Oscotec R&D Day

October 24, 2023

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CEO

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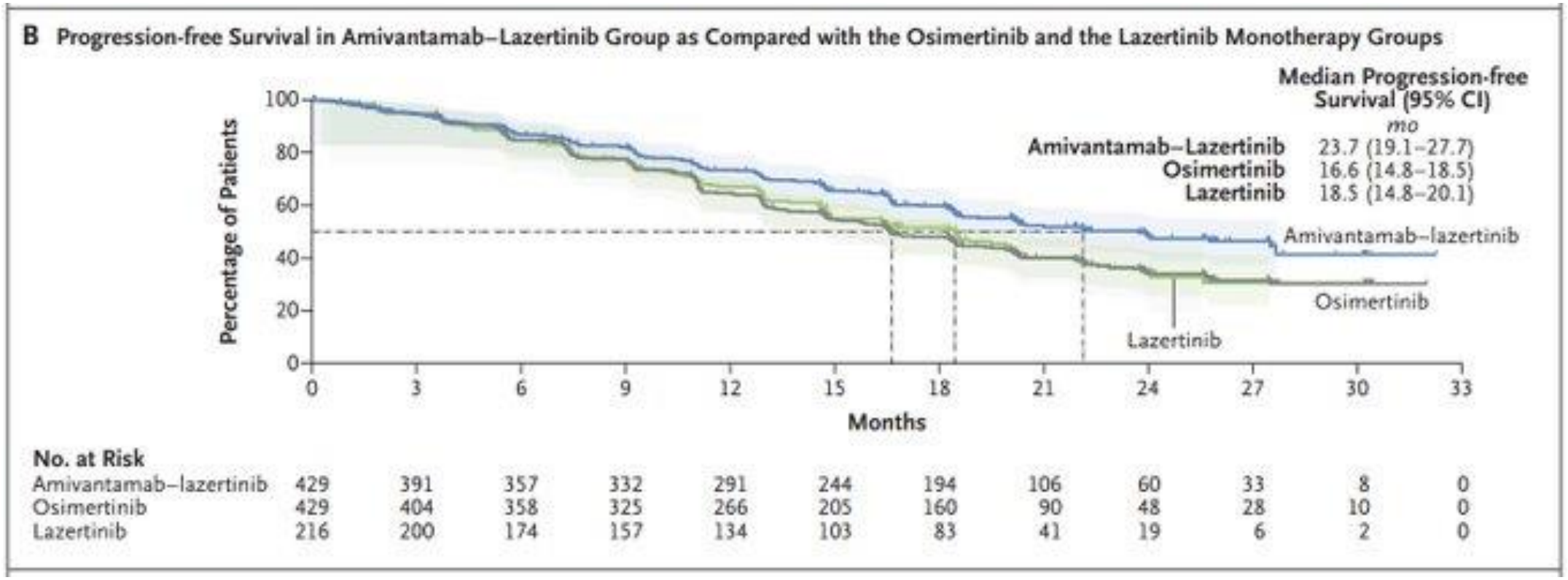
Agenda

- Clinical pipeline update
  - Lazertinib/amivantamab approved by FDA (1st line NSCLC)
  - Cevidoplenib BD ongoing; ITP 1st line study to start (IIT)
  - Denfivontinib (SKI-G-801) wrapped up Phase 1a in solid tumors
  - ADEL-Y01 initiated first-in-human dosing
- Oscotec oncology R&D strategy
  - Focus on cancer therapy resistance and relapse
  - Multiple internal programs gearing up for in vivo PoC
  - OCT-598 readies IND
- Q&A

Clinical Pipeline Update

Lazertinib

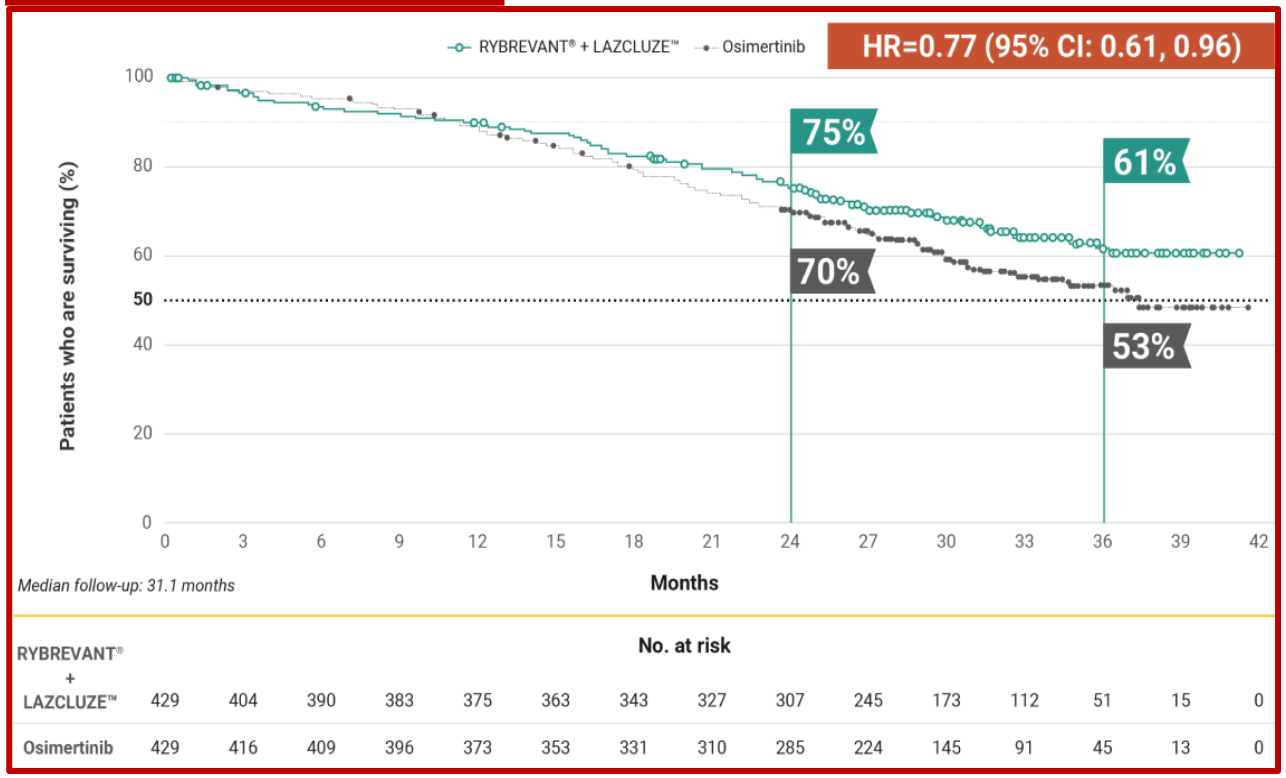
- Lazertinib-amivantamab combination approved
  - For the first line treatment of patients with locally advanced or metastatic NSCLC with EGFR exon 19 deletion or L858R mutations
  - Median PFS 23.7 months vs 16.6 with osimertinib (p = 0.0002, HR 0.70)



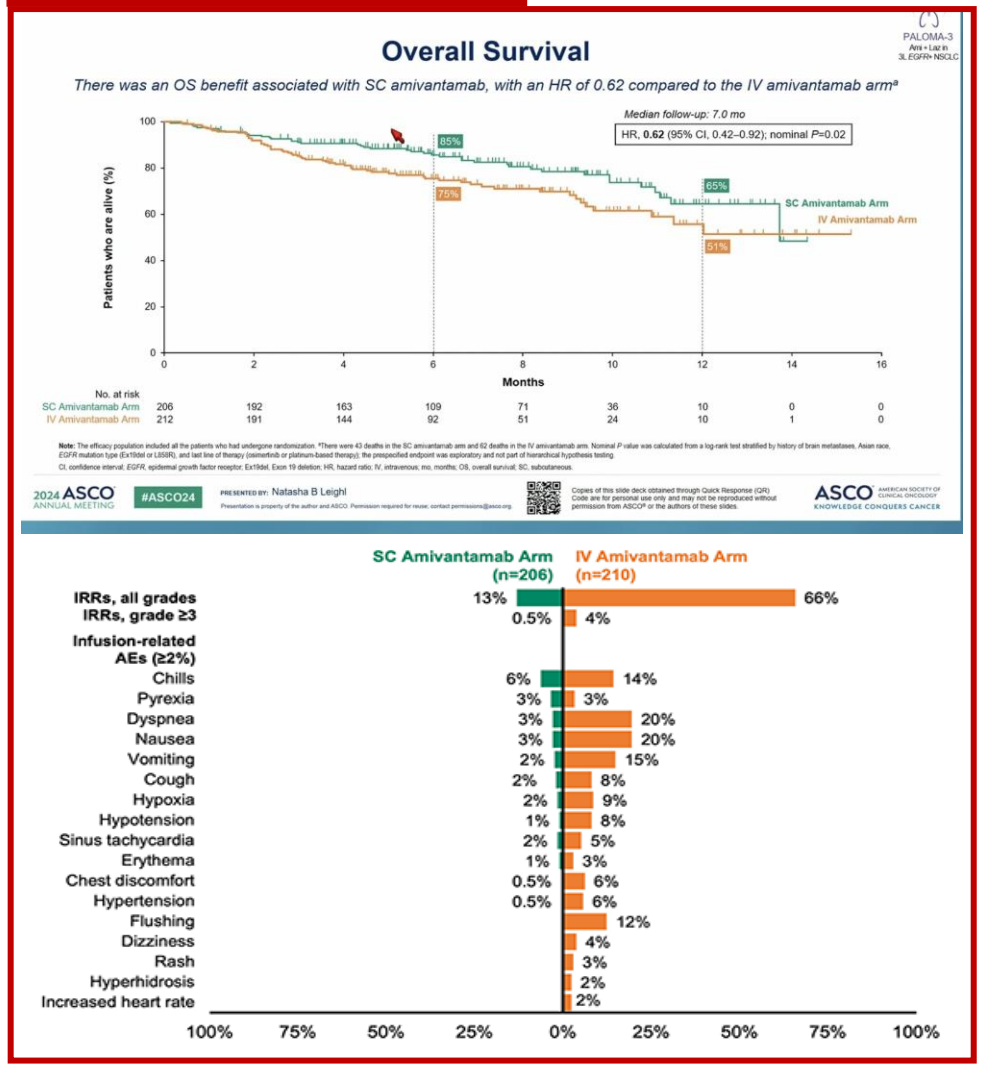
Lazertinib

- Lazertinib-amivantamab combination
  - Overall survival still immature
  - Subcutaneous injection superior to IV

1st line EGFR*mut* NSCLC

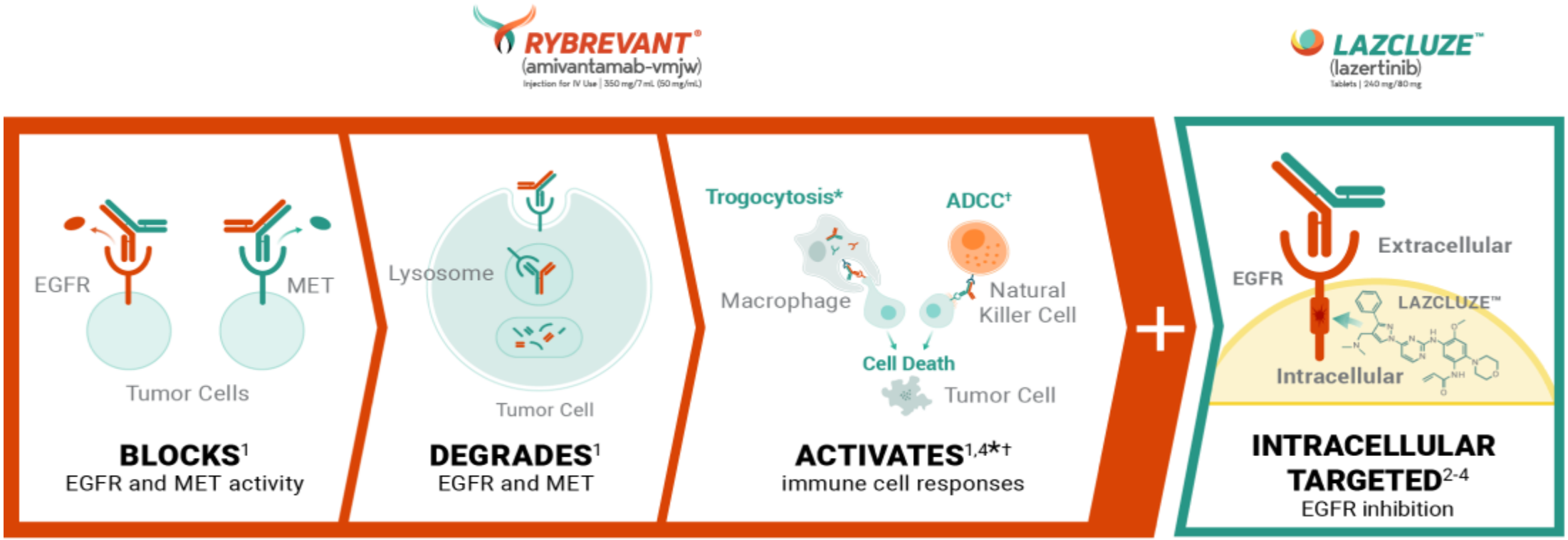


2nd line EGFR*mut* NSCLC

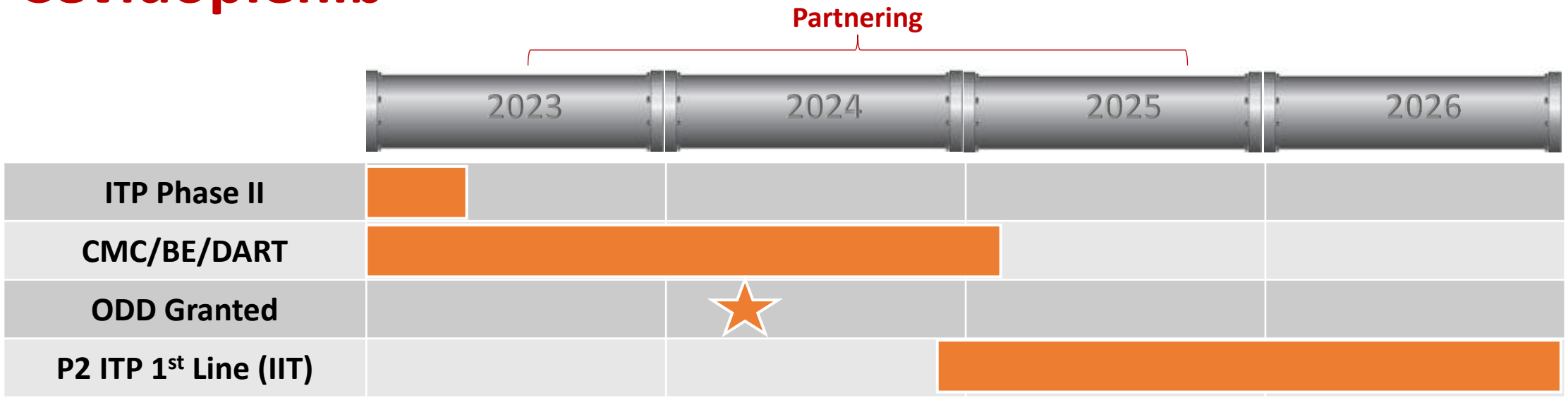


Lazertinib

- Mechanism of action alleged to involve enhanced cell killing
- However, ORR is similar to that of osimertinib monotherapy (86 vs 85%)
- PFS is dramatically improved (23.7 vs 16.6 months)
- C-MET is implicated in development of **therapy-resistance**



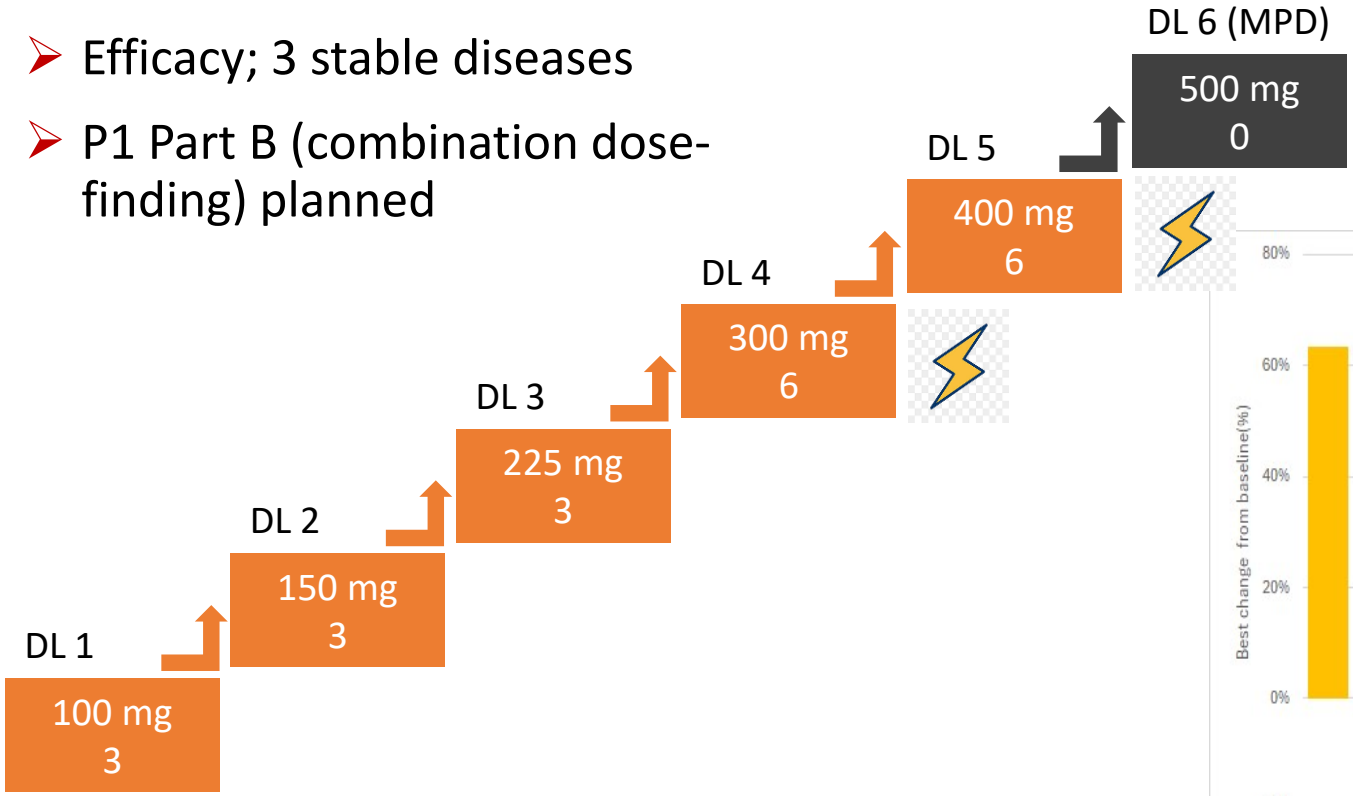
Cevidoplenib

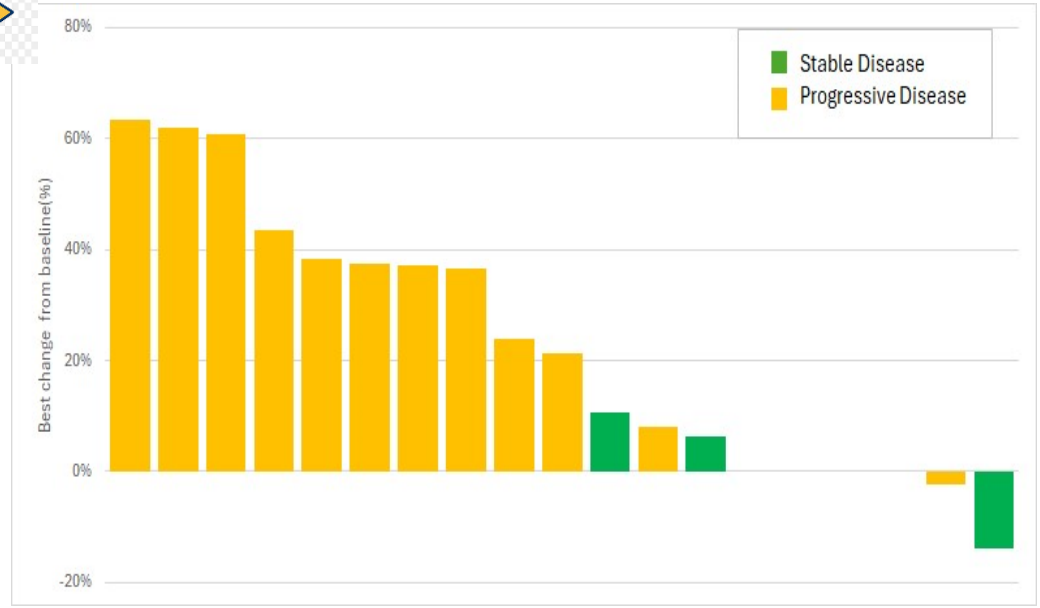


- Orphan drug designation granted by FDA (priority review, market exclusivity)
- CMC and nonclinical readiness for further development, incl new patents
- Partnering activities ongoing
- Investigator-initiated trial for ITP (St. Mary's Hospitals)
  - Frontline treatment in conjunction with the SoC (steroid or IVIG)
  - Aims to prevent progressing to chronic ITP, potentially "cure"
  - IND to be filed (MFDS) in Dec 2024

Denfivontinib (SKI-G-801)

- Completed P1 Part A (monotherapy dose escalation)
- 21 patients dosed (10 NSCLC, 5 breast cancer, etc)
- Safety; well-tolerated; 2 DLTs, MTD not established
- PK; nonlinear dose-exposure relationship
- Efficacy; 3 stable diseases
- P1 Part B (combination dose-finding) planned



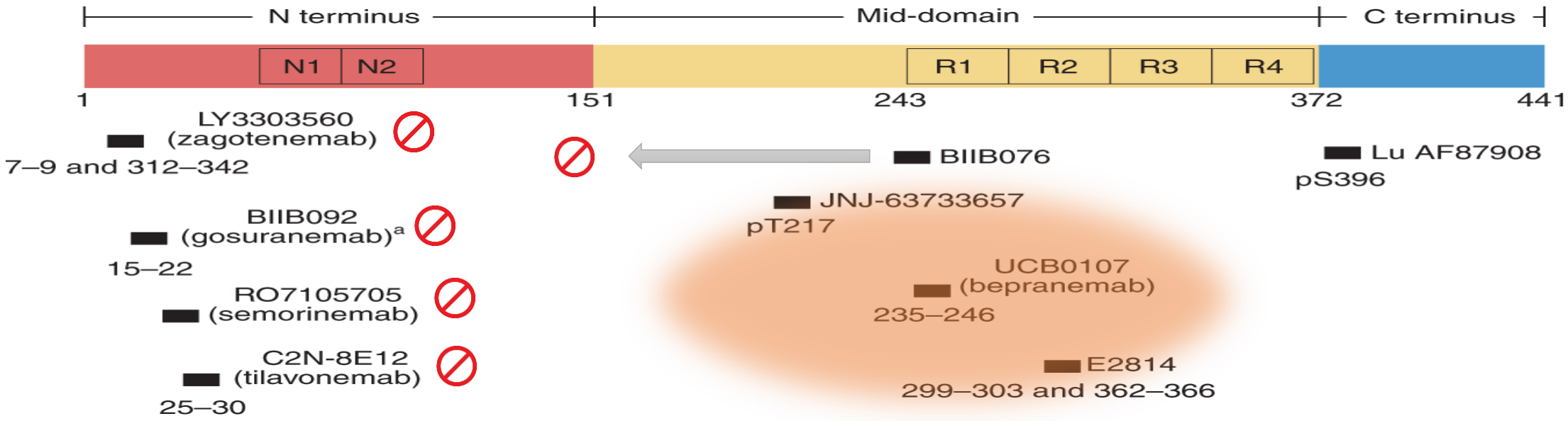


ADEL-Y01

- ADEL-Y01 is a monoclonal antibody targeting a pathological form of tau protein (AcK280) to treat tauopathies including Alzheimer Disease
- First-in-human study underway (US)
  - First in Human, Phase Ia/b study for safety, tolerability, pharmacokinetics, and clinical activity evaluation of ADEL-Y01 in healthy participants and in participants with Mild Cognitive Impairment due to Alzheimer's disease or mild Alzheimer's disease
  - Successfully completed SAD cohort 1 & 2 (dose levels 2.5 & 7.5 mg/kg)
  - No safety concern reported to date
  - PK analysis in progress
  - Gearing up for Part II MAD study

Phase 1		2024				2025				2026			
		Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4	Q1	Q2	Q3	Q4
Part I (SAD)	Healthy volunteers (n = 40)												
Part II (MAD)	MCI from AD or mild AD (n = 33)												
	Open label extension												

ADEL-Y01



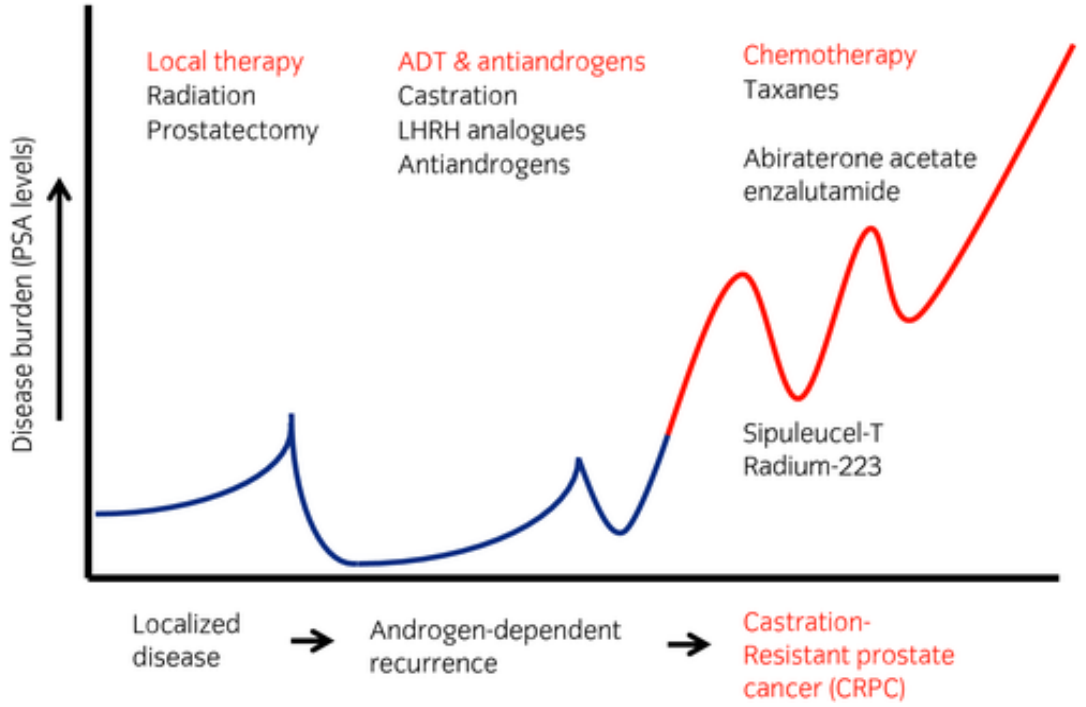
Drug	Company	Start	Primary	Completion	N	Dosing	Phase	Epitope
Bepranemab	Roche	Jun 2021	May 2024	Jul 2025	466	80 + 48 wks	P2	235-246
Posdinemab JNJ-63733657	JNJ	Jan 2021	Feb 2026	Dec 2032	523	104 wks + LTE	P2	pT217 (MTBR)
E2814	Eisai	Jun 2021	May 2024	May 2024	8	12 + 96	P1b/2, DIAD (OL)	299-303/362-366
PRX005 BMS-986446	BMS	Oct 2023	Mar 2024	Mar 2024	24		P1, Japanese only	
MK-2214	Merck	Sep 2022	Sep 2025	Sep 2025	48		P1 (HV)	pS413
Lu AF87908	Lundbeck	Sep 2019	Jul 2023	Jul 2023	86		P1 (HV + AD) SAD	386-408

Preclinical/Discovery Pipeline

Problem; Tumor Relapse

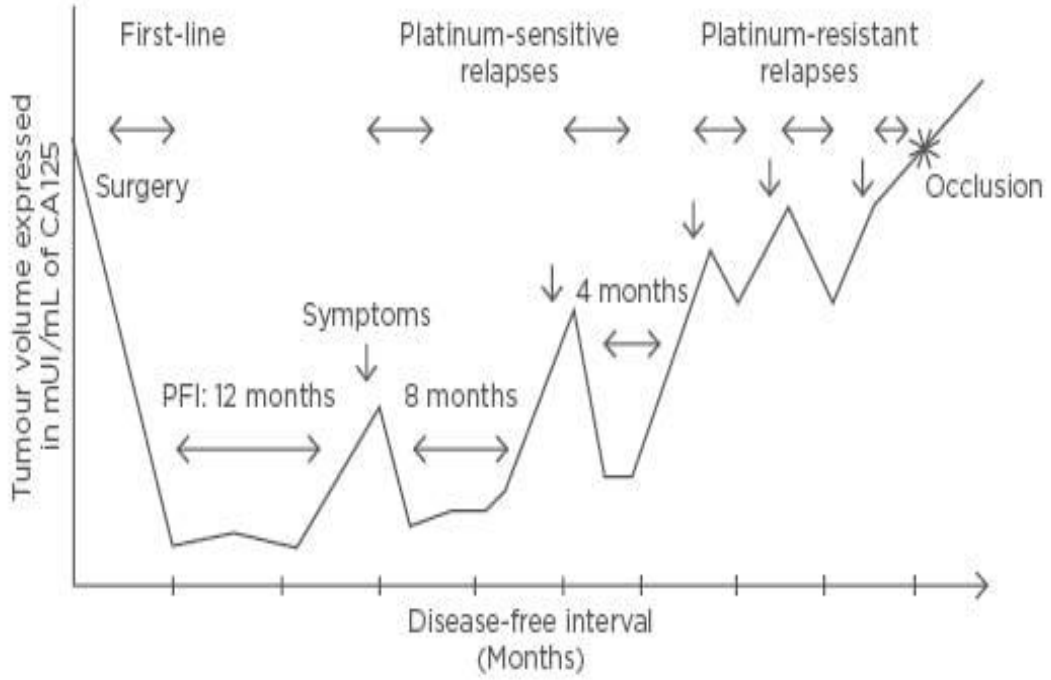
Current antitumor therapeutic paradigm is based on tumor-selective maximal cell killing. However, most tumors relapse after treatment and relapsed tumors are more malignant and lethal

Prostate Cancer



Imamura & Sadar, Int. J. Urology 2016

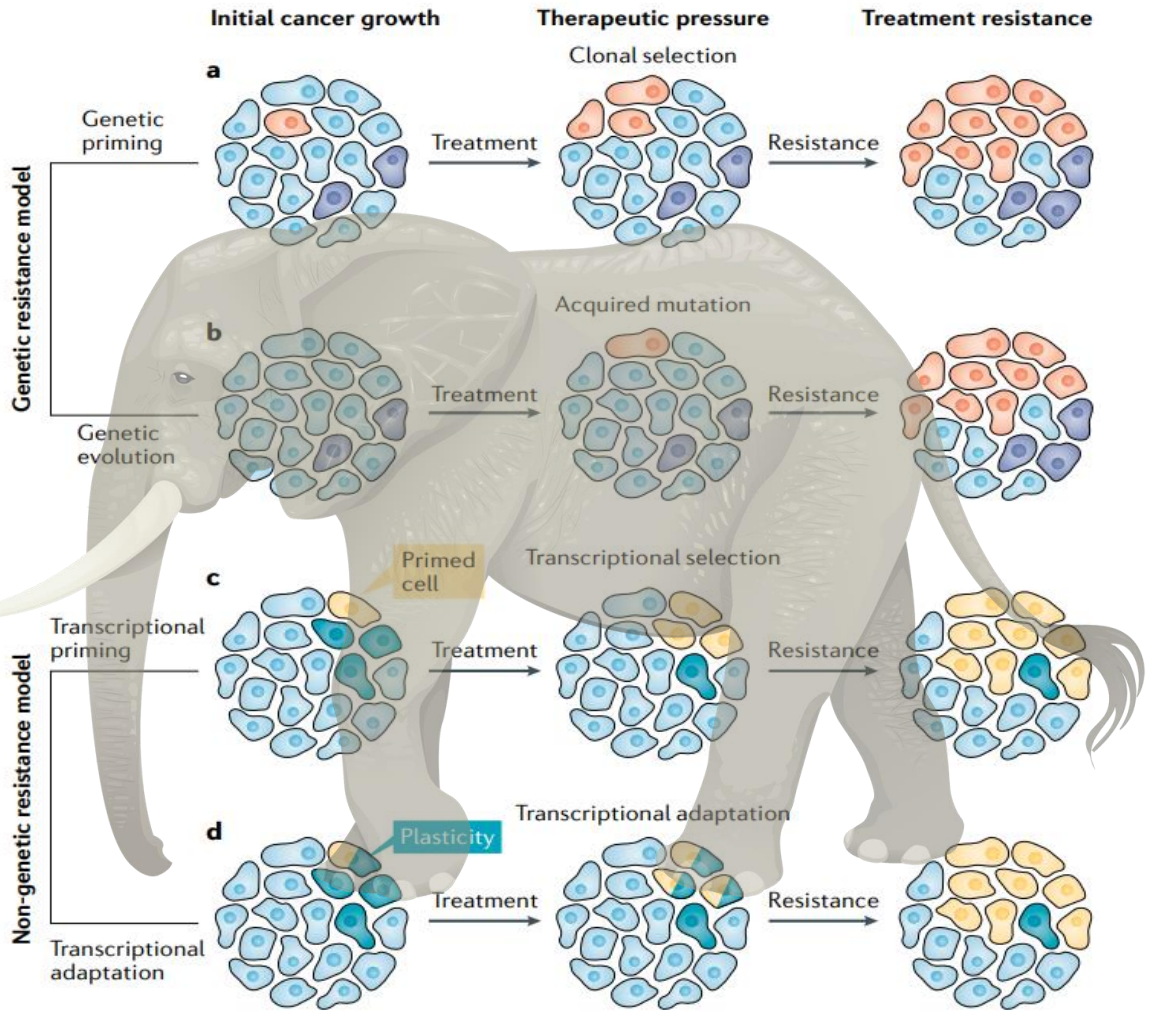
Ovarian Cancer



Giornelli & Mando, Eur Med J 2017

Rethinking Cancer Therapy Resistance

- Common features of acquired resistance
  - Intratumor heterogeneity
  - Chromosome instability (CIN), aneuploidy
  - De-differentiation, stemness
  - Immune suppression in TME
- Various genetic and non-genetic mechanisms of therapy resistance have been proposed
  - Clonal selection of intrinsic resistance
  - Acquired mutations
  - Drug tolerant phenotype (DTP)
  - Transcriptional plasticity (e.g., EMT)
  - Cancer stem cell



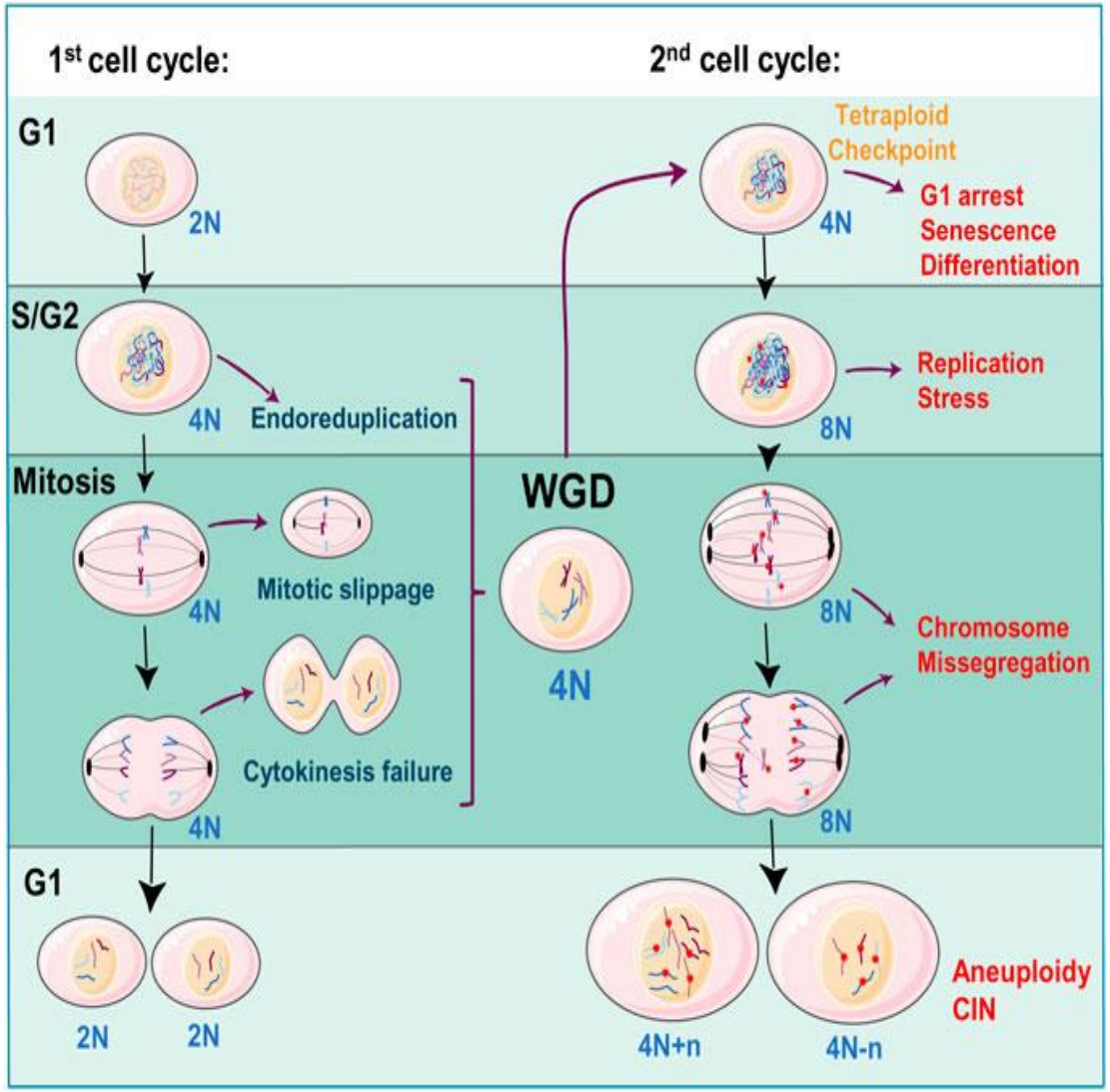
Marine et al., Nat Rev Cancer 2020

Is there a unified underlying process?

Whole Genome Doubling (WGD)

- An ancient mechanism of stress-adaptation and evolution
  - Unicellular organisms (e.g., yeast) under stressful environmental conditions
  - A driving force of species evolution (e.g., plants) leading to genetic and biological complexity
  - Physiological polyploidy in specialized cell types (hepatocytes, cardiomyocytes, megakaryocytes, and trophoblasts) – stress response, wound healing and tissue regeneration

Polyploidy via WGD fosters aneuploidy and thereby intratumor heterogeneity

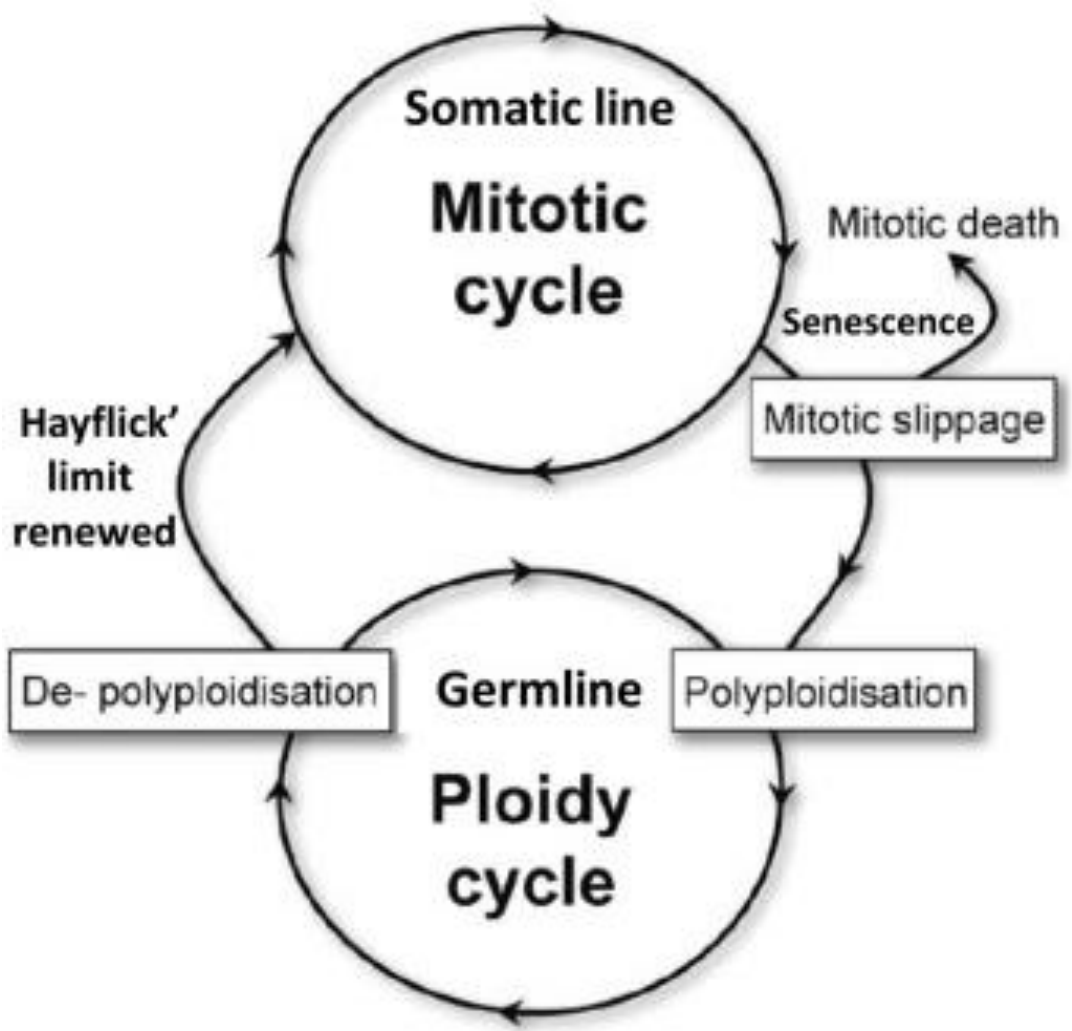


Sanz-Gomez et al., Front Cell Dev Biol 2023

Ploidy Cycle Confers Stress-Adaptability and Evolvability

- Polyploid cells are **stress-resistant**
  - Large size, protective quiescence
  - Increased genomic content buffers the negative impacts of accumulation of deleterious mutations
  - Mitigating Muller's ratchet
- **Accelerated evolution**
  - Reductive division (de-polyploidization)
  - Meiotic recombination, directed evolution
  - Generates para-diploid progeny cells with **stem cell** characteristics

Targeting ploidy cycle may impede the development of therapy resistance



Erenpreisa et al., Seminars in Cancer Biol 2020

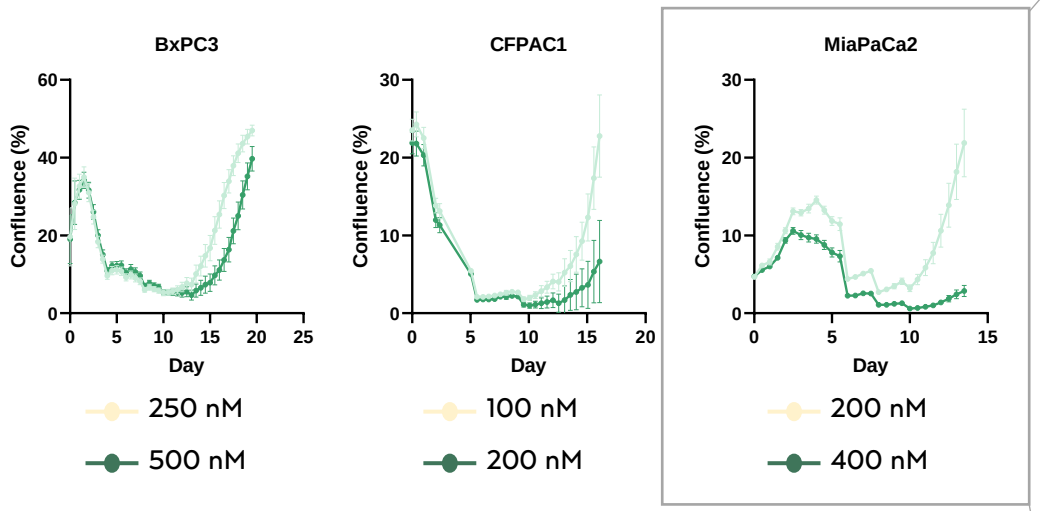
Focus on Cancer Therapy Resistance

- Suppressing "ploidy-mediated induction of cancer therapy resistance"
- Multiple novel targets implicated in various stages of the ploidy cycle
- To be developed as an adjuvant to established standard-of-care anticancer treatment
- Anticipated to prolong progression-free survival and possibly **'cure cancer'**

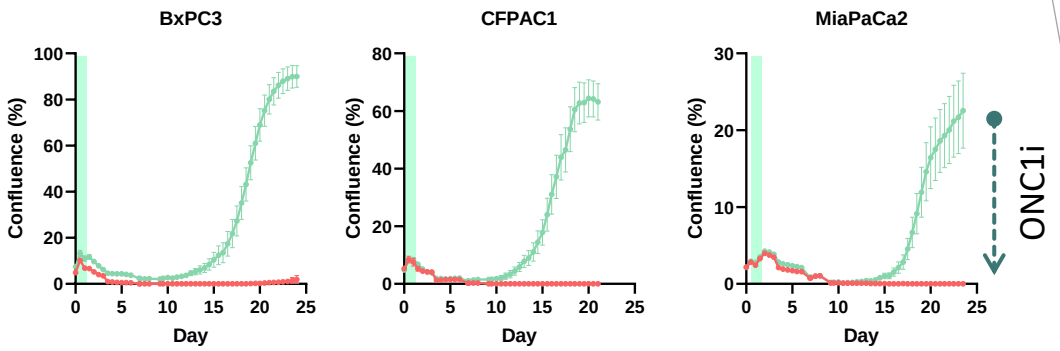
Program	Target	In-cell PoC	In vivo PoC	Candidate	Clinical dev
OCT598	**EP2/4**				
ONC1	(undisclosed)				
ONC2	(undisclosed)				
ONC3	(undisclosed)				
ONC4	(undisclosed)				

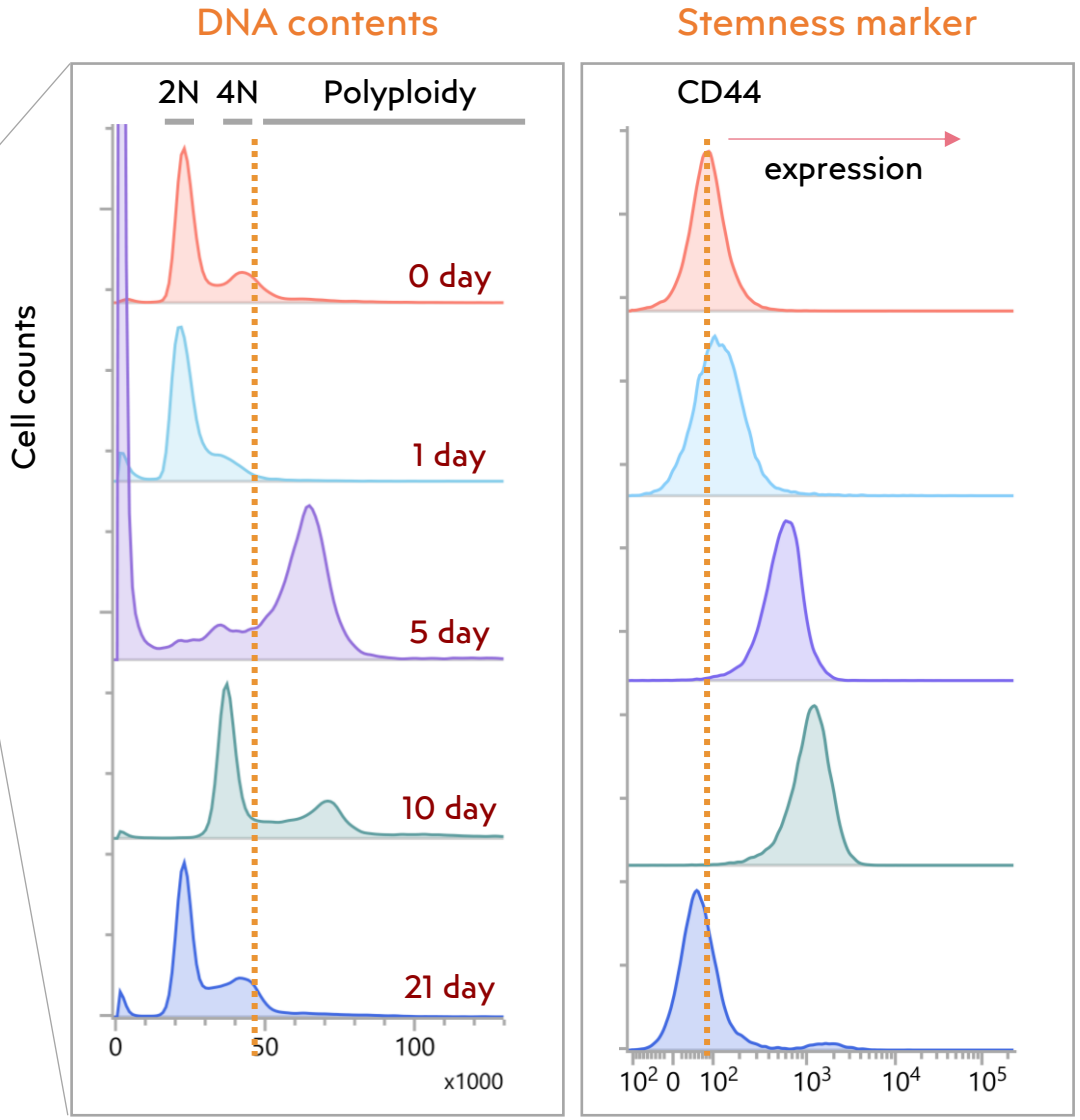
ONC1 Inhibition Abolishes Chemoresistance

PDAC cells acquire resistance to gemcitabine via ploidy cycle

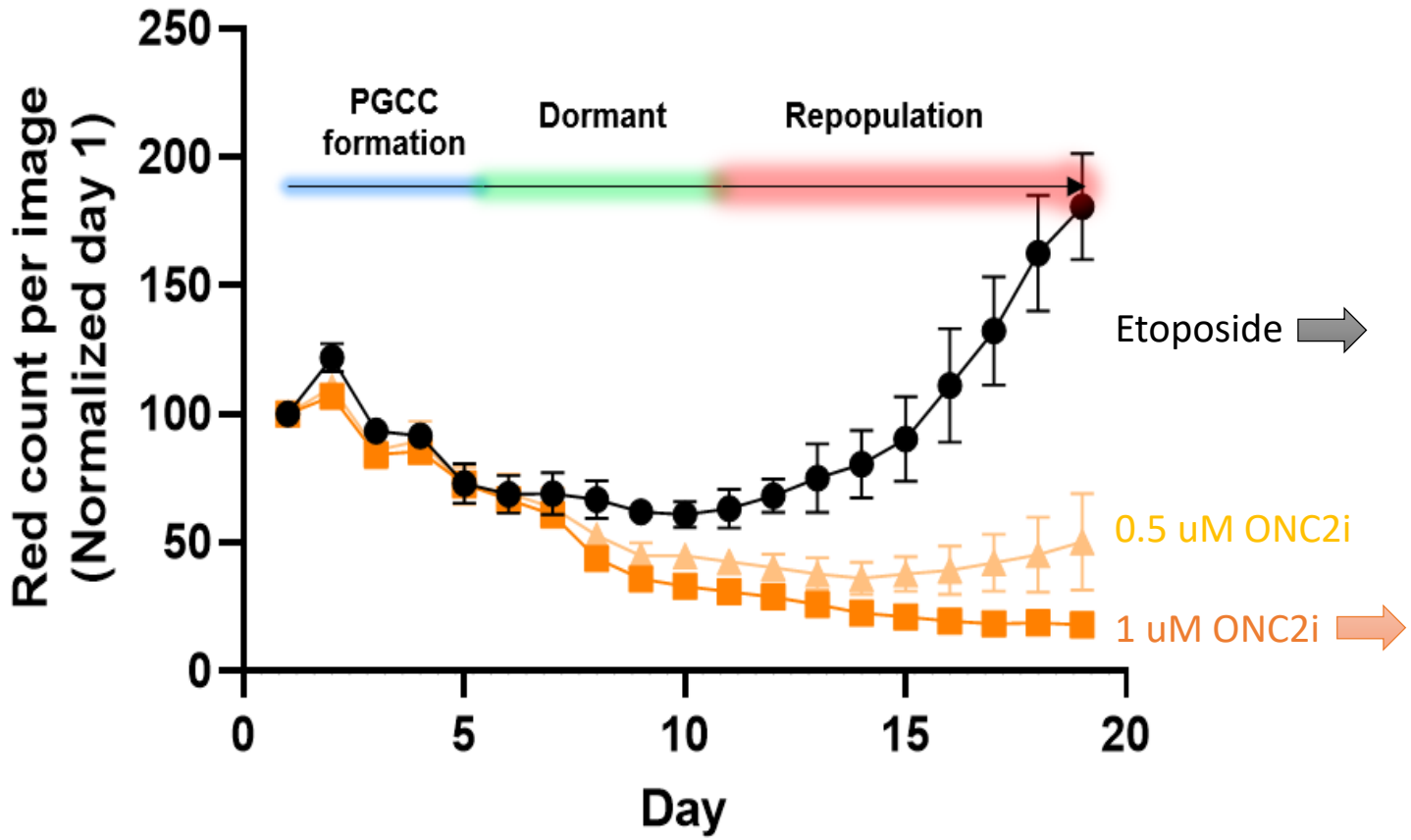


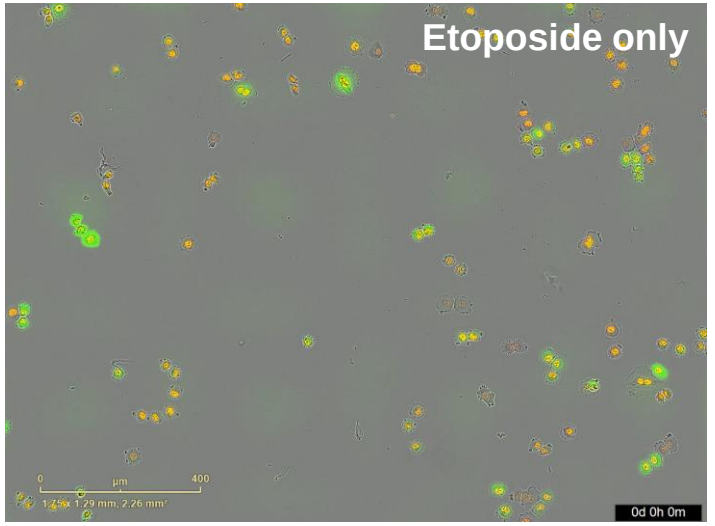
Combination with ONC1i

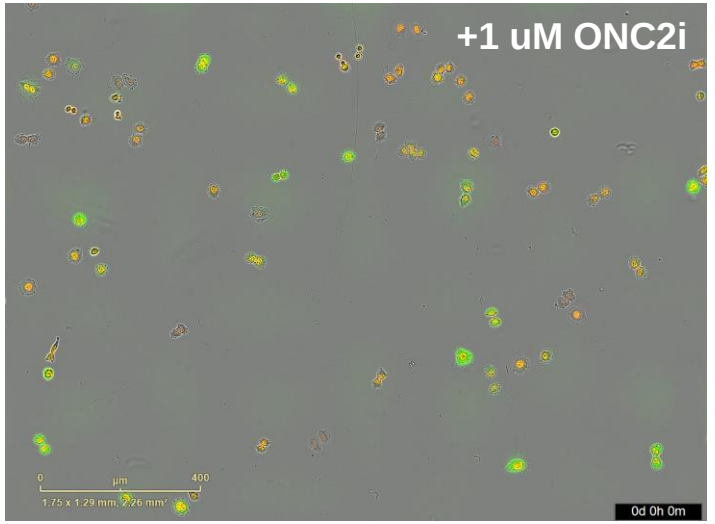




ONC2 Inhibition Abrogates Repopulation

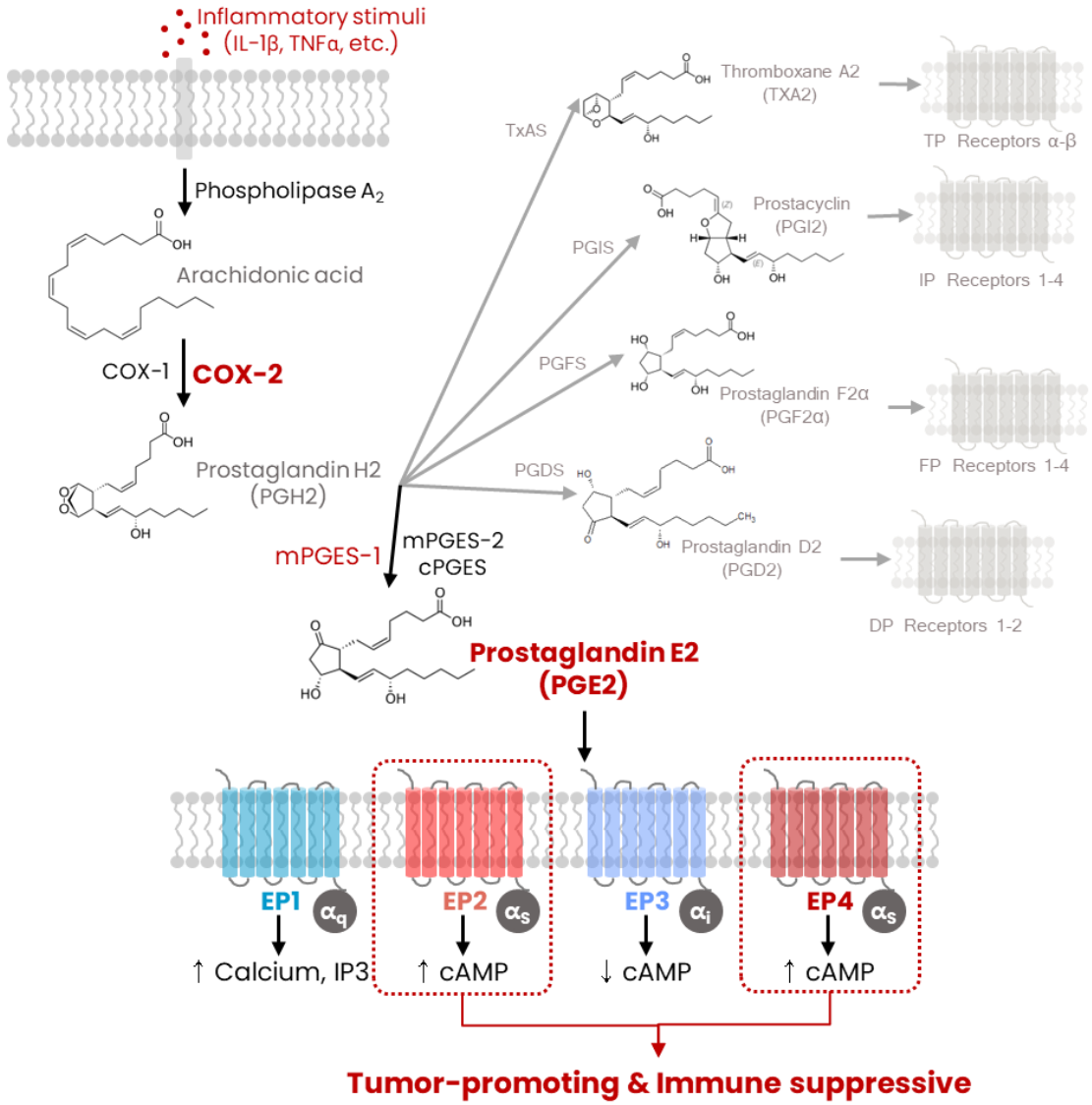






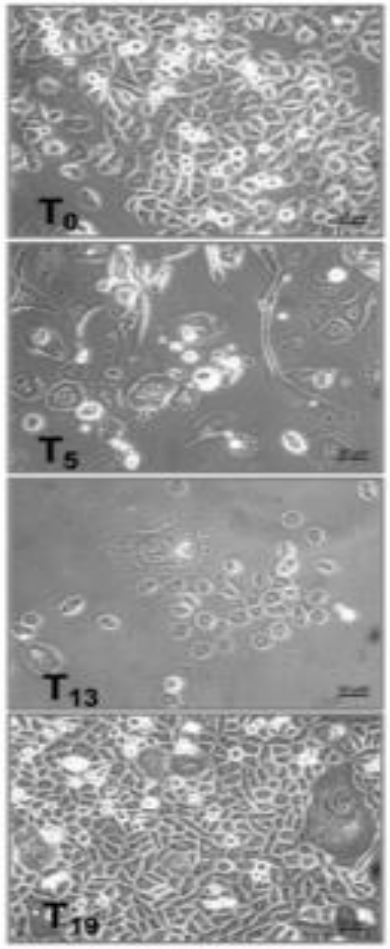
OCT-598; an EP2/4 Dual Antagonist

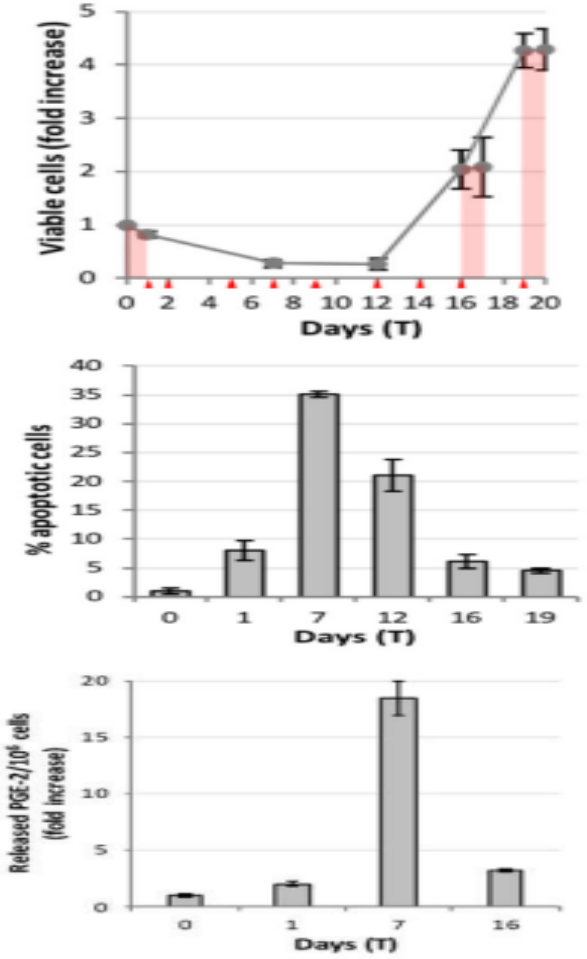
- Injury- and inflammation-induced apoptotic signal promotes the synthesis of prostaglandins from arachidonic acid by COX-2
- COX-2 inhibitors (e.g., NSAIDS) long known for putative anti-cancer effects
- Of the many PGs, PGE2 known to mediate tumor promotion and immune-suppression through EPs
- OCT-598 is a dual antagonist of EP2 and EP4, licensed from Kanaph Therapeutics

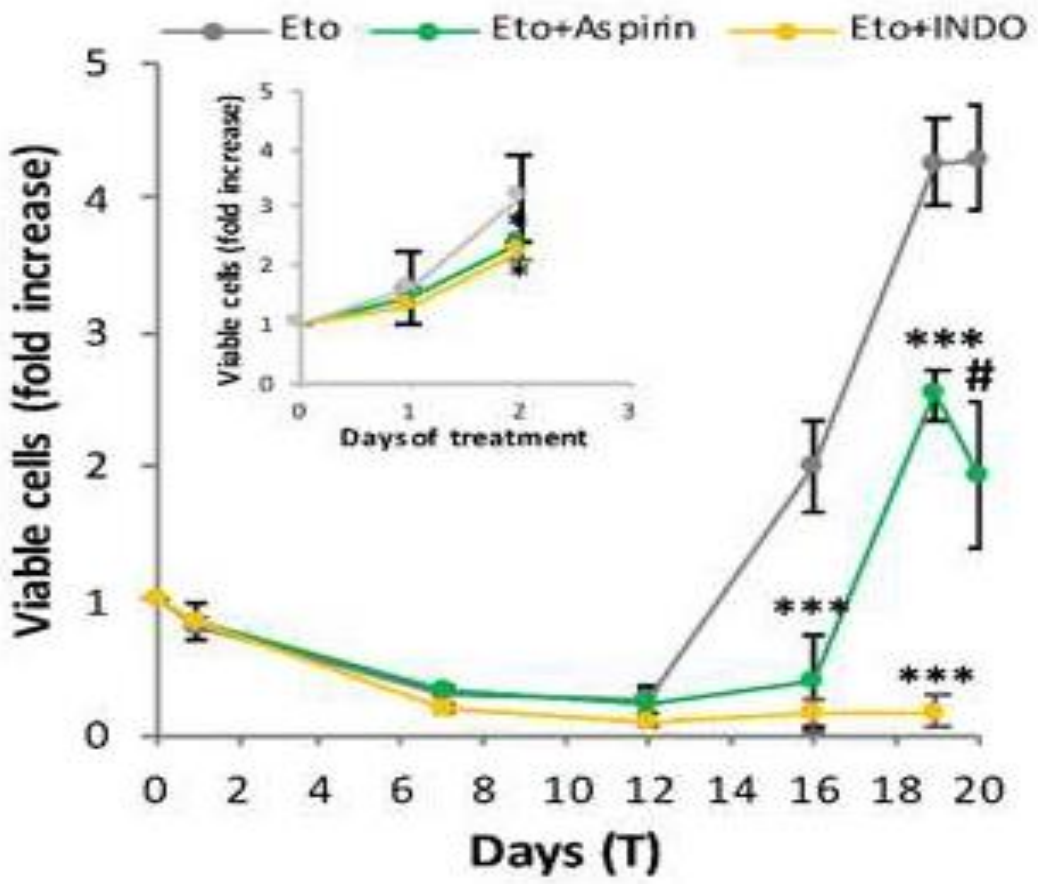


Targeting the 'Phoenix Rising' Pathway

- Indomethacin, a COX1/2 inhibitor, was reported to ameliorate development of chemoresistance and repopulation in PC3 cells



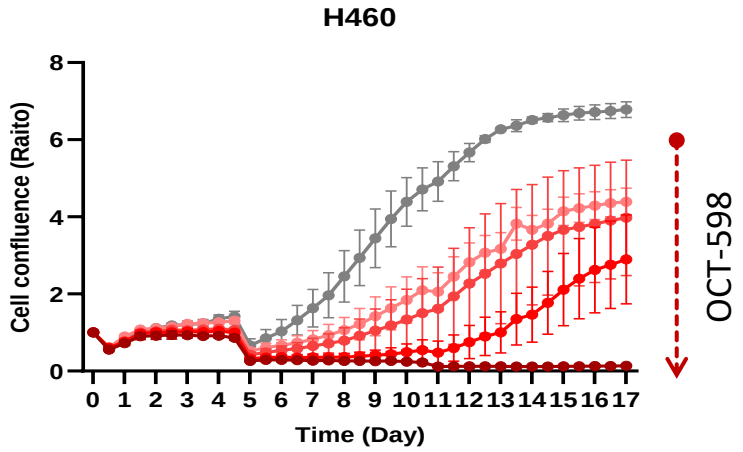




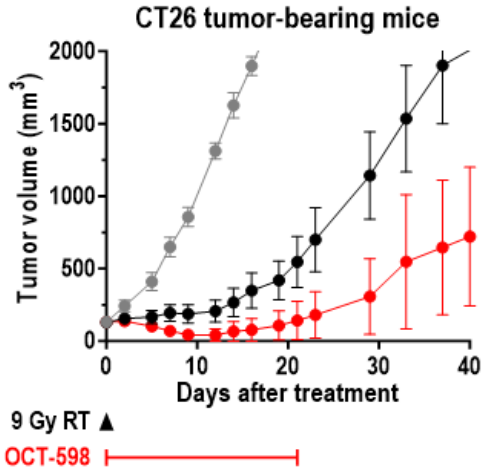
Corsi et al., Int. J Mol Sci 2022

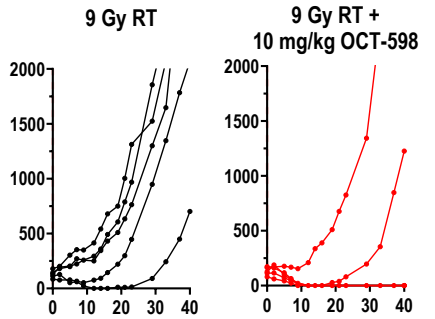
OCT-598 Proof-of-Concept

Inhibition of repopulation after docetaxel treatment



Inhibition of regrowth of CT26 tumor after irradiation

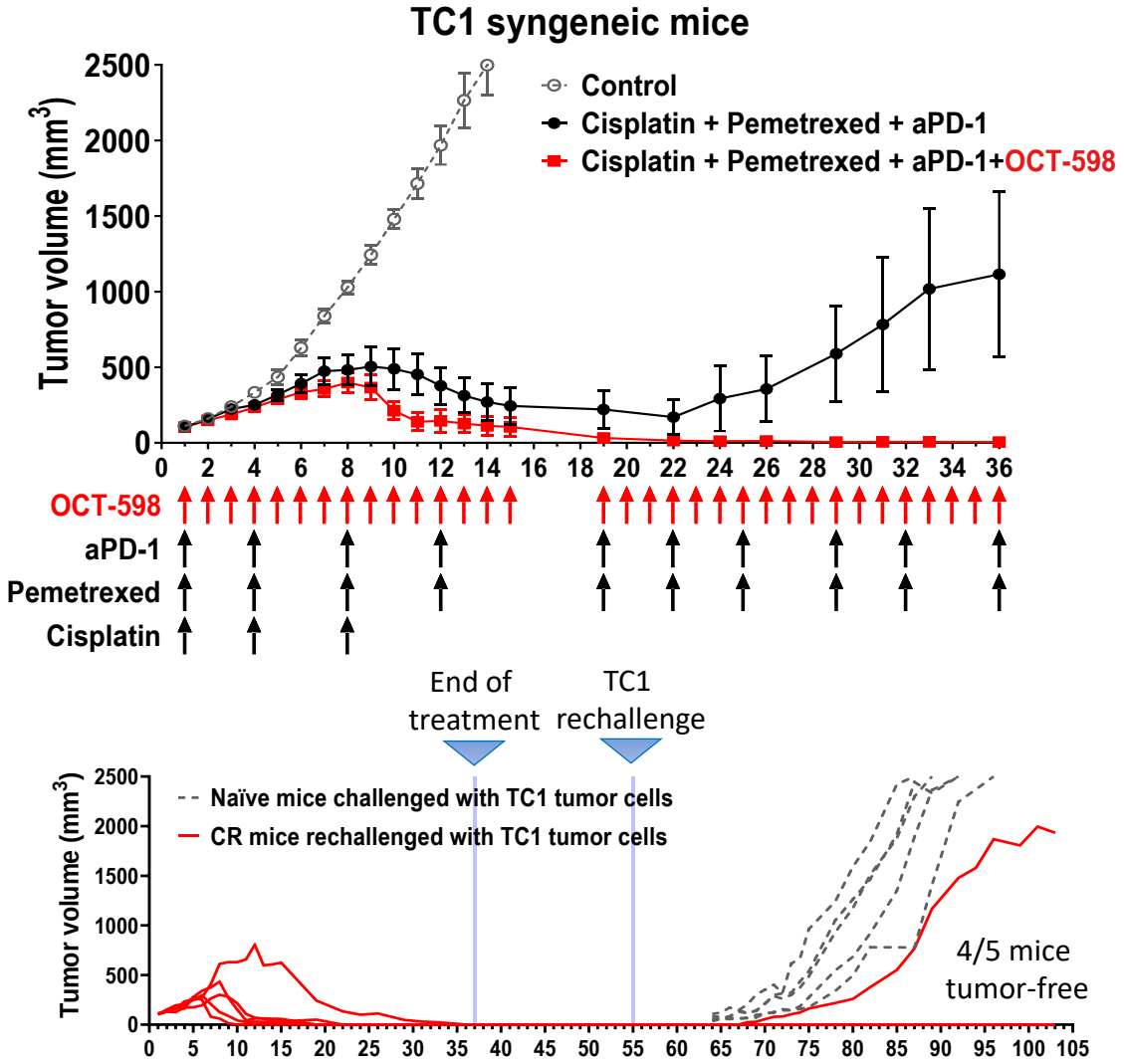




CR = 1/5
Relapse = 1/1

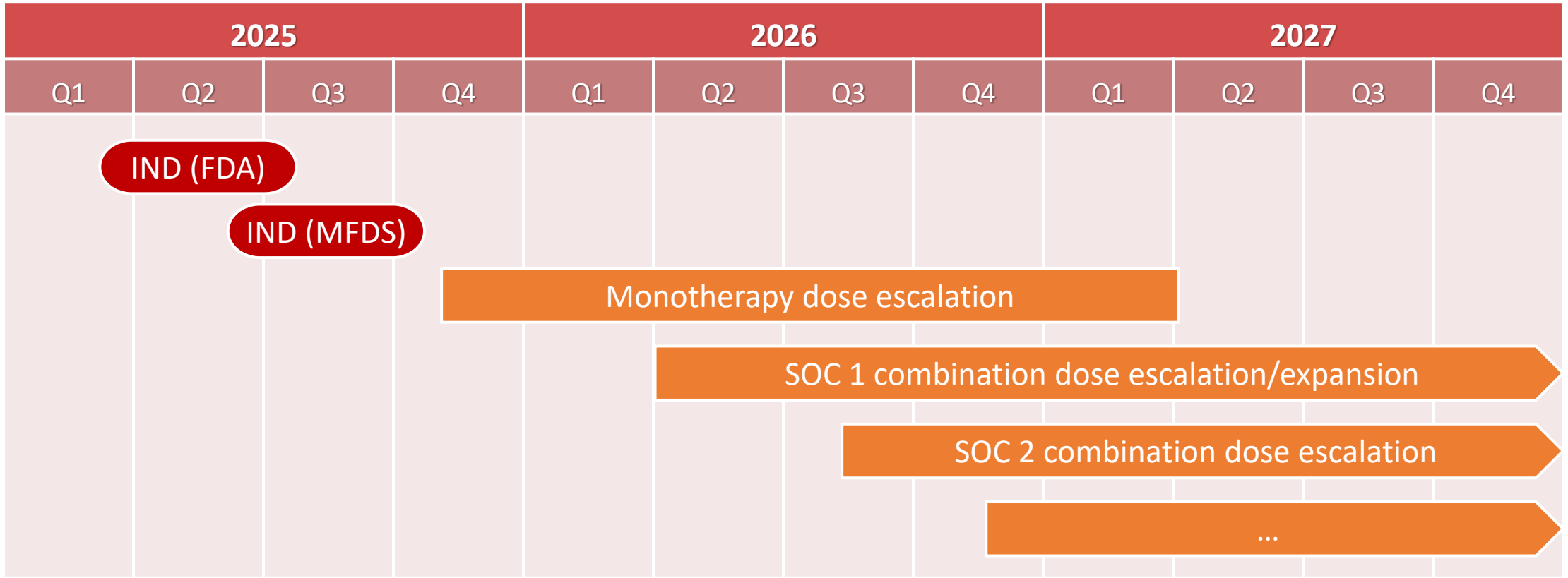
CR = 4/5
Relapse = 1/4

Complete remission of TC1 tumor after lung cancer SoC



OCT-598 to Enter the Clinic

- A Phase 1 Dose-Escalation Study to Evaluate the Safety, Tolerability, Pharmacokinetics, and Efficacy of OCT-598 as Monotherapy and in Combination With Standard-of-Care Treatment in Patients with Advanced Solid Tumors



Q & A