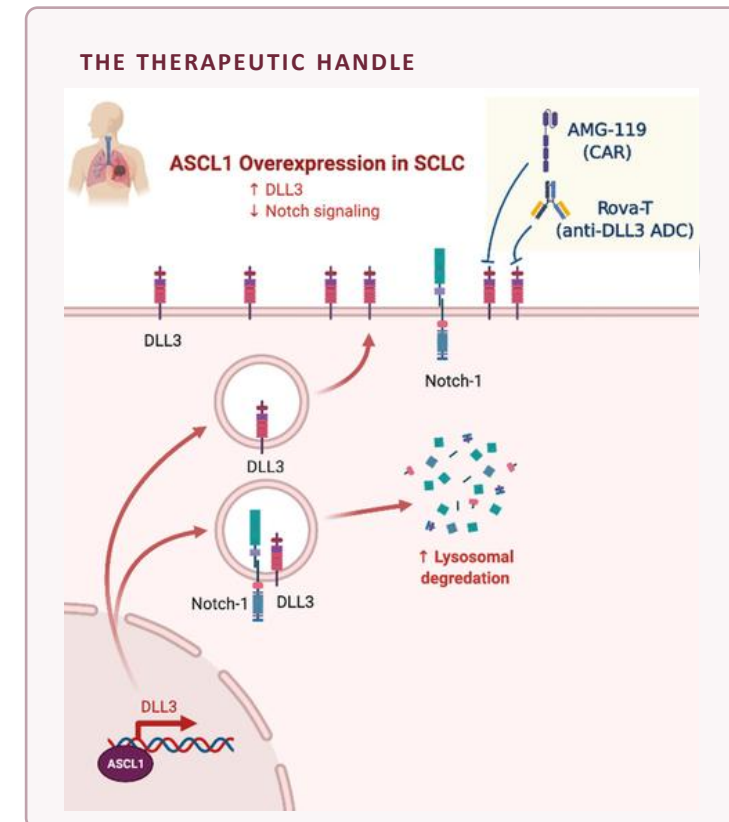
And medians can mislead

Median-based survival gains may overstate what most patients experience; the shape of the curve matters more than its midpoint.

A Protein in the Wrong Place



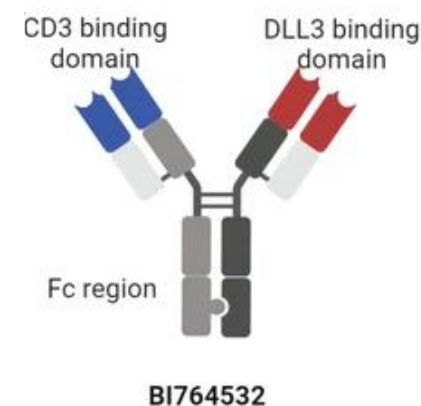
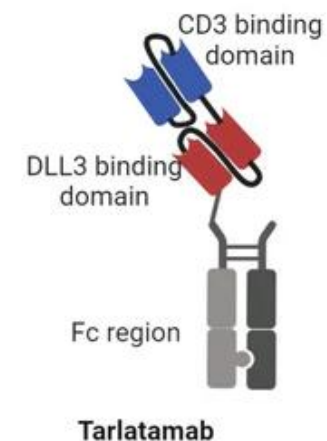
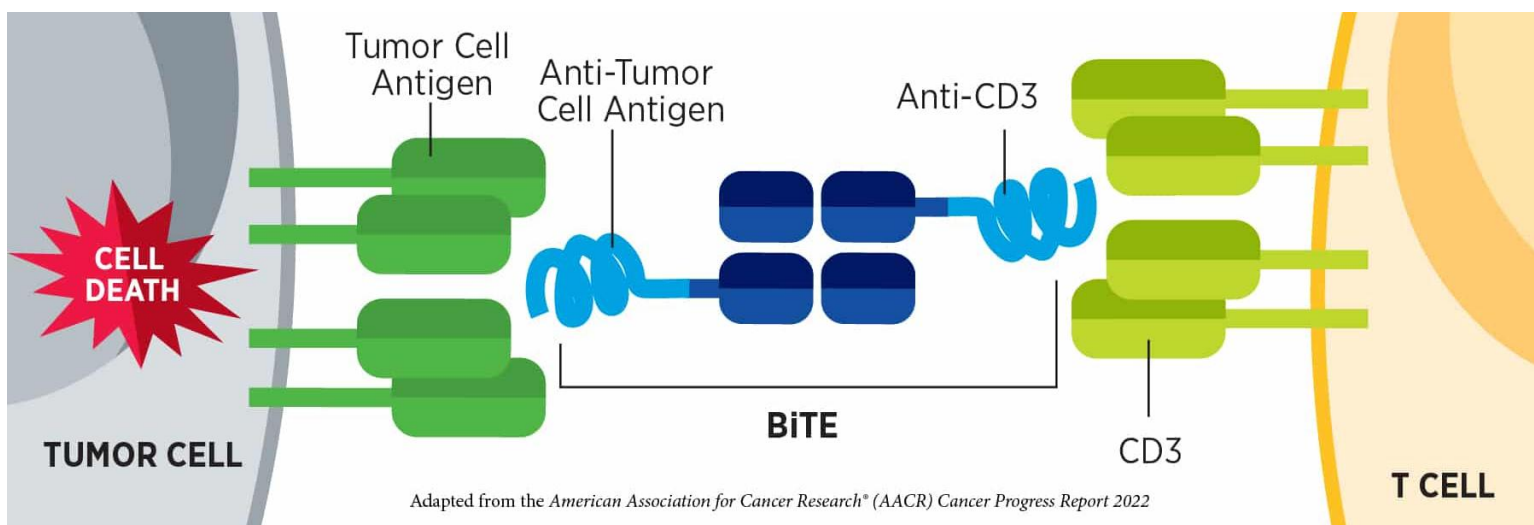
ASCL1 — the transcription factor that drives neuroendocrine identity — pushes DLL3 transcription up. DLL3 now appears on the cell surface, where it blocks NOTCH and locks the neuroendocrine phenotype in place.



The same event does both jobs. Mislocalised DLL3 is what sustains the neuroendocrine phenotype — and it is also what puts a target on the outside of the cell.

MECHANISM OF ACTION

A Bispecific Bridge in “Immune-Cold” SCLC



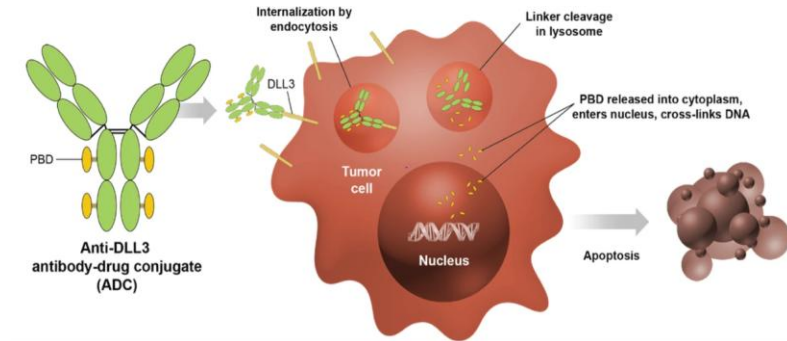
■ DLL3 is expressed on 85–96% of SCLC tumours, with minimal normal-tissue surface expression.

■ Engagement is independent of MHC class I — an advantage in tumours that downregulate antigen presentation.

■ Rova-T, an ADC against the same target, failed — the modality, not the target, was the problem.

Targeting NOTCH signaling pathway

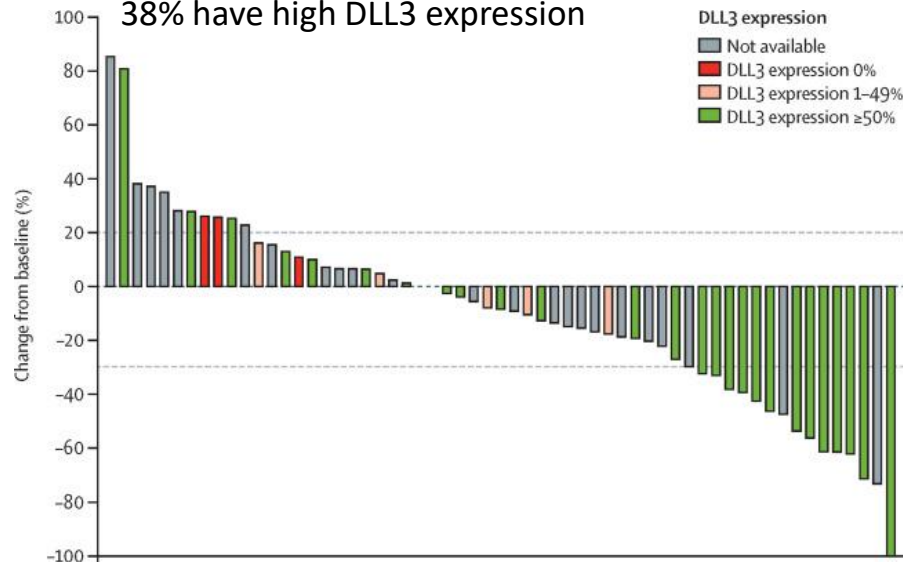
- Rovalpituzumab-tesirine (Rova-T)
 - Antibody-drug conjugate, DLL3-targeting
 - Anti-DLL3 moAb+ pyrrolobenzodiazepinePBD toxin



PhI trial. (N=74)

11/60 (18%) ORR

38% have high DLL3 expression



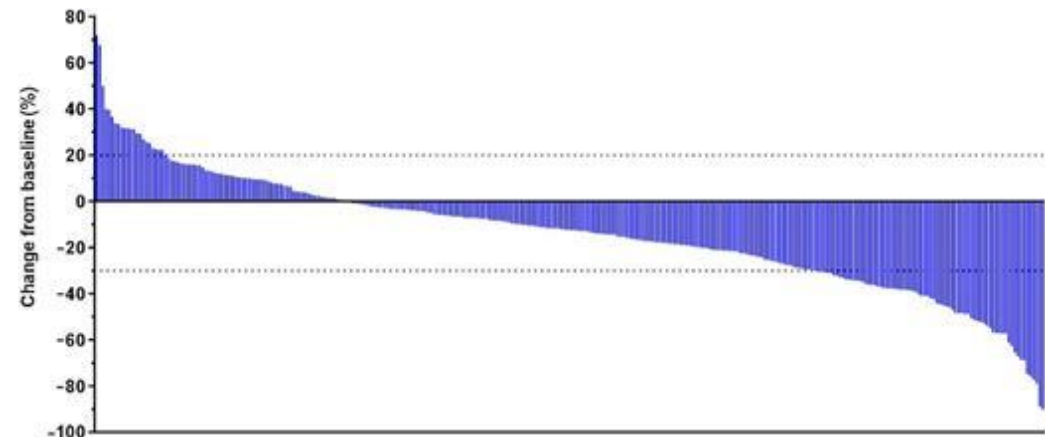
PhII TRINITY

In DLL+ patients, mostly in 3L : ORR 12%

In DLL3-high ($\geq 75\%$) ORR 14.3%, mPFS 3.8M, mOS 5.7M

Grade 3-5 213 (63%) patients

: fatigue, photosensitivity reaction, and pleural effusion



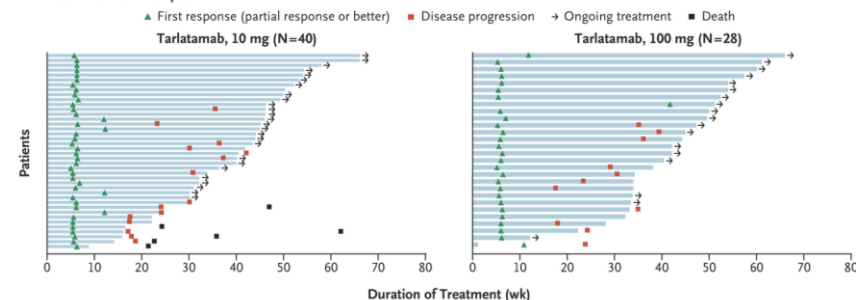
Tarlatamab — From DeLLphi-301 to DeLLphi-304

PHASE 2

DeLLphi-301

Activity in relapsed/refractory ES-SCLC

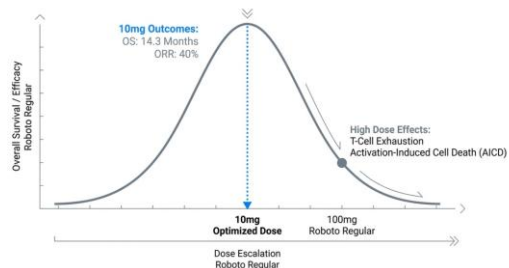
Onset and Duration of Response



PHASE 3

DeLLphi-304

Randomized vs investigator's-choice chemotherapy



MAY 2024

FDA accelerated

Approval on phase 2 data

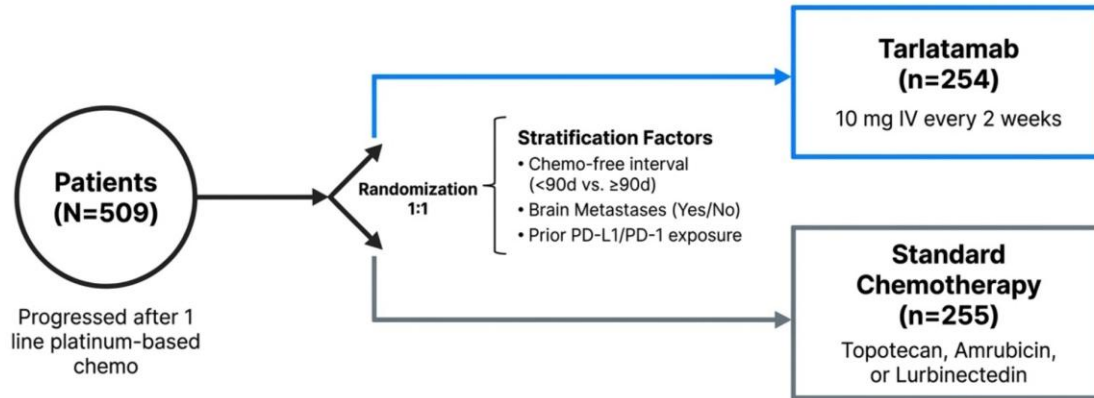
2025

Full approval

Confirmed OS → second-line standard

From first-in-human activity to a confirmed overall-survival benefit in roughly four years — unusually fast for this disease.

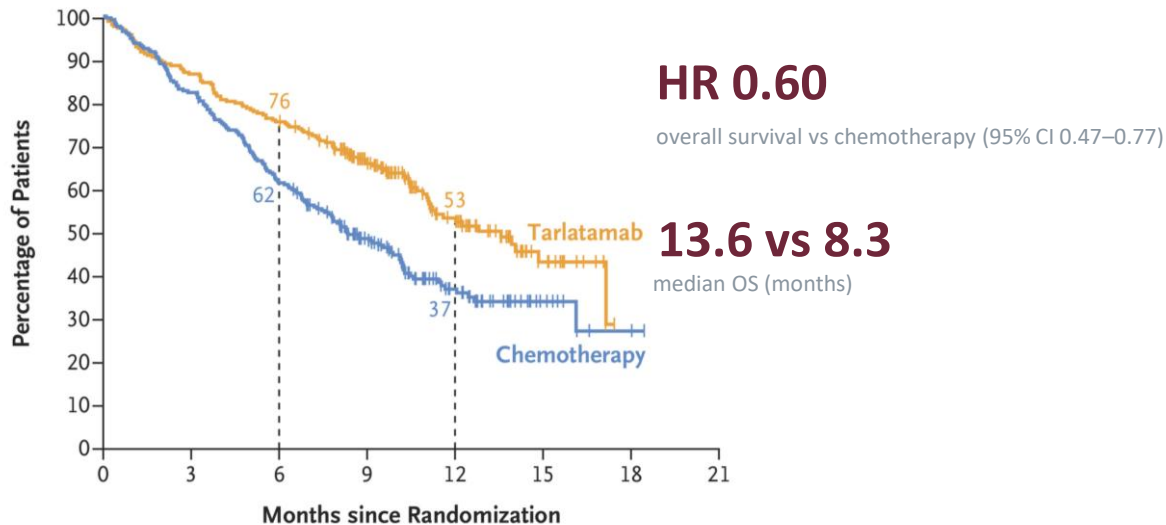
DeLLphi-304



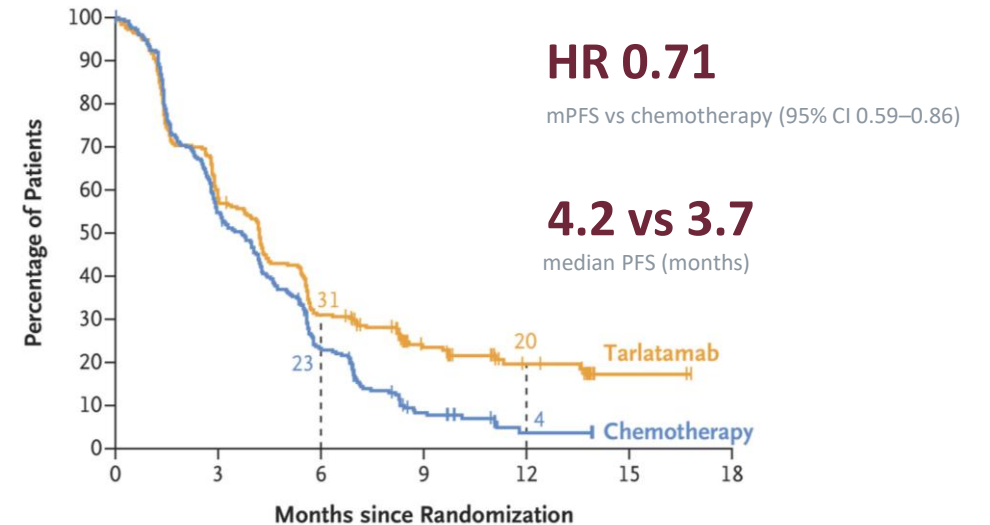
ENDPOINT & STRATIFICATION

Primary: overall survival. Stratified by prior PD-(L)1, chemo-free interval, and brain metastases. No crossover permitted.

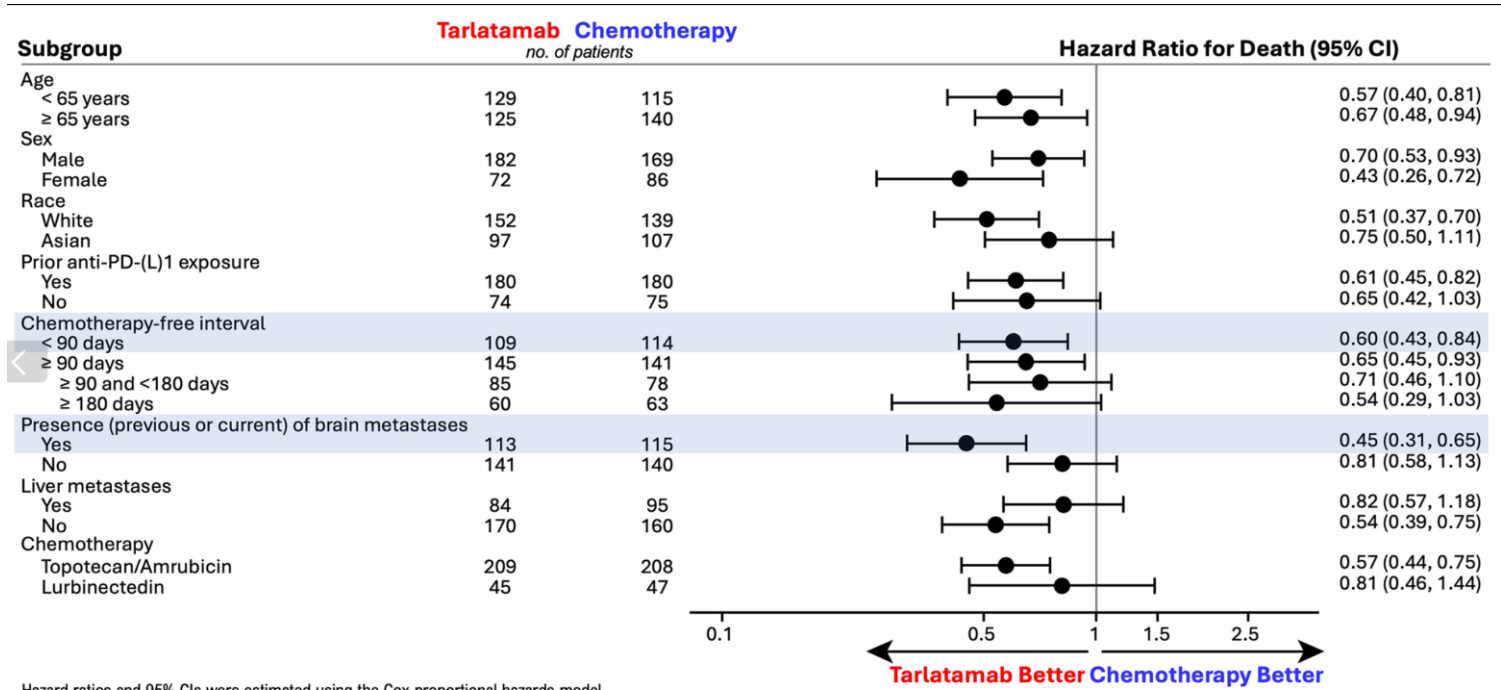
OVERALL SURVIVAL



PROGRESSION-FREE SURVIVAL



Consistent Benefit Across Subgroups



Hazard ratios and 95% CIs were estimated using the Cox proportional hazards model.

Direction never reverses

Every prespecified subgroup favours tarlatamab, including brain metastases and prior immunotherapy.

Better experience, too

Patient-reported dyspnea and cough improved versus chemotherapy.

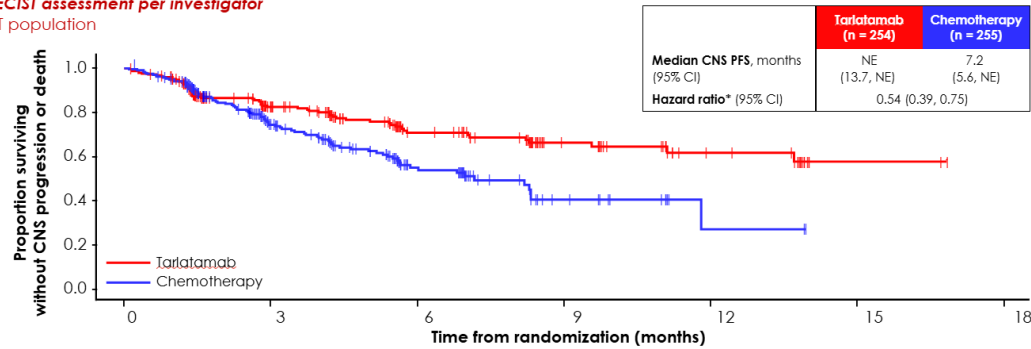
Safer profile

Fewer grade ≥3 treatment-related events and fewer dose interruptions than chemotherapy.

Brain Metastases — Where the Gap Is Widest

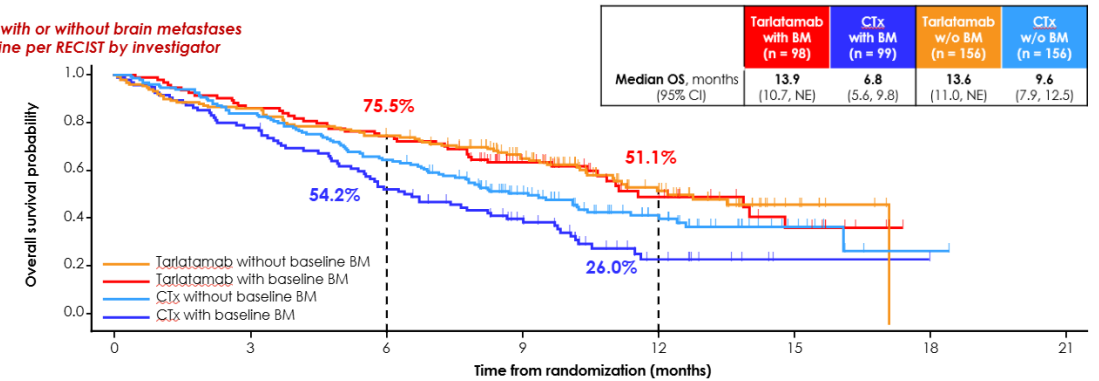
Tarlatamab delayed time to CNS progression or death in all patients

RECIST assessment per investigator
ITT population



Tarlatamab improved overall survival vs chemotherapy regardless of the presence of brain metastases

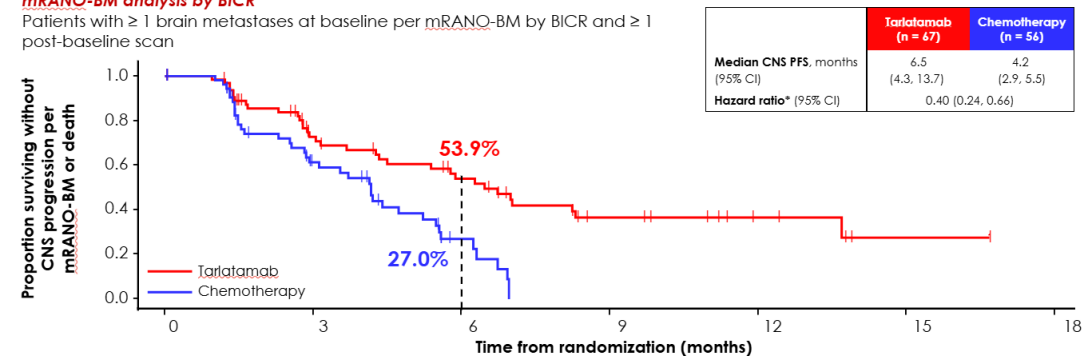
Patients with or without brain metastases at baseline per RECIST by investigator



- Tarlatamab performs the same either way. It is chemotherapy that collapses once the brain is involved — the widening gap comes from the comparator falling, not from tarlatamab rising.
- Post hoc and small. And over 70% had already had CNS-directed therapy — this is control of treated disease, not prevention

mRANO-BM analysis by BICR

Patients with ≥ 1 brain metastases at baseline per mRANO-BM by BICR and ≥ 1 post-baseline scan



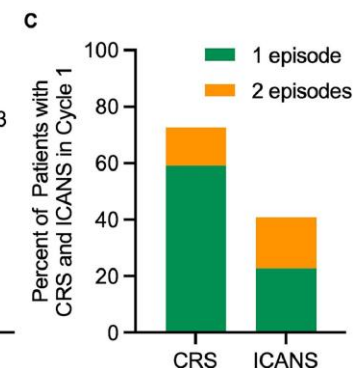
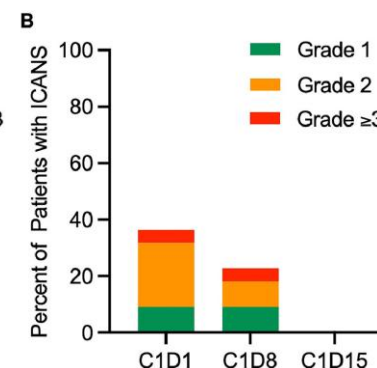
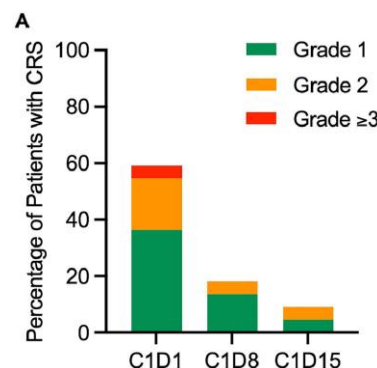
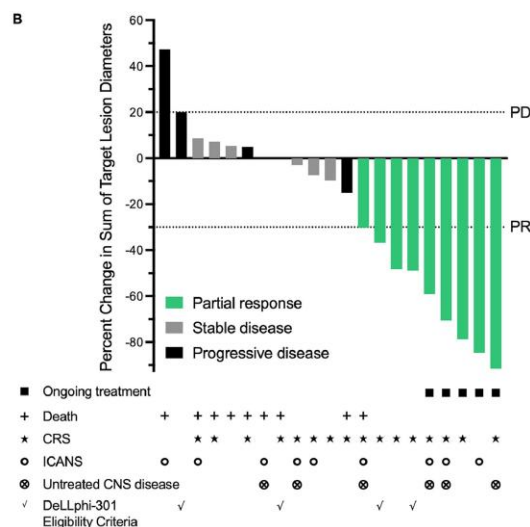
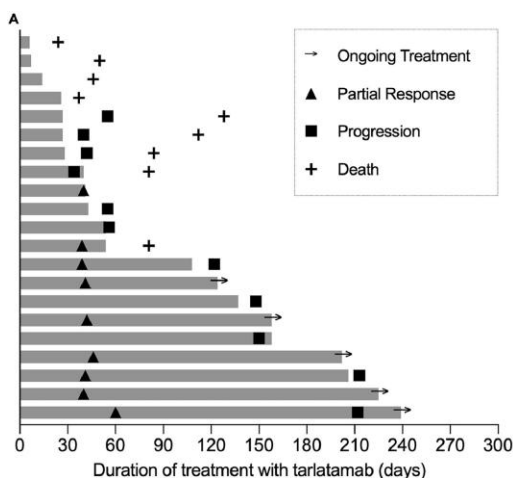
Outside the Trial — Efficacy Travels, Toxicity Does Not

Real-World Outcomes of Tarlatamab in Small Cell Lung Cancer, Including Patients With Untreated Brain Metastases

82%

of this cohort would not have met DeLLphi-301 eligibility criteria

41% had brain metastases, 64% liver metastases — the patients trials leave out.



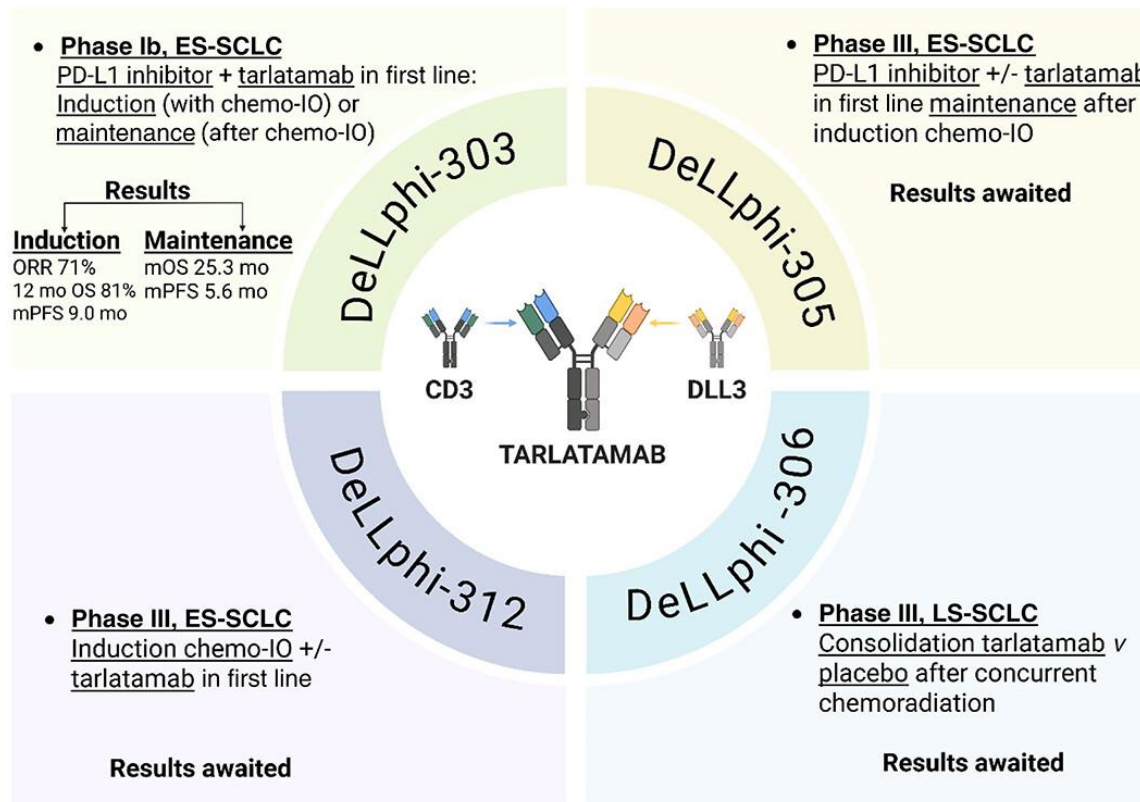
Both CRS and ICANS began within about 15 hours of the infusion — the risk is concentrated in the first day of cycle 1, which is exactly where the monitoring pathway has to be built.

Twenty-two patients at one centre — too small to set a rate. What it does establish is a direction: the same drug behaves differently in a sicker population.

Moving in Every Direction at Once

**DLL3
x
CD3**

One target,
one molecule —
now being tested
across the whole
disease course.



SECOND LINE

DeLLphi-304

settled

FIRST LINE · MAINTENANCE

DeLLphi-303 → 305

mOS 25.3 mo in phase 1b

FIRST LINE · INDUCTION

DeLLphi-312

Korea participating

LIMITED STAGE

DeLLphi-306

after chemoradiation

Second line is settled. The same molecule is now in first-line induction, first-line maintenance, and consolidation after chemoradiation — three read-outs still awaited.

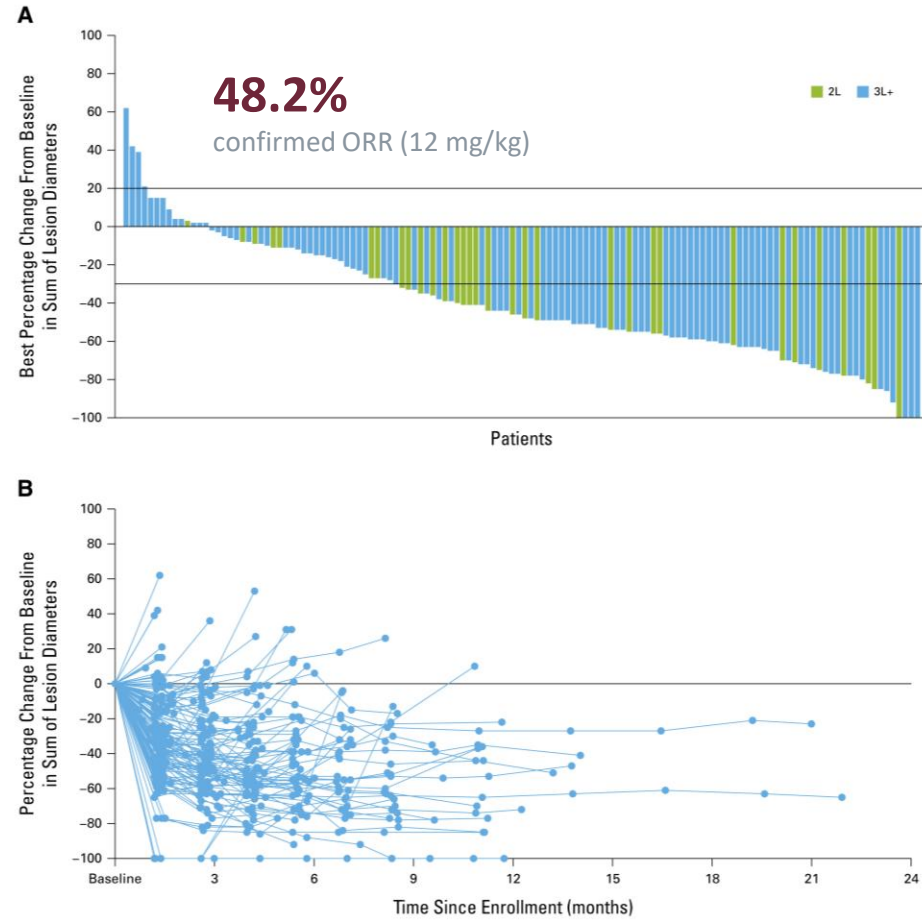
Four Targets, Four Modalities

	T-cell engager	Antibody–drug conjugate	Radiopharmaceutical	CAR-T
DLL3 85–96% of SCLC	Tarlatamab ✓ phase 3 Obixtamig · Gocatamig Alvetamig · Peluntamig	ZL-1310 · SHR-4849 (Rova-T failed)	¹⁷⁷ Lu-SC16 ²²⁵ Ac-ABD-147	AMG 119 (enrolment paused)
B7-H3 immune regulator	—	I-DXd — priority review HS-20093 · YL201 MHB088C · BNT324	—	—
SEZ6 neuronal antigen	—	ABBV-706 → phase 3	—	—
TROP-2 broadly epithelial	—	Sacituzumab govitecan (EVOKE-SCLC-04) Sac-TMT · SKB264	—	—

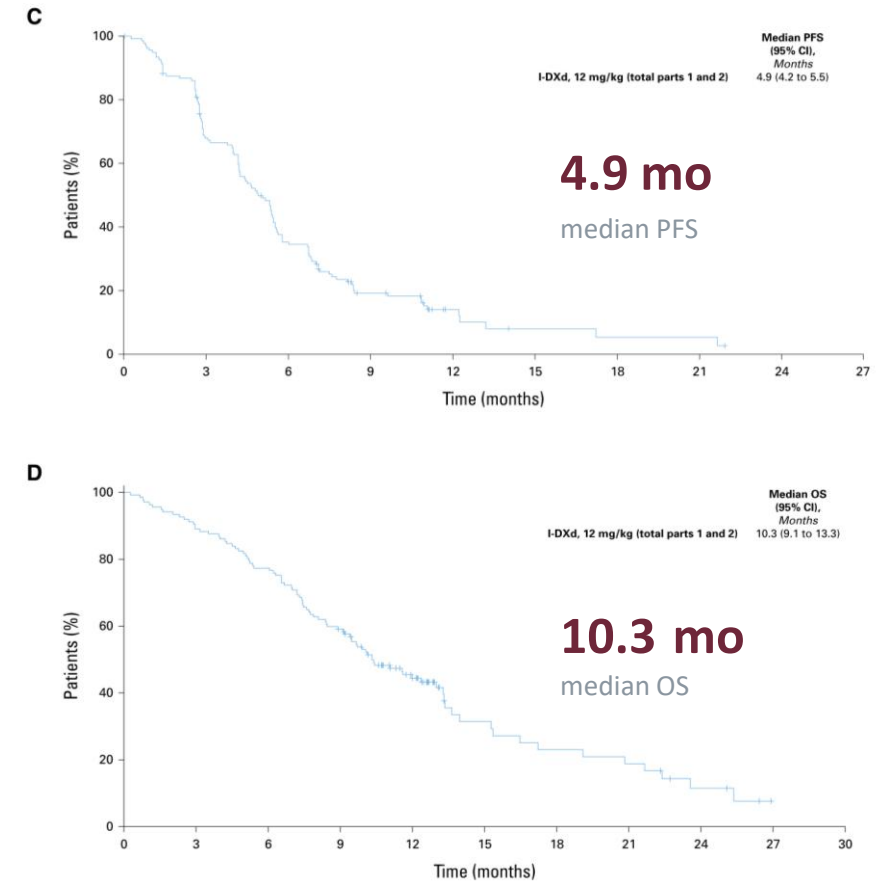
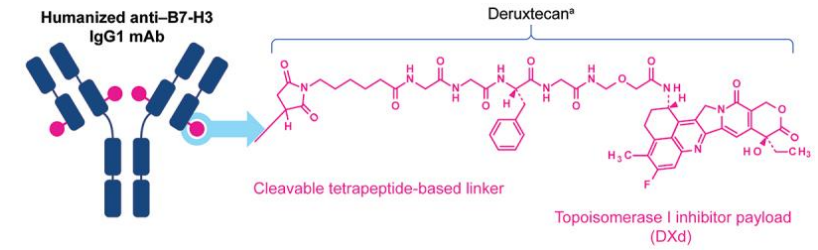
T-cell engagers act independently of MHC and stay potent at low antigen density, at the cost of CRS and ICANS. ADCs kill neighbouring antigen-negative cells, at the cost of payload toxicity — cytopenias and interstitial lung disease.

■ B7-H3 ANTIBODY-DRUG CONJUGATE

Ifinatamab Deruxtecan (I-DXd) — IDeate-Lung01



Representative best-response distribution (12 mg/kg cohort) — confirmed ORR 48.2%.

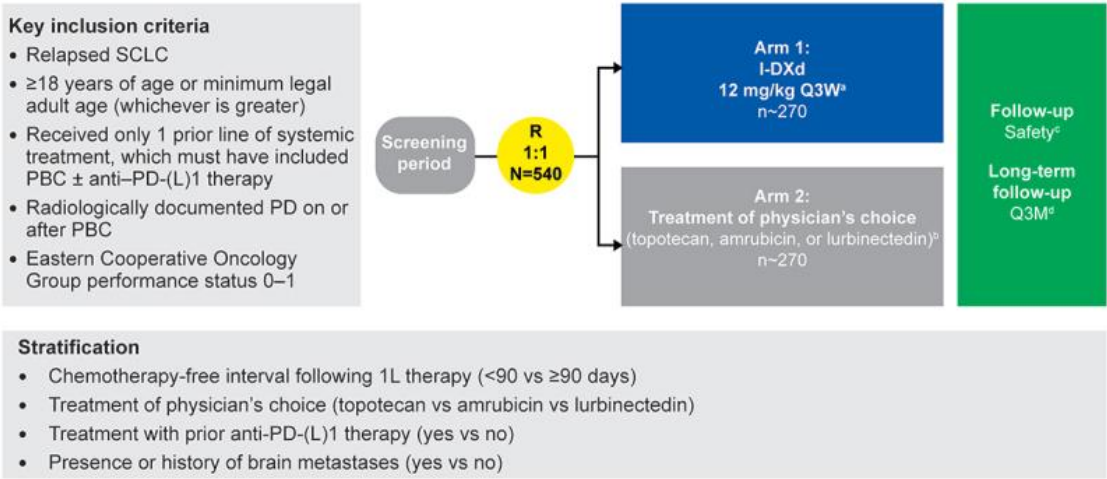


The B7-H3 Lung-Toxicity Signal

■ IDeate-Lung02

Phase 3 · I-DXd monotherapy

Placed on global hold in December 2025 after a higher-than-expected incidence of grade 5 interstitial lung disease.



■ DeLLphi-310

Phase 1b · tarlatamab + B7-H3 ADC

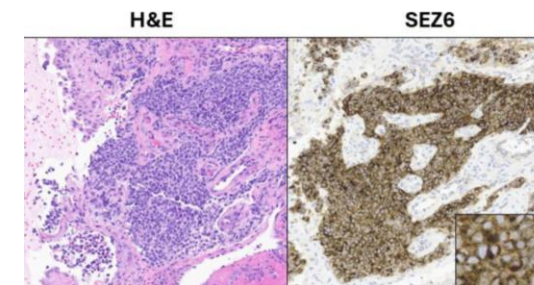
Halted following a fatal pneumonitis event while testing tarlatamab with tambotatug pelitecan.

Trial for SCLC ADC Paused After Patient Deaths FREE



December 19, 2025

ApexOnco and other media outlets reported that the FDA has placed a partial clinical hold on the phase III IDeate-Lung02 trial after more patients died from interstitial lung disease than anticipated; the number of deaths was not disclosed. The trial is evaluating Merck and Daiichi Sankyo's B7-H3-directed antibody–drug conjugate (ADC) ifinatamab deruxtecan (I-DXd) for patients with relapsed small cell lung cancer (SCLC). Previously enrolled patients will be able to continue treatment, but patient recruitment and enrollment will be halted as safety data are reviewed; the hold does not affect other studies of I-DXd. Daiichi Sankyo is working with the FDA to determine next steps.



ABBV-706 — Earlier Lines Do Better

Phase 1, 124 patients with relapsed/refractory SCLC. Recommended phase 2 dose confirmed at 1.8 mg/kg every 3 weeks — the lower of the two tested.

52%

objective response — all 124 patients

82%

in second line

n = 17 at the recommended dose

12.4 mo

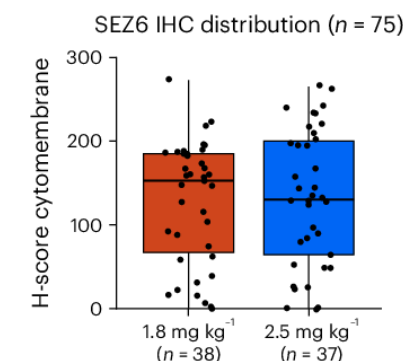
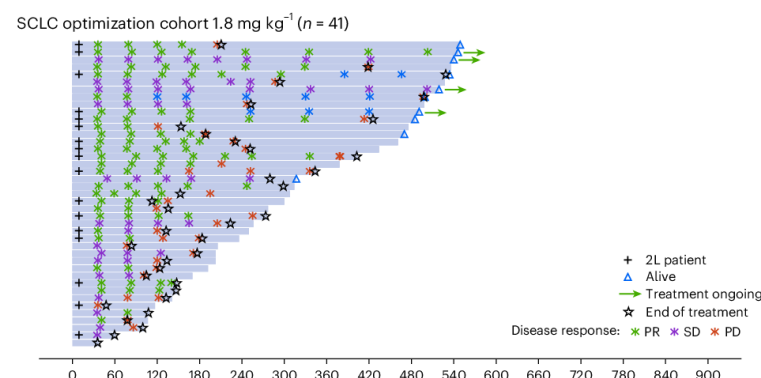
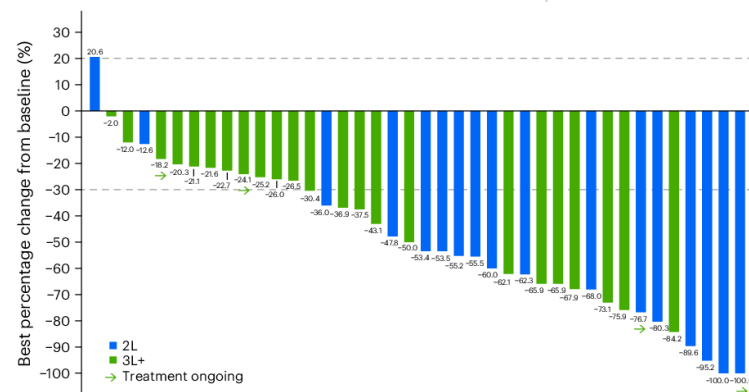
median OS at 1.8 mg/kg

14.3 months in the second-line subset

93%

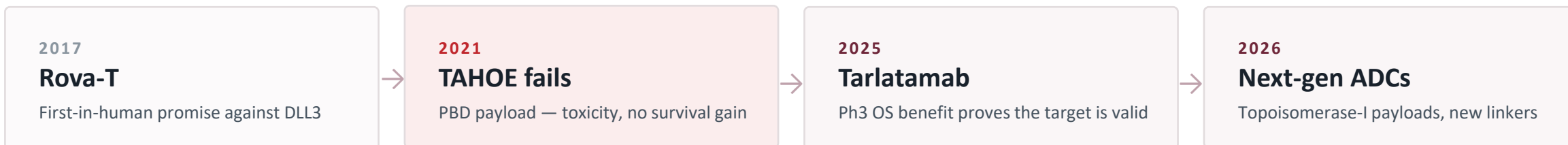
of tumours express SEZ6

median H-score 145



82% response with one prior line, 38% with two or more. Prior exposure to a topoisomerase-1 payload also halves it — 62% versus 42% — which is cross-resistance to the payload, not to the target. This is the argument for moving ABBV-706 into first line.

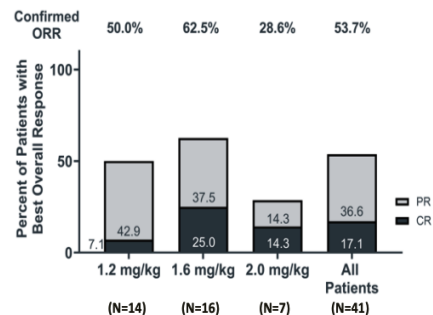
Return of the DLL3 Antibody–Drug Conjugate



ZL-1310 *zocilurtatug pelitecan*

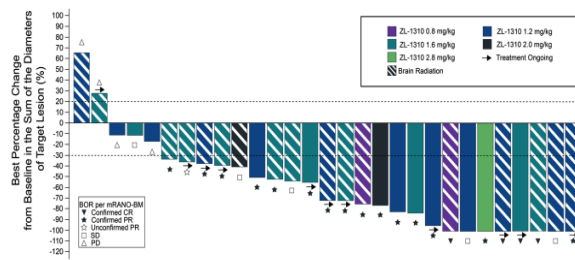
ZL-1310 demonstrated significant intracranial lesion regression as assessed by blind independent review

Best Intracranial Response by mRANO-BM (Confirmed Responses)^a



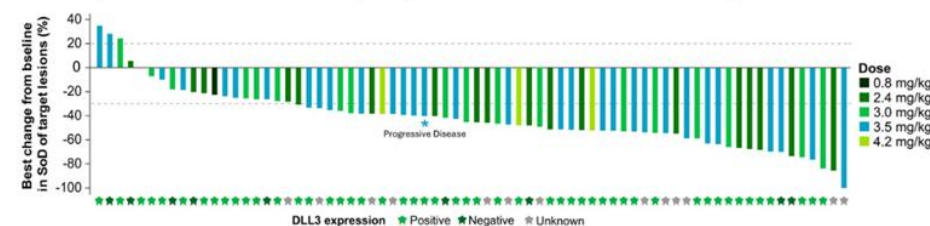
^a One additional patient response at the 1.6 mg/kg dose level is pending confirmation at the time of data cutoff.
ORR: objective response rate; DCR: disease control rate

Measurable Intracranial Tumor Reduction



SHR-4849 *IDE849*

IDE849 (SHR-4849) Demonstrated Compelling Initial Efficacy in SCLC
Robust responses observed across multiple expansion doses tested in Phase 1 study



	2.4 mg/kg		3.0 mg/kg		3.5 mg/kg		4.2 mg/kg		Total (≥2.4 mg/kg)	
	ZL Setting (n=10)	All (n=19)	ZL Setting (n=8)	All (n=18)	ZL Setting (n=16)	All (n=31)	ZL Setting (n=1)	All (n=3)	ZL Setting (n=35)	All (n=71)
ORR, n (%)	8 (80.0%)	14 (73.7%)	6 (75.0%)	12 (66.7%)	12 (75.0%)	23 (74.2%)	1 (100.0%)	3 (100.0%)	27 (77.1%)	52 (73.2%)
Confirmed ORR, n (%)	7 (70.0%)	11 (57.9%)	2 (25.0%)	4 (22.2%)	11 (68.8%)	16 (51.6%)	1 (100.0%)	3 (100.0%)	21 (60.0%)	34 (47.9%)
Response pending confirmation, n (%)	0	1 (5.3%)	4 (50.0%)	8 (44.4%)	0	1 (3.2%)	0	0	4 (11.4%)	10 (14.1%)
DCR, n (%)	10 (100.0%)	18 (94.7%)	8 (100.0%)	17 (94.4%)	15 (93.8%)	28 (90.3%)	1 (100.0%)	3 (100.0%)	34 (97.1%)	66 (93.0%)

The target was never the problem — the payload and the linker were.

Three Competing Backbones — the 2026–27 Read-Outs

Rebuild the chemotherapy backbone

- ROSETTA Lung-01 — pumitamidarone phase 3
- TAXIO / IFCT-2203 — etoposide-free
- SEZanne — ABBV-706 + atezolizumab

Add DLL3 to first line

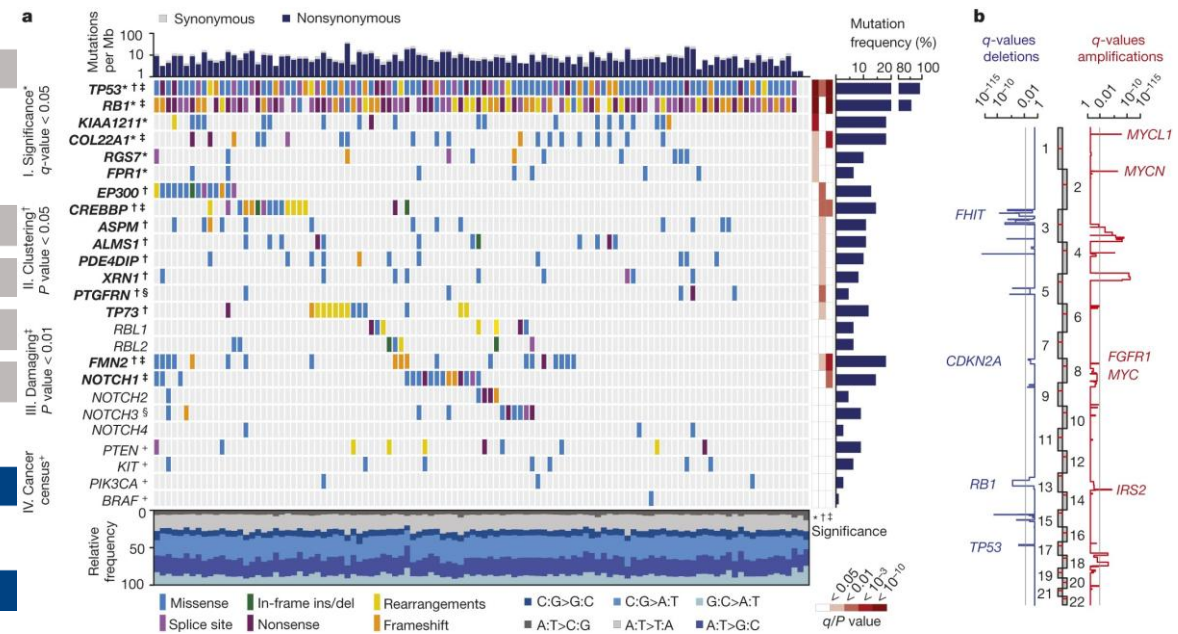
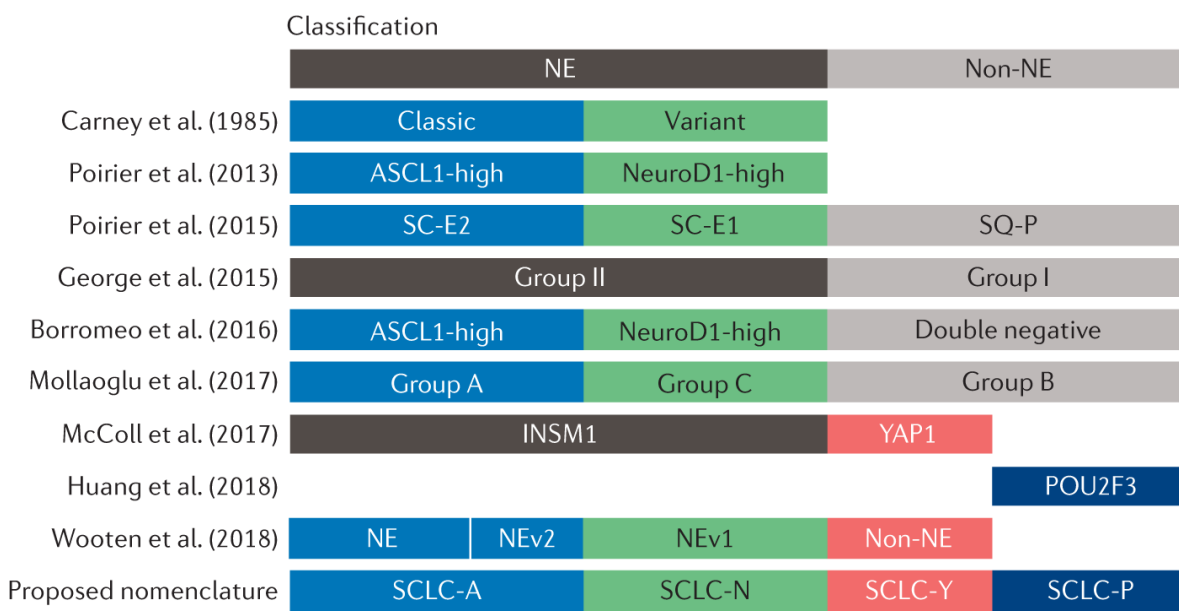
- DeLLphi-305 — maintenance, phase 3
- DeLLphi-312 — induction, phase 3
- Korea participating in DeLLphi-312

Push into curative-intent disease

- DeLLphi-306 — after chemoradiotherapy
- CONCUR — induction before cCRT
- MAVERICK — PCI versus MRI surveillance

If even two of these read out positive, first-line SCLC will have competing standards rather than a single backbone — and the sequencing question becomes the hard one.

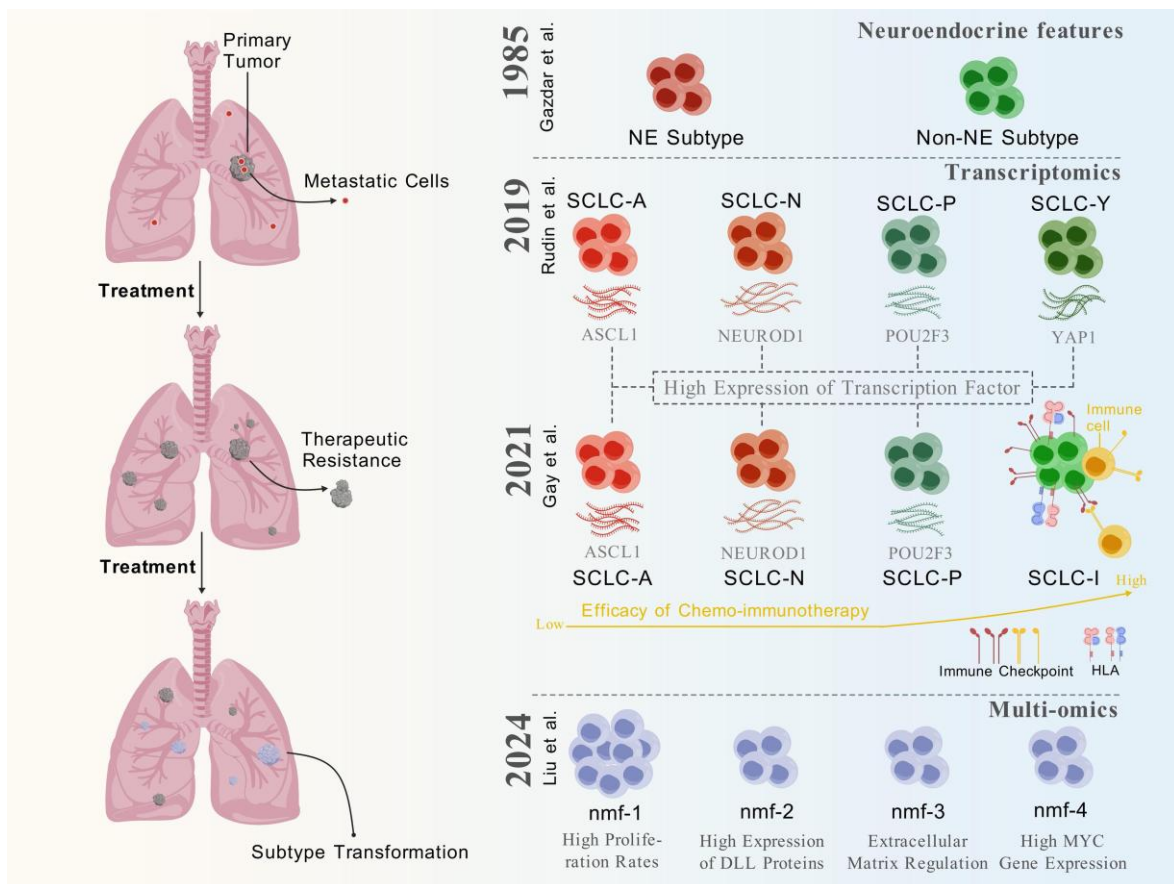
A Classification Still Being Rewritten



One subtype has already disappeared.

SCLC-Y was in the 2019 consensus and gone by 2021. And the four we use now are not fixed labels — under treatment pressure tumours move from A towards N and on to non-neuroendocrine states. This is the best working hypothesis available, not settled fact — which is worth remembering when the next slides use it to assign therapy.

Transcription-Factor Subtypes & Their Vulnerabilities



SCLC-A

ASCL1 · neuroendocrine-high

VULNERABILITIES

DLL3 · BCL2 · LSD1

SCLC-N

NEUROD1 · neuroendocrine-high

VULNERABILITIES

Aurora kinase · MYC

SCLC-P

POU2F3 · non-neuroendocrine

VULNERABILITIES

PARP7 · IGF1R

SCLC-I

inflamed · immune-enriched

VULNERABILITIES

Immune checkpoint blockade

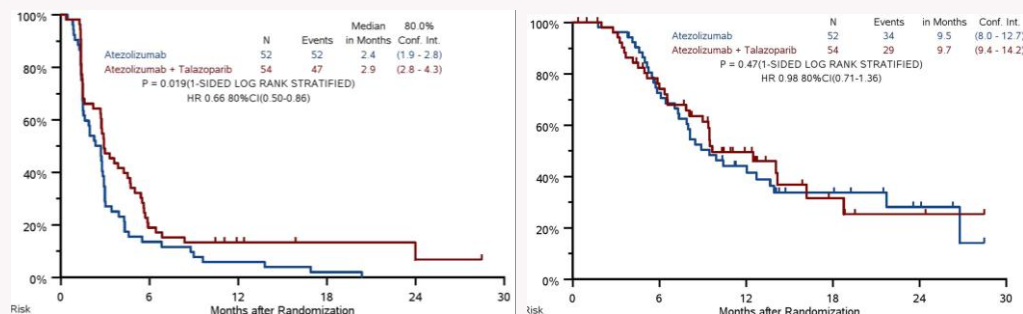
The clinical wall. IHC surrogates reproduce imperfectly, tumours are heterogeneous within themselves, and subtype shifts under treatment — A towards N, then to non-neuroendocrine states.

Subtype-Directed Therapy — Entering the Clinic

SLFN11 → PARP inhibition

SWOG S1929

Maintenance atezolizumab + talazoparib versus atezolizumab in SLFN11-positive ES-SCLC.



The first prospective biomarker-selected trial in SCLC — proof it can be done. The honest caveat: this PFS gain has not translated into a clear survival benefit.

PRISM — subtype-directed umbrella

Step 1: Screening

Step 2: Randomization

- ES-SCLC Screening
- Tissue available for testing
- Asymptomatic or Stable Treated Brain Lesions

Subtypes A/N, SLFN11 pos
or
Subtype P

Immunotherapy + PARP inhibitor
Immunotherapy

Subtypes A/N, SLFN11 neg

Immunotherapy + ATR inhibitor
Immunotherapy

Subtype I

Immunotherapy + NK cell activator
Immunotherapy

Primary Endpoints: PFS

Secondary Endpoints: OS, Frequency, Severity of Adverse Events

Subtype itself becomes the axis of treatment assignment. The remaining obstacle is real-time subtyping — turnaround time and tissue adequacy.

Redefining SCLC — Three Messages

1

No longer one disease, one regimen

Positive phase 3 data now span limited-stage, first-line maintenance and second-line treatment — the whole continuum has changed, though the survival tail remains thin.

2

DLL3 anchors a targeted era — with its first limits

Tarlatamab rewrote second line and is moving to first line, while B7-H3, SEZ6 and next-generation DLL3 agents advance — and combination toxicity has drawn the first boundary.

3

Selection by biology is becoming real

From mature subtype classification to the first biomarker-selected and umbrella trials — with real-time subtyping the remaining obstacle.

Thank you

Questions & Discussion