Inspirna

# Corporate Overview

Q1 2025

# Forward Looking Statements / Safe Harbor

This presentation contains forward-looking statements as defined in the Private Securities Litigation Reform Act of 1995. These statements may include the use of words or phrases such as “expects,” “plans,” “will”, “should”, “projects," or other similar words or phrases. These statements are predictions that are based on current information and they may not come to fruition. There are risks and unknowns that mean that Inspirna's actual results may change materially from the expected results. Inspirna has no approved drugs available for sale & marketing at this time and may never have an approved drug. Inspirna is highly dependent on its lead investigational drug candidate, which is in clinical development and may not reach regulatory approval(s). Inspirna faces significant risks involved in drug development including drug substance and drug product manufacturing & stability, contracting with contract research organizations (CROs) and clinical trial sites to conduct its clinical trials, and identifying and hiring high quality and trustworthy employees. Additional risks involve general regulatory and market risks, and competition from many cancer drug candidates in development. Inspirna may reach approval for RGX-202 (ompenaclid), RGX-104 (abequolixron), and/or RGX-019 and still not recoup the investment in the program if there are substantial pricing changes outside of its control, undue competition, or lack of demand or reimbursement due to alternative treatments or safety concerns. Inspirna’s RNA-DRIVEr platform has not yet yielded an approved drug and may not do so with existing or future drug candidates. Its success is based on several scientific activities being recapitulated in different tumor types and settings. It is also reliant on the availability of human tumor tissues, mouse models, genetic sequencing technology, bioinformatics expertise and adequate research staff and scientific leadership. Inspirna is highly dependent on patent protection for its programs, including licensed intellectual property, and the company is still prosecuting patent protection for key aspects of its programs in the US and abroad. You are cautioned not to rely on Inspirna’s forward-looking statements, which are only made as of the date hereof. Inspirna is under no obligation to update these statements.

# Company Highlights

- Inspirna's mission is to develop first-in-class drugs to treat cancer types of high-unmet medical need
- Lead drug candidate ompenaclid is a novel small molecule SLC6A8 inhibitor with activity in **pan-RAS mutant** colorectal cancer with Phase 2 randomized data in 1H 2025
- Second program abequolixron is a small molecule LXR agonist in Phase 1b/2 development for lung cancer
- Robust pipeline generated using novel miRNA-DRIVEr platform to discover key drivers of cancer
- Strong management team supported by top-tier institutional investors, collaborators and advisors

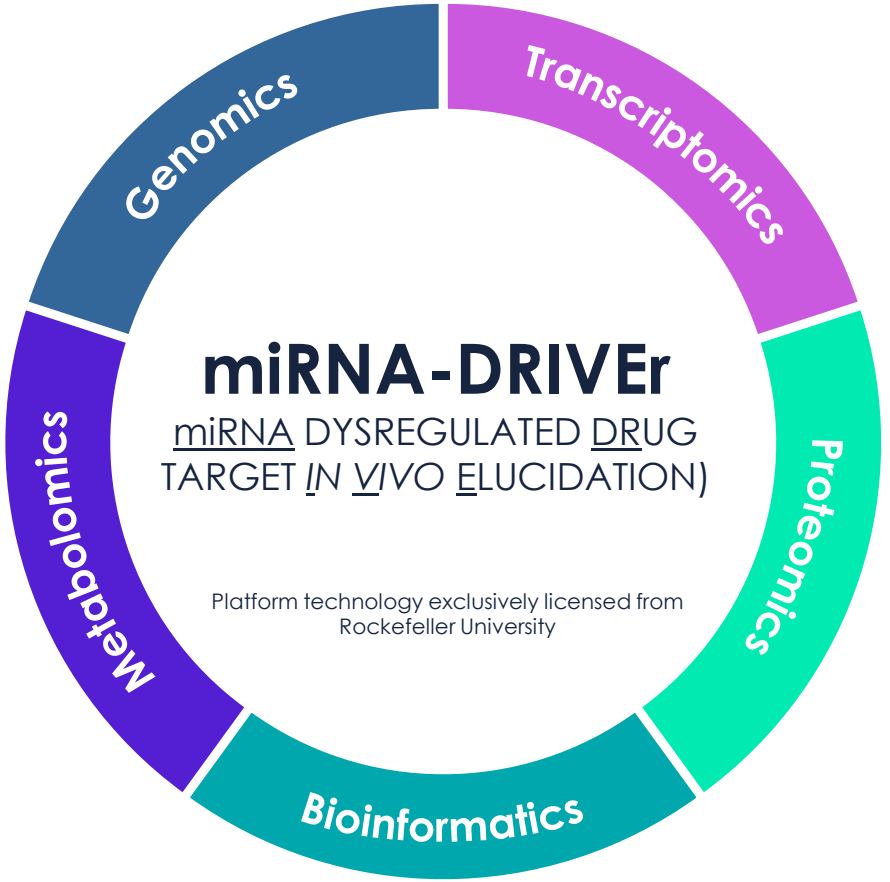
SLC6A8 -Solute Carrier family 6 member 8, LXR- Liver X Receptor, miRNA DRIVEr- miRNA DYSREGULATED DRUG TARGET *IN VIVO* ELUCIDATION

# miRNA-DRIVEr: Proprietary and clinically-validated platform to discover actionable targets of miRNA dysregulation

## *Platform technology exclusively licensed from Rockefeller University*

### Why target miRNA regulated pathways?

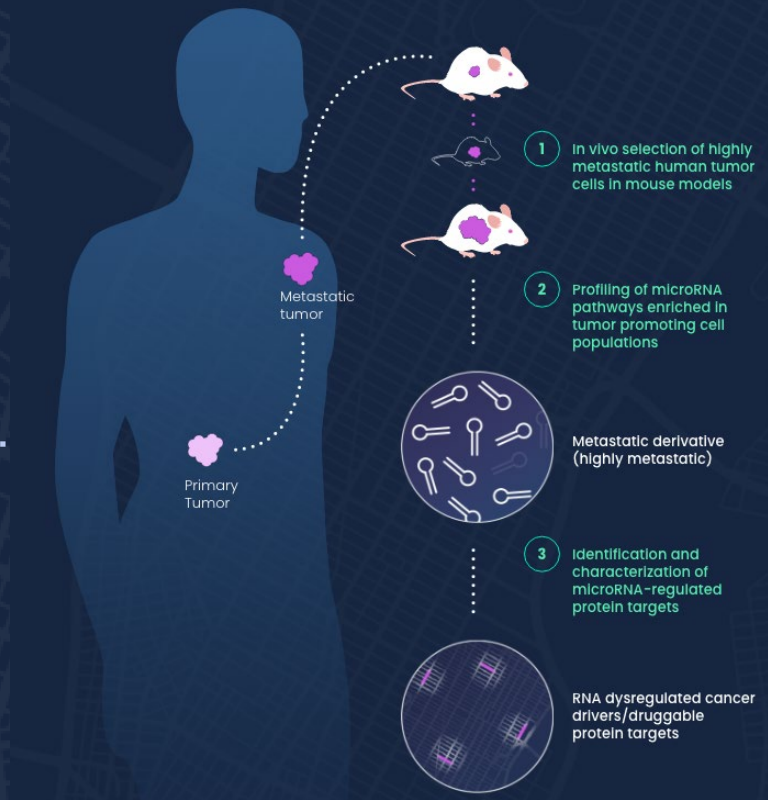
- Micro RNAs (miRNAs)* have important functions in controlling many cell properties:
  - Regulate gene expression at the post-transcriptional level;
  - Modulate cell growth and differentiation;
  - Regulate pathways and networks via coordinated activities;
  - Act as control nodes or hubs in regulatory networks; and
  - Act co-operatively with other miRNAs and with transcription factors, which are frequent targets of miRNAs
- miRNA dysregulation is a frequent contributor to cancer growth and progression.
  - Delineating miRNA functional effects requires elucidation of their upstream regulators and downstream targets




*short, endogenous noncoding RNAs 19–22 nucleotides long

# Overview - miRNA-DRIVEr platform

RNA-DRIVEr platform reveals actionable RNA-dysregulated cancer targets





**We discover critical targets that drive the growth of metastatic disease.**

**We test and validate the role of the discovered gene in driving metastatic disease.**

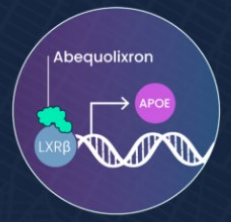

Tavazoie et al. Cell. 2018 Feb 8;172(4):825-840, 2. Loo JM et al. Cell. 2015 Jan 29;160(3):393-406, 3. Pencheva N et al. Cell. 2014 Feb 27;156(5):986-1001, 4. Pencheva N et al. Nat Cell Biol. 2013 Jun;15(6):546-54, 5. Pencheva N et al. Cell. 2012 Nov 21;151(5):1068-82, 6. Png KJ et al. Nature. 2011 Dec 14;481(7380):190-4

# Overview - miRNA-DRIVEr platform (continued)

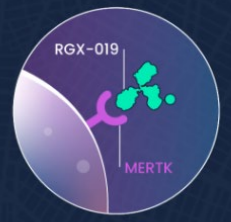
## *First-in-class targets discovered and developed*

RNA-DRIVEr platform reveals actionable RNA-dysregulated cancer targets (continued)

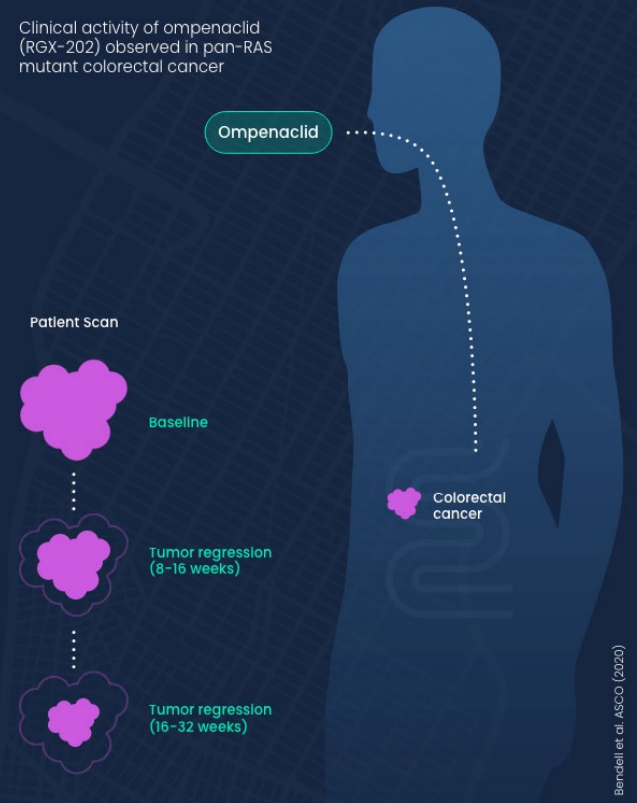

Ompenaclid (RGX-202)
(CKB/SLC6A8 pathway)




Abequolixron (RGX-104)
(APOE pathway)




RGX-019 ADC
(MERTK pathway)



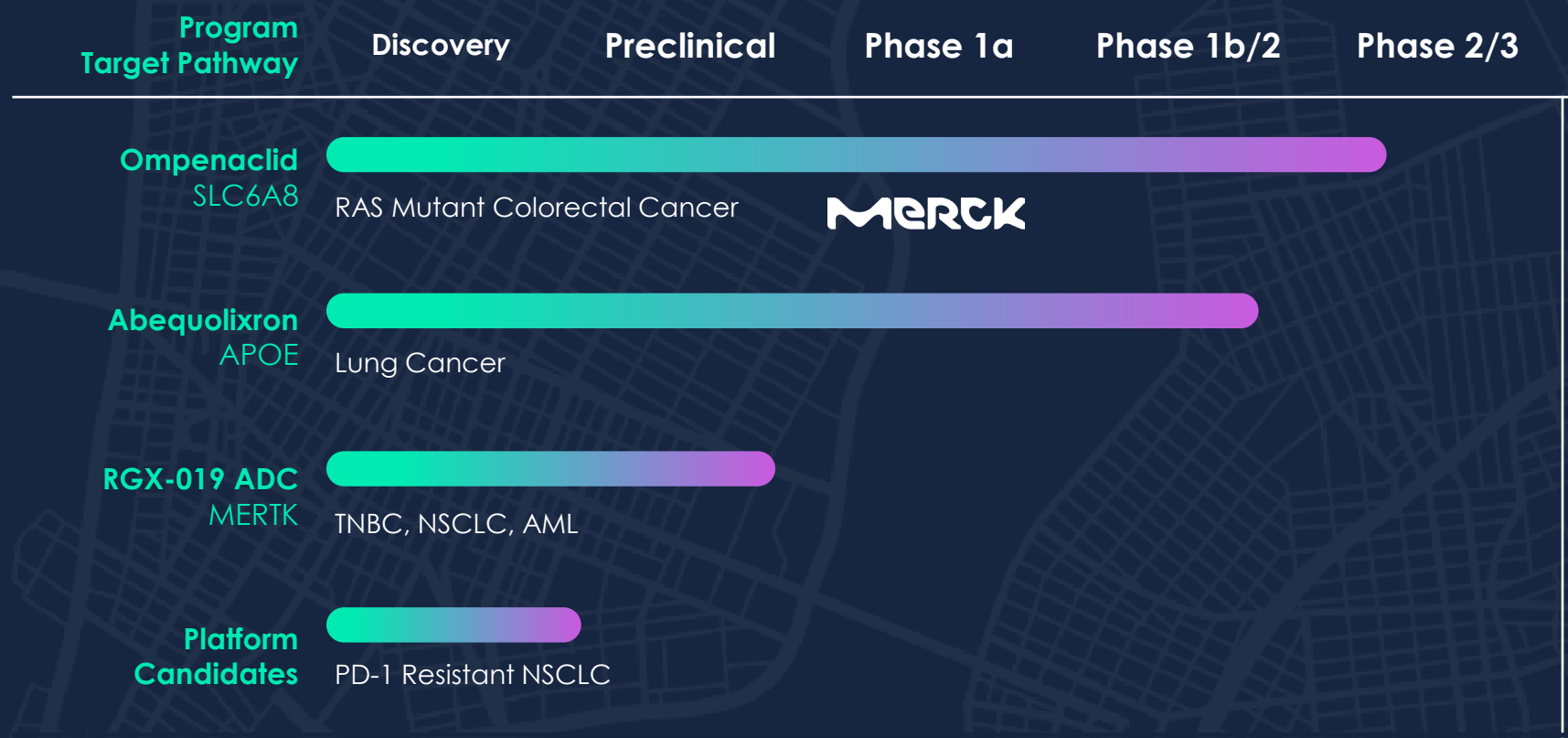

**Our experienced team develops new drug candidates suitable for clinical evaluation.**

**We then transform cancer medicine by leveraging RNA biology driven insights.**

CKB- Creatine Kinase B, SLC6A8- Solute Carrier family 6 member 8, APOE- Apolipoprotein E, MERTK- MER Proto-Oncogene, Tyrosine Kinase


Tavazoie et al. Cell. 2018 Feb 8;172(4):825-840, 2. Loo JM et al. Cell. 2015 Jan 29;160(3):393-406, 3. Pencheva N et al. Cell. 2014 Feb 27;156(5):986-1001, 4. Pencheva N et al. Nat Cell Biol. 2013 Jun;15(6):546-54, 5. Pencheva N et al. Cell. 2012 Nov 21;151(5):1068-82, 6. Png KJ et al. Nature. 2011 Dec 14;481(7380):190-4

# Inspirna's first-in-class oncology pipeline




**Key Milestones**

- Phase 2 RCT interim data in 1H 2025
- Phase 1b/2 follow up data in 2H 2024 (ESMO conference)

- Phase 1b/2 data read-out in 2H 2024 (SITC conference)

- Lead candidate identified

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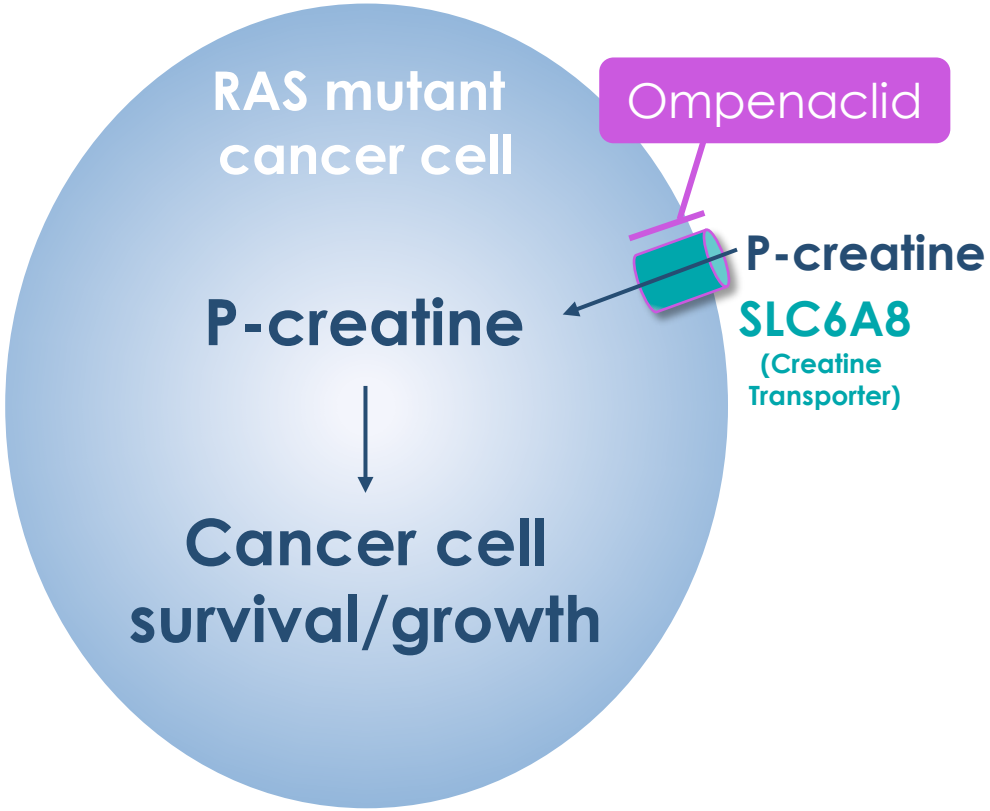
# Ompenaclid
(RGX-202)

- First-in-class orally-administered small molecule SLC6A8 inhibitor
- Targets key pathway that drives RAS mutant tumor growth
- Monotherapy clinical activity in RAS mutated relapsed/refractory metastatic colorectal cancer (CRC) demonstrated during Phase 1
- Clinical activity in combination with standard-of-care (SOC) demonstrated in patients with pan-RAS mutated second line advanced or metastatic CRC in an ongoing Phase 1b/2 trial
- Excellent safety profile with no dose-limiting toxicities observed as a single agent or in combination with SOC in Phase 1 dose escalations
- Completed enrollment of a Phase 2 double blinded randomized controlled trial
- Phase 3 preparation activities underway to enable a global pivotal trial
- Issued global patent protection to 2040+

*FOLFIRI: FOL – folinic acid (leucovorin), F – fluorouracil (5-FU), IRI irinotecan (Camptosar),

# Ompenaclid mechanism of action



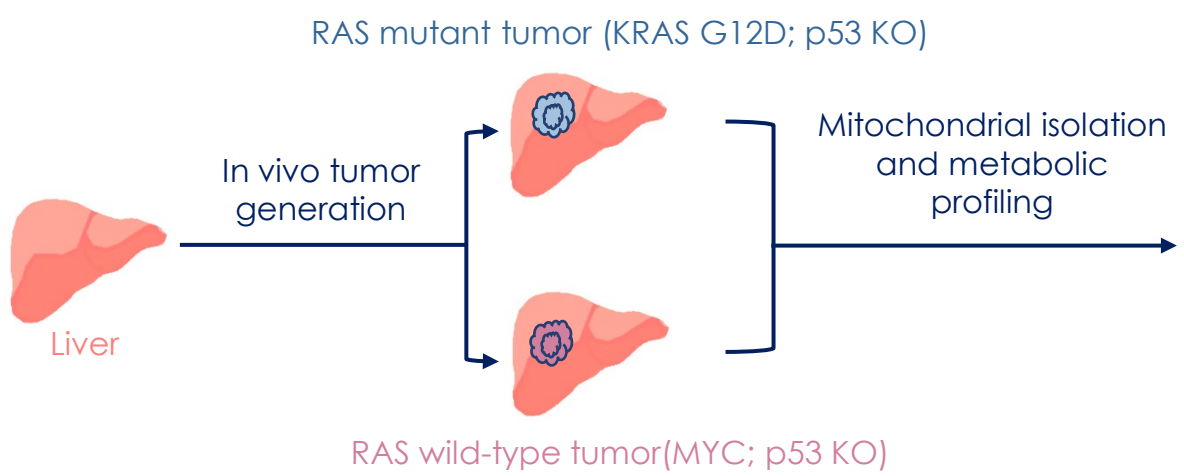

- Ompenaclid is an oral small-molecule competitive inhibitor of the SLC6A8 creatine transporter
- Blockade of SLC6A8 by ompenaclid depletes creatine and phospho-creatine in RAS mutant cancer cells, resulting in impairment of ATP synthesis and induction of apoptotic cancer cell death

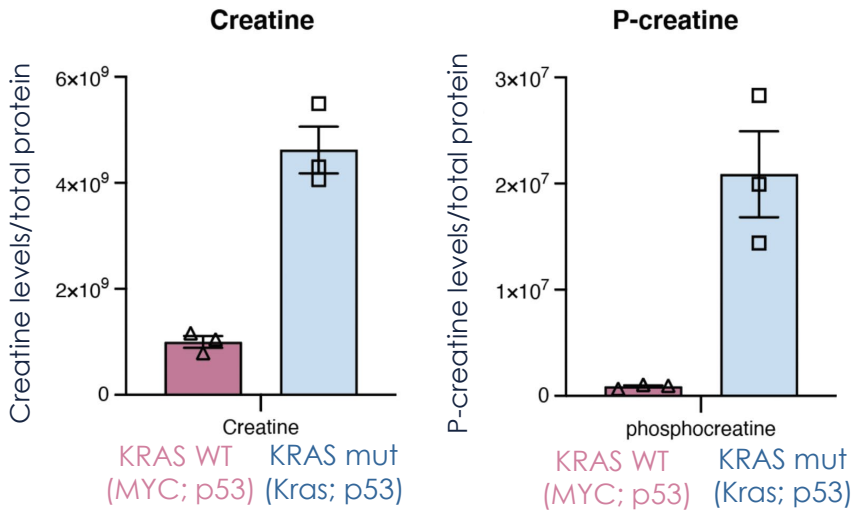
Sources: 1) Kurth et al. Sci Adv. 2021 Oct 8;7(41); 2) Loo et al. Cell. 2015 Jan 29;160(3):393-406; 3) Bendell et al. ASCO (2020); 4) Kurth et al. AACR (2018)

* Solute carrier family 6 member 8

# RAS mutated cancers are dependent on activated creatine metabolism

- RAS mutant tumors significantly activate creatine metabolism
- Metabolic profiling demonstrates substantial increase in creatine and phospho-creatine (P-creatine) levels in the mitochondria of RAS mutant tumors relative to RAS wild-type (WT) tumors or healthy tissue



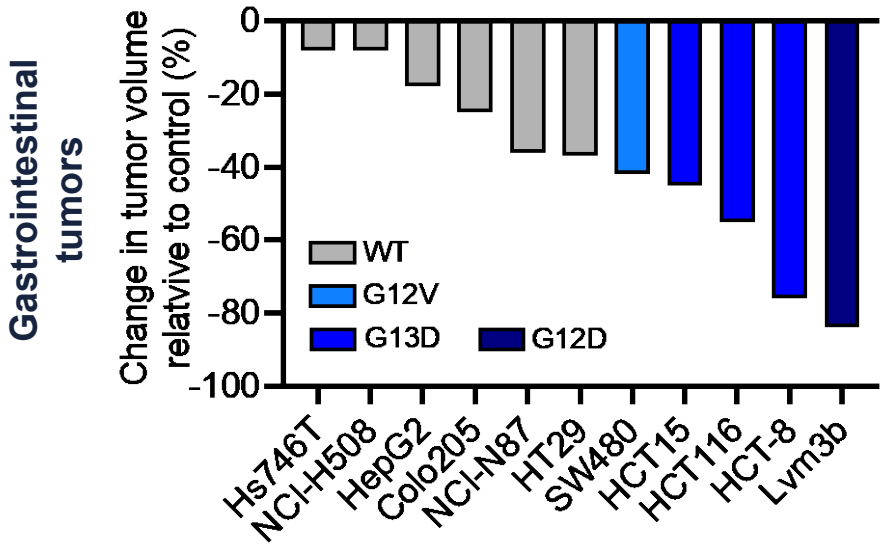



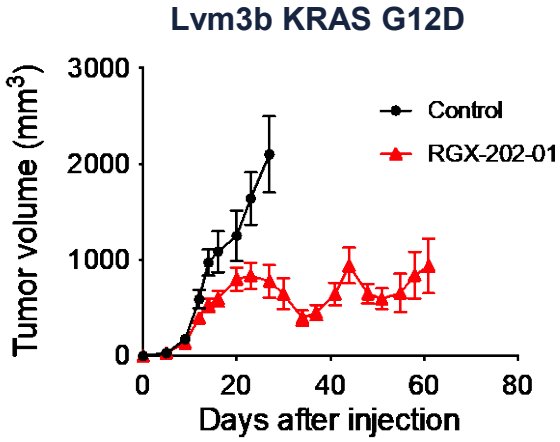

Creatine and phosphocreatine levels were measured in the mitochondria of p53 KO liver tumors that harbor either a KRAS G12D mutation or MYC overexpression. Tumors were induced by hydrodynamic injection of a plasmid carrying a p53 CRISPR construct to knock out p53, a mitochondrial tag and either MYC or KRAS G12D overexpression constructs.

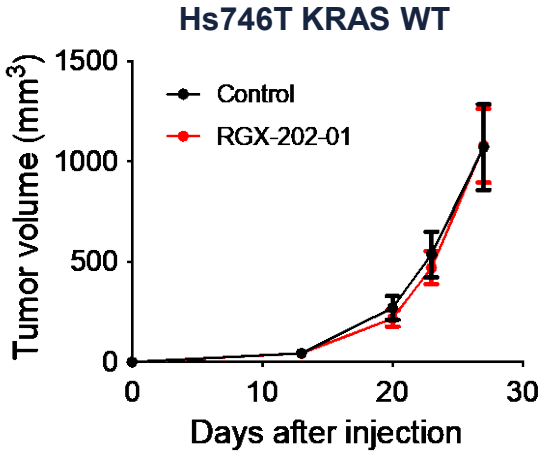
Source: Unpublished Data from Professor Kivanc Birsoy, Rockefeller University

# Greater sensitivity to ompenaclid is seen in RAS mutated xenografts

- Anti-tumor efficacy is seen in both KRAS WT and mutant tumor models
- Efficacy is greater in KRAS mutant tumors



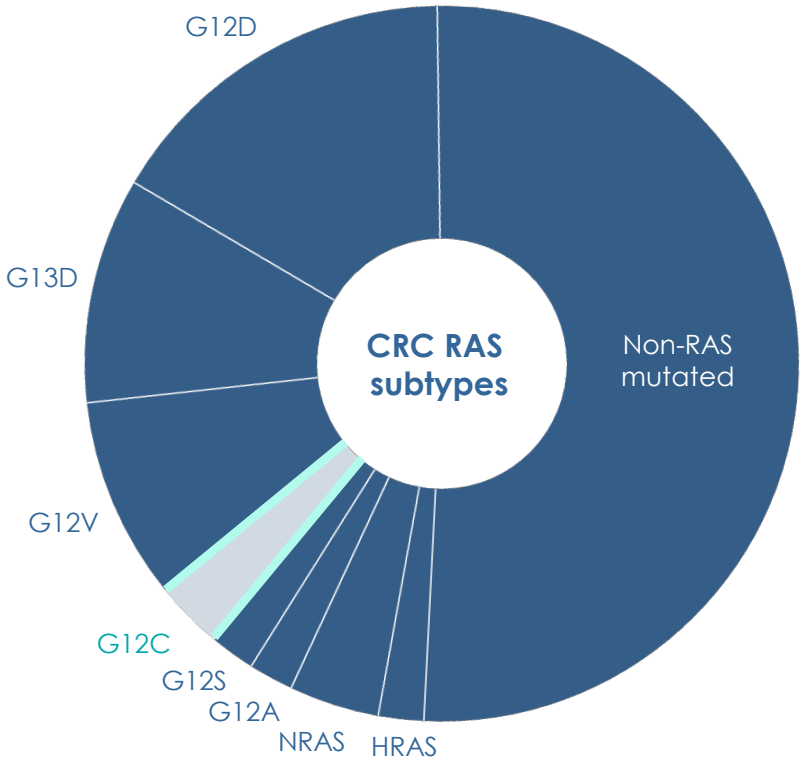

(**Left**) Waterfall plot of tumor size change relative to control tumors. Indicated cell lines were injected subcutaneously into athymic nude mice. Treatment with a control or RGX-202-01 formulated chow (800 mg/kg) started when tumors reached 50-150 $mm_3$; n=8-10. (**Right**) Tumor growth of representative KRAS mutant CRC and WT cell lines.

# RAS-mutated colorectal cancer (CRC) remains a high-unmet medical need requiring a novel targeted therapy

- CRC is the 3rd most common cancer diagnosed in the United States
- ~45% of patients with CRC have RAS-mutated cancer
- Only ~3% of CRC can be addressed with targeted KRAS G12C inhibitors currently in development

mCRC KRAS G12C mutation patients in USA ***at peak sales year ~2,000***

**Potentially addressable CRC patient population**
*(KRAS G12C inhibitors)*




Sources: 1) American Cancer Society; 2) Serebriiskii et al. Nat Commun 10, 3722 (2019); 3) Pfeiffer P, Qvortrup C. Lancet Oncol. 2022 Jan;23(1):10-11; 4) Fakih et al; The Oncologist, 2022, 27, 663–674

# Ompenaclid is a first-in-class clinical stage SLC6A8 inhibitor with activity in pan-RAS-mutated CRC

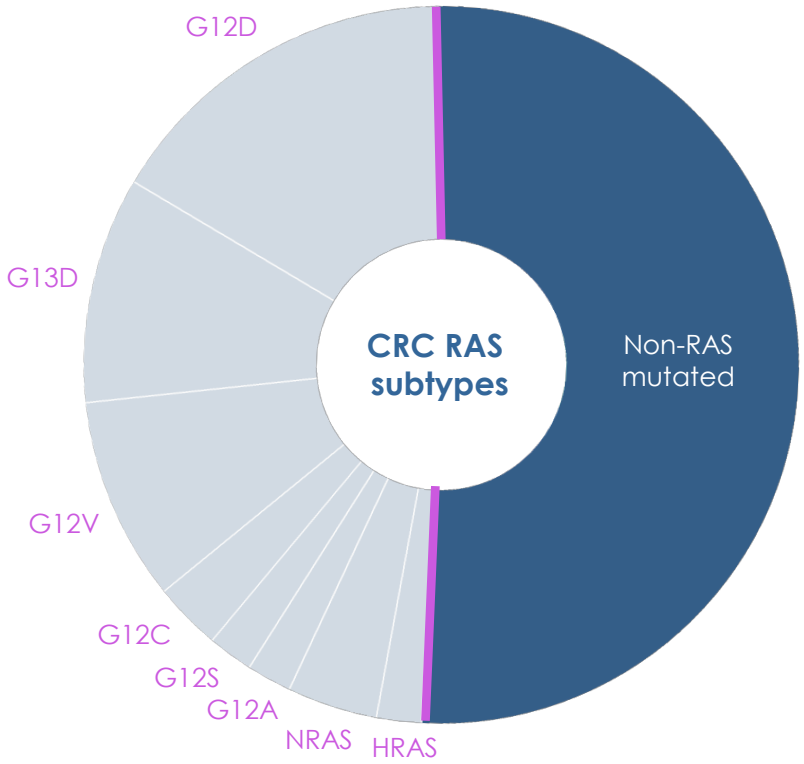
**Ompenaclid targets an essential dependency in pan-RAS-mutated tumors with potential to address ~45% of CRC patient population**

mCRC pan-RAS mutation patients in USA ***at peak sales year ~20,000***

- Clinical activity observed against RAS-mutated CRC tumors (e.g. G12V, G13D, G12D, etc.) outside of G12C mutant cancers
- Orally-administered drug candidate with excellent safety profile observed to date

## Potentially addressable CRC patient population

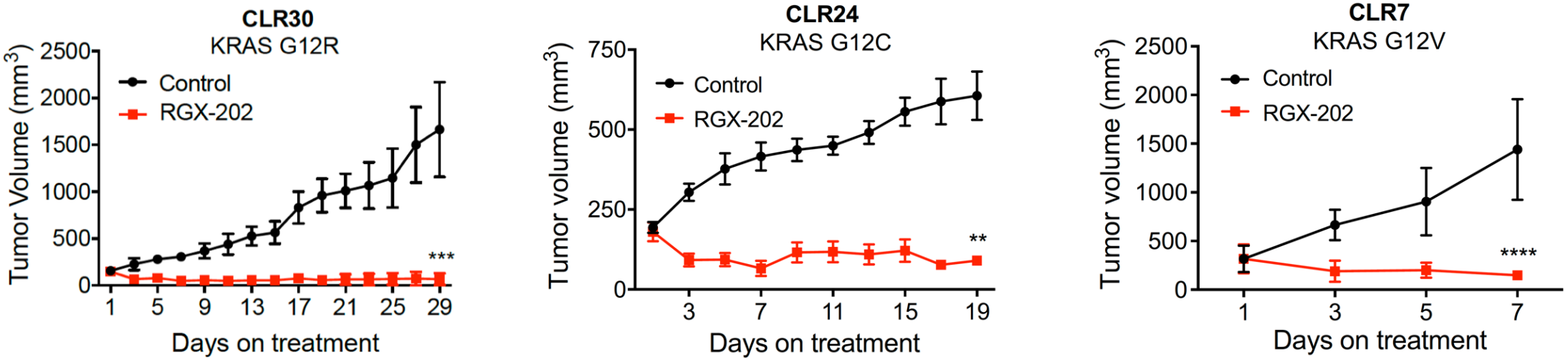
*(ompenaclid)*




Sources: 1) Bendell et al. ASCO (2020); 2) Hendifar et al. ASCO (2022); 3) Fakih et al; The Oncologist, 2022, 27, 663–674

# Ompenaclid has potent anti-tumor activity in CRC models across RAS mutant subtypes

## Regressions of large established KRAS mutant CRC tumors treated with ompenaclid (RGX-202) (patient derived xenografts)




Treatment with control or RGX-202 at 800 mg/kg (daily) started when average tumor size reached 250 $mm^3$ (CLR7), 200 $mm^3$ (CLR24) and 150 $mm^3$ (CLR30), n = 5 per cohort; ****p<0.0001 ***p<0.001, **p<0.01

Sources: 1. Kurth et al. Sci Adv. 2021 Oct 8;7(41); 2. Bendell et al. ASCO (2020); 3. Kurth et al. AACR (2018)

# Ompenaclid monotherapy dose escalation Phase 1 *Efficacy Overview*

**Completed monotherapy Phase 1a dose escalation in patients with refractory/relapsed GI cancers**

**Total patients enrolled**
CRC (n=13); pancreatic (n=4)
600mg, 1200mg, 2400mg and 3600mg (all BID)

**Patient Demographics**

| | |
|---|---|
| >3 prior lines of therapy | 12 (70.6%) |
| 2-3 prior lines of therapy | 5 (29.4%) |
| RAS mutant | 10 (58.8%) |
| RAS WT/unknown | 7 (41.2%) |
| MSI-High | 0 (0%) |
| ECOG 0 | 5 (29.4%) |
| ECOG 1 | 12 (70.6%) |

**Efficacy**
n = 10 CRC evaluable

**KRAS mutant**
n = 5

**1PR, 2SD, 2PD**

- cPR at 3600mg dose
  (KRAS G12V; DOR: 6 months)
- SDs at 3600mg and 1200mg
  (both KRAS G13D)

cPR = confirmed PR

**KRAS WT**
n = 5

**0PR, 1SD, 4PD**

**Benchmark ORR for SOC (TAS-102; regorafenib) in same clinical setting is < 2%[1,2]**

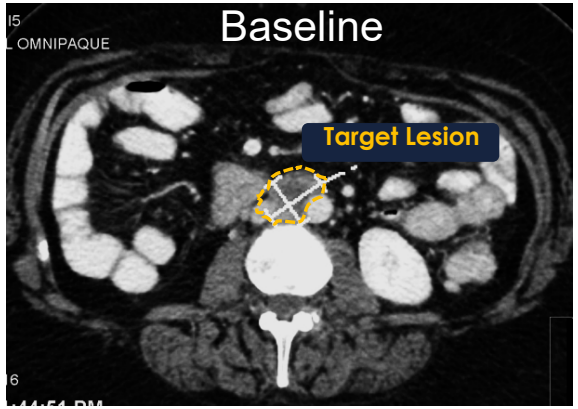
[1]Mayer et al. *N Engl J Med* 2015; 372:1909-1919;
[2]Grothey et al. *Lancet*. 2013;381:303–312

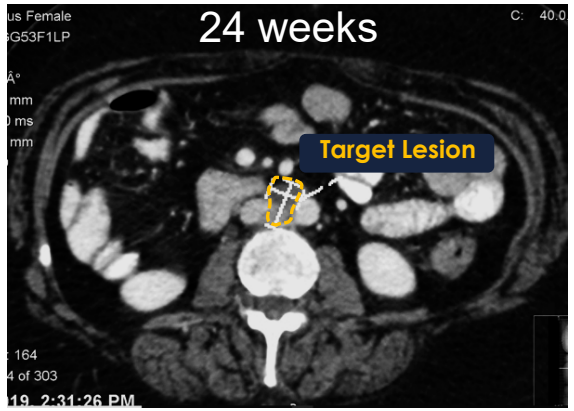
- Safety demonstrated
- Anti-tumor activity observed in KRAS mutant CRC at doses >2400mg BID
- Moved program rapidly into combination 2L setting on strength of preclinical and initial phase 1 monotherapy data

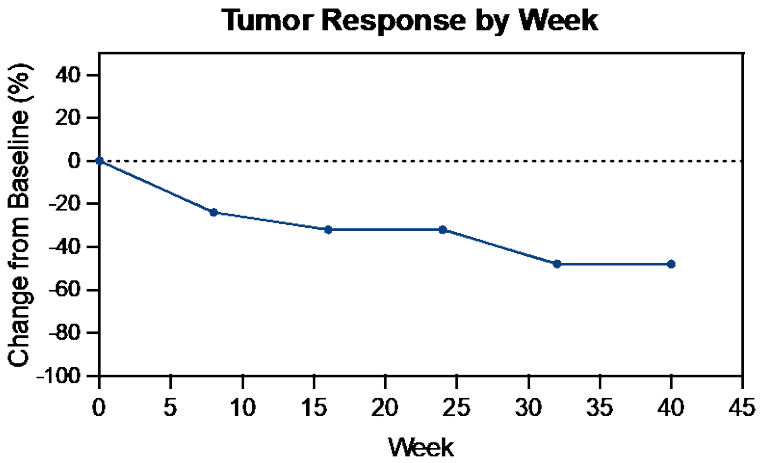
# Ompenaclid single agent clinical activity – Phase 1 data presented at ASCO 2020

## Confirmed Partial Response (PR) with single-agent ompenaclid in 55-year-old woman with KRAS$^{G12V}$ mutant colon cancer

- Patient had 6 prior lines of therapies including regimens containing 5-FU, oxaliplatin, irinotecan, and bevacizumab (standard-of-care)
- Treated with ompenaclid monotherapy in Phase 1a dose escalation study
- PR by RECIST 1.1 at 16 weeks (confirmed at 24 weeks) with progression at week 40 with growth of a non-target lesion (6-month duration of response; 48% tumor shrinkage)




Bendell et al. ASCO (2020)

# 2L CRC is a setting of high unmet medical need

- The most commonly used standard-of-care (SOC) regimen for 2L metastatic CRC is FOLFIRI* + bevacizumab
- The benchmark efficacy data for this regimen in published clinical studies[1-5] are:
  - **Response Rate (ORR): ~15%**
  - **Median PFS: ~6 months**
- Patients with KRAS mutant tumors trend towards worse outcomes with standard-of-care than those with KRAS WT tumors

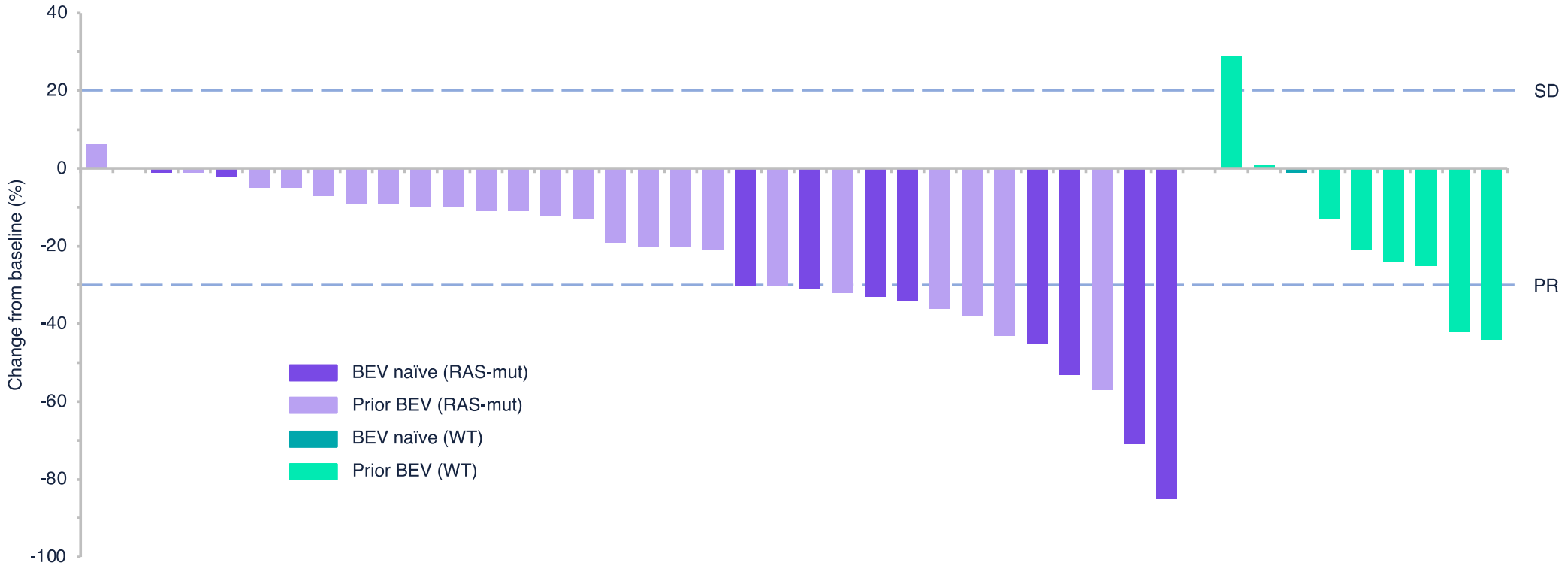
*FOLFIRI: FOL – folinic acid (leucovorin), F – fluorouracil (5-FU), IRI irinotecan (Camptosar),

1) (ML18147 Trial): Bennouna et al. Lancet Oncol. 2013 Jan;14(1):29-37, 2) (TRUSTY Trial): Terazawa et al. ASCO (2021), 3) (SPIRITT Trial): Hecht et al. Clin Colorectal Cancer. 2015 Jun;14(2):72-80, 4) Ottaiano et al. Front. Oncol. 9:766. (2019), 5) Yamada et al. Anticancer Res. 2021Jan;41(1):533-541

# Efficacy Overview

41% ORR observed in RAS mutant patients
30% ORR (ITT) observed in RAS mutant patients

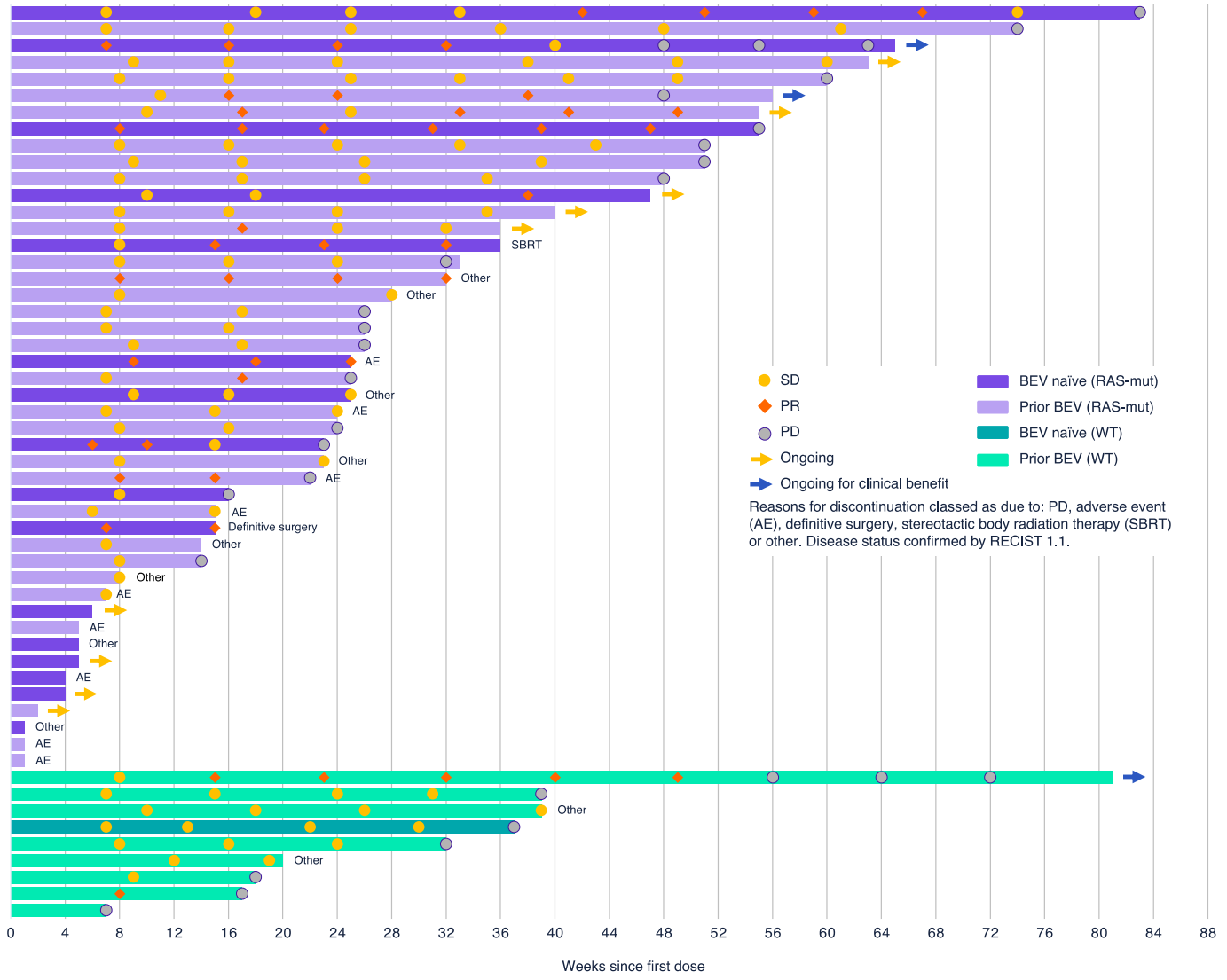
Best response in all evaluable patients (n=43)




Percentage change from baseline of tumors in evaluable patients. Data cut-off 4 Jun 2024; open database, data subject to change.
Presented at ESMO 2024.

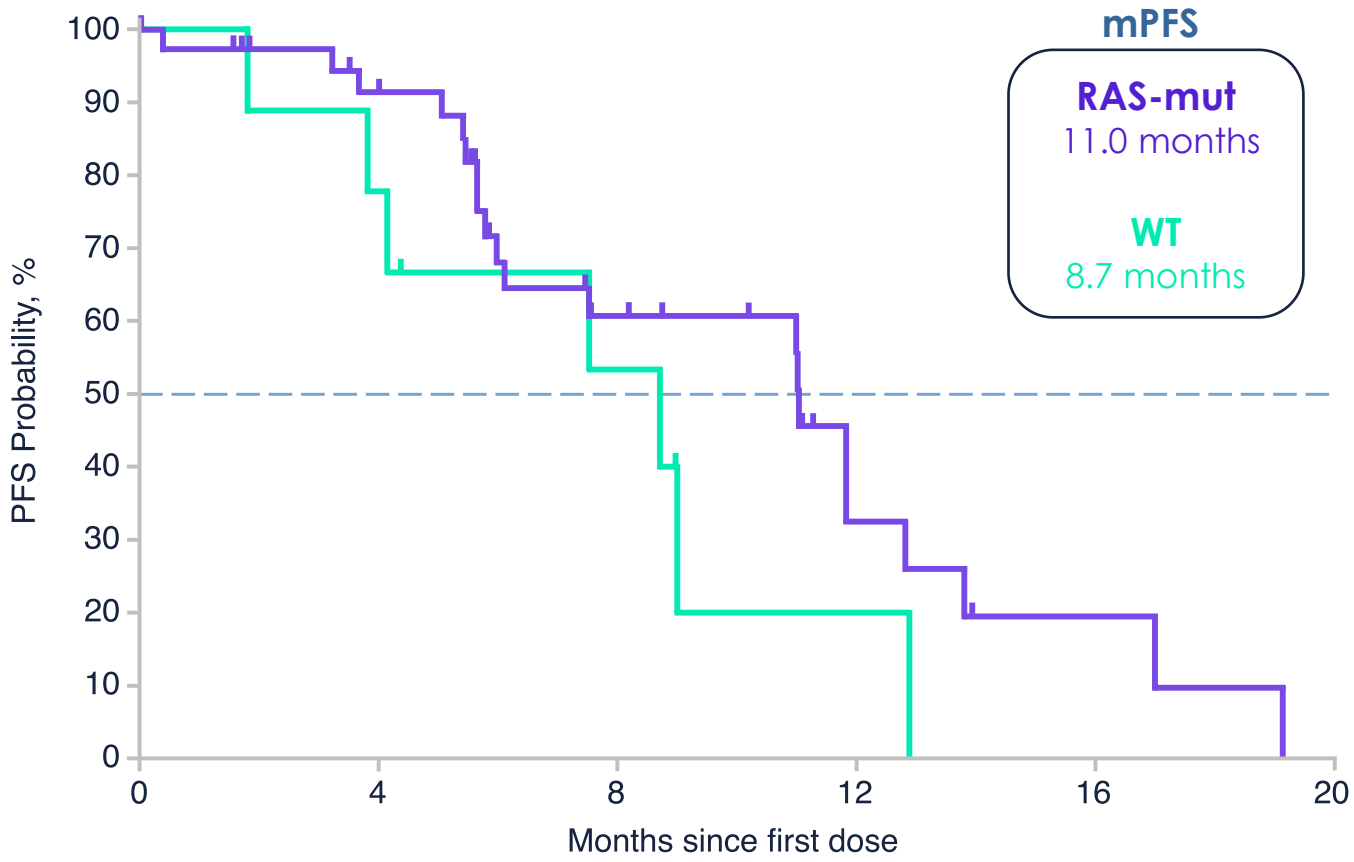
# Duration of treatment and response in all patients (n=55)

Data cut-off 4 Jun 2024; open database, data subject to change. Presented at ESMO 2024.



# PFS in RAS-mut vs WT mCRC

## Ompenaclid + SOC increased PFS in RAS-mut vs WT mCRC



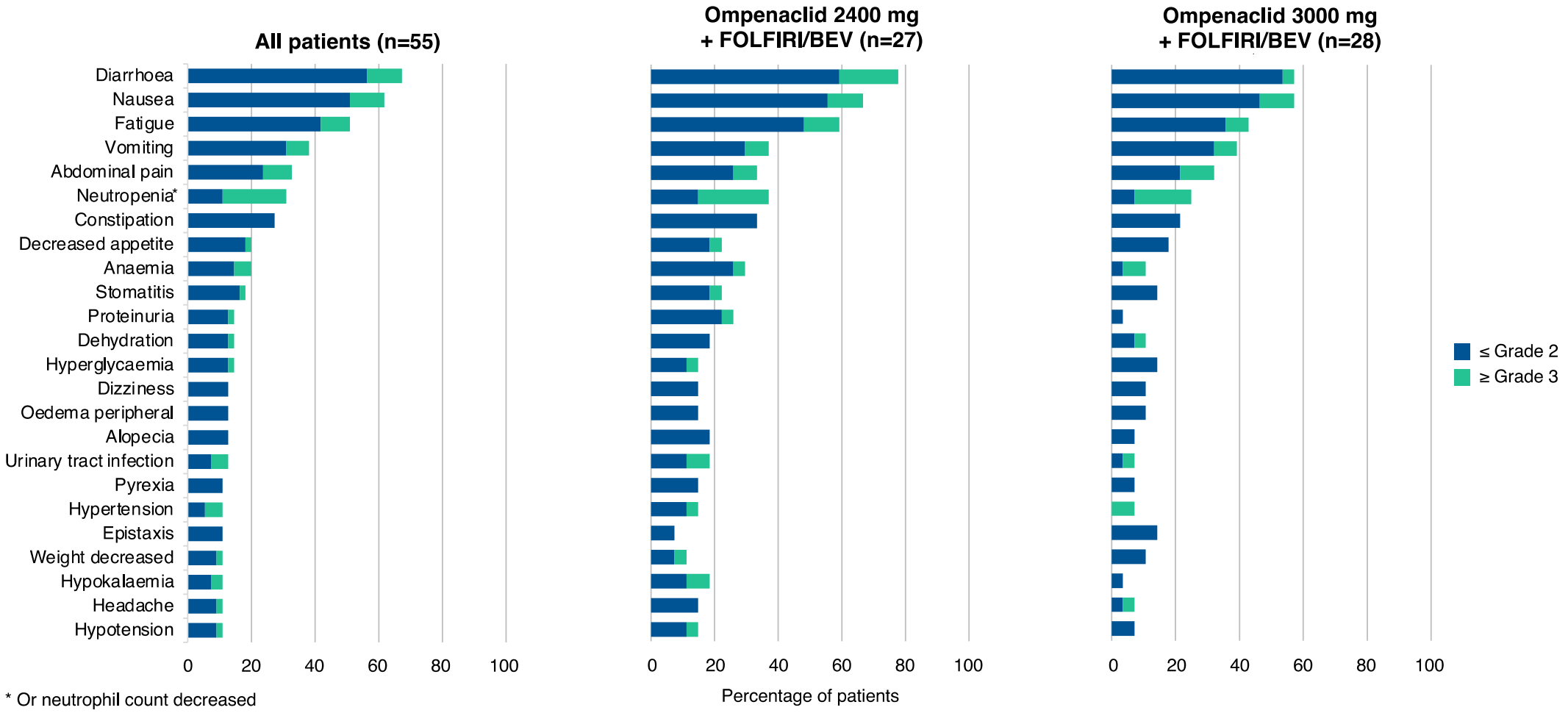

PFS analysis included all patients enrolled (n=55). PFS data are not yet final as of the cut-off date 4 Jun 2024 due to continuing patient follow-up and the limited number of PD events. Presented at ESMO 2024.

# Safety summary – ompenaclid + FOLFIRI/ bevacizumab

- In the Dose Escalation Phase, there were no DLTs observed for either the 2400 mg BID or 3000 mg BID dose with the combination.
- The most common Grade ≤ 2 TEAEs were diarrhea (56%) and nausea (51%).
- The most frequent Grade ≥ 3 TEAEs were neutropenia (20%), diarrhea (11%) and nausea (11%).
- The only Grade 5 TEAE was 1 patient with an intestinal perforation, deemed related to BEV.
- At the evaluated dose levels, ompenaclid added to FOLFIRI/BEV was well tolerated.

Data cut-off 4 Jun 2024; open database, data subject to change.
Presented at ESMO 2024.

# Ompenaclid Safety – TEAEs occurring in ≥ 10% of patients




* Or neutrophil count decreased

Data cut-off 4 Jun 2024; open database, data subject to change.
Presented at ESMO 2024.

# Ompenaclid + FOLFIRI/ Bev compared to historical SOC with FOLFIRI combined with Bev or other approved anti-angiogenic inhibitors

| | Ompenaclid (Bevacizumab) | RAISE* (Ramucirumab) | VELOUR** (Aflibercept) | ML18147*** (Bevacizumab) |
|---|---|---|---|---|
| ORR (WT and RAS) | 41% (RAS only)[1]<br>30% (RAS only, ITT)[1] | 13% | 19% | 5% |
| DCR (WT and RAS) | 100% (RAS only) [1] | 74% | 85% | 88% |
| **mPFS (RAS)** | **11.0 months[1]** | **5.7 months** | **Unreported** | **5.5 months** |
| **mOS (RAS)** | **19.1 months[2]** | **12.9 months** | **12.6 months** | **10.4 months** |
| mPFS (WT and RAS) | | 5.7 months | 6.9 months | 5.7 months |
| mOS (WT and RAS) | | 13.3 months | 13.5 months | 11.2 months |

*Patients had either prior Oxaliplatin- or Irinotecan- first line therapies and Investigators had the option to use either Oxaliplatin- or Irinotecan-based second line therapies

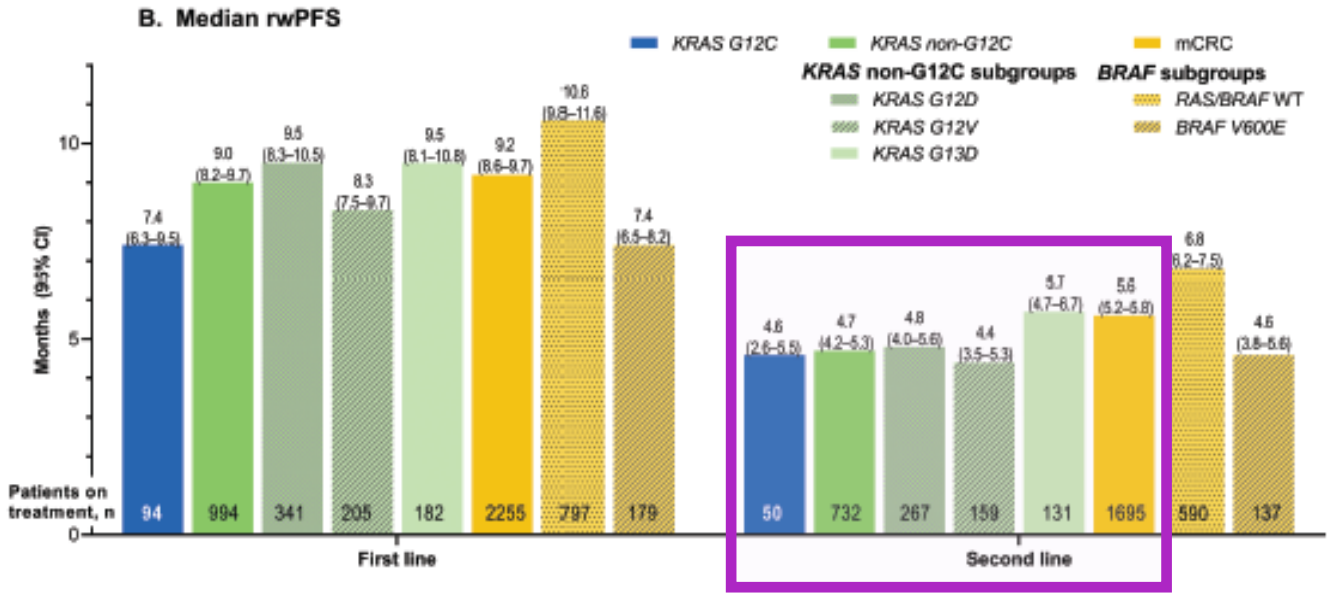

1. ESMO 2024, Hendifar et al.
2. ESMO 2023, Hendifar et al.

*RAISE Trial: Tabernero et al. Lancet Oncol. 2015 May;16(5):499-508
**VELOUR Trial: Van Cutsem et al. Journal of Clinical Oncology 2012 30:28, 3499-3506
***ML18147 Trial: Bennouna et al. Lancet Oncol. 2013, Jan;14(1):29-37,

# Real-world data on PFS in 2nd line RAS mutant mCRC

- Largest and most robust real world / RWE study in over 6000 mCRC patients, with and without RAS mutations
- Treatment patterns are generally comparable in patients in mCRC with or without the KRAS p.G12C mutation and consistent across successive LOT*
  - Since anti-VEGF therapy introduced over a decade ago in 2L CRC, standard of care unchanged

* LOT- line(s) of treatment



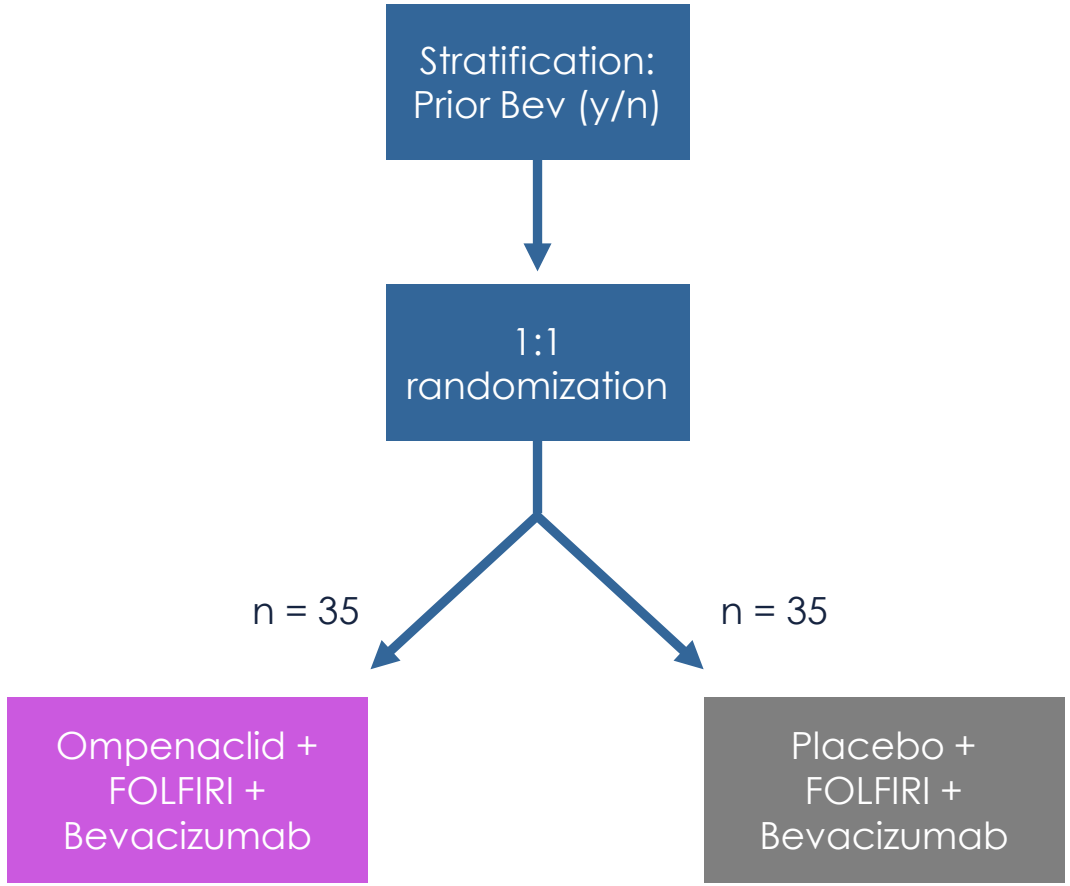

- Median (95% CI) rwPFS in second line settings
  - KRAS G12C cohort – 4.6 (2.6-5.5) months
  - KRAS non-G12C cohort – 4.7 (4.2-5.3) months
  - mCRC overall cohort – 5.6 (5.2-5.8) months

In Phase 1b/2, Ompenaclid demonstrates a meaningful clinical effect in 2L mCRC on top of standard of care of FOLFIRI + Bevacizumab

Fakih et al; Real-World Study of Characteristics and Treatment Outcomes Among Patients with KRAS p.G12C-Mutated or Other KRAS Mutated Metastatic Colorectal Cancer. The Oncologist, 2022, 27, 663–674

# Phase 2 – Ompenaclid in second line CRC

- Efficacy endpoints: ORR (primary), PFS (secondary)
- Assumptions: 70 randomized, 40 PFS events; a=0.05 1-sided; 80% power for ORR, 48% power for PFS
- Interim analysis of PFS once 30 PFS events are observed
- ORR MSD between groups >20% to yield 1-sided p<0.05
- PFS MSD HR<0.59 to yield 1-sided p<0.05



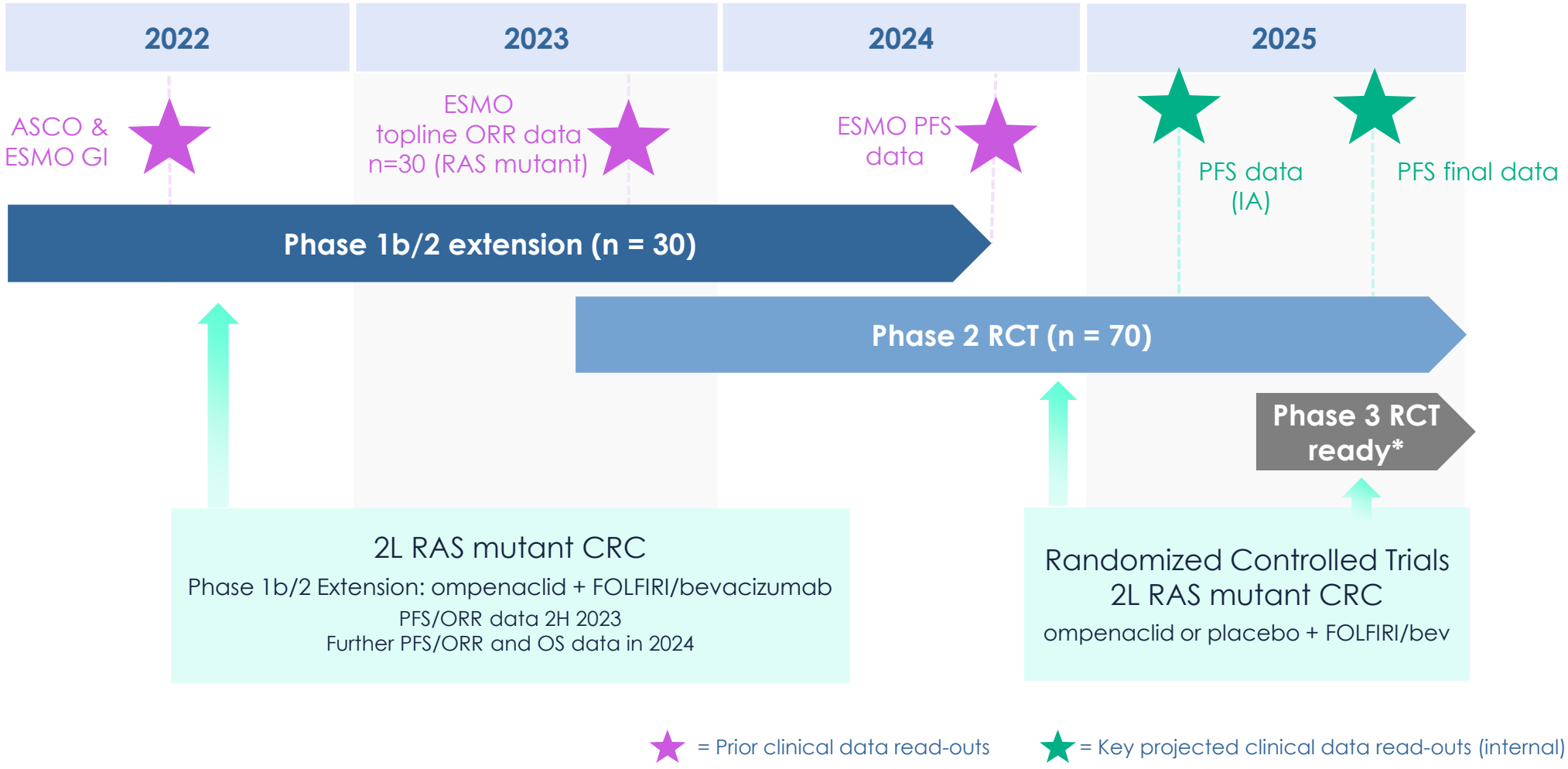

**ORR/PFS Assumptions:**

ORR = 35%
Median PFS = 10 months

**ORR/PFS Assumptions:**

ORR = 10%
Median PFS = 6 months

# Projected clinical data read-outs for ompenaclid




*Note: clinical trial shown in gray arrow requires additional funding and is dependent on further FDA feedback

# Licensing Agreement with Merck KGaA, Darmstadt, Germany

Announced in January 2024 to Accelerate the Global Development of Ompenaclid

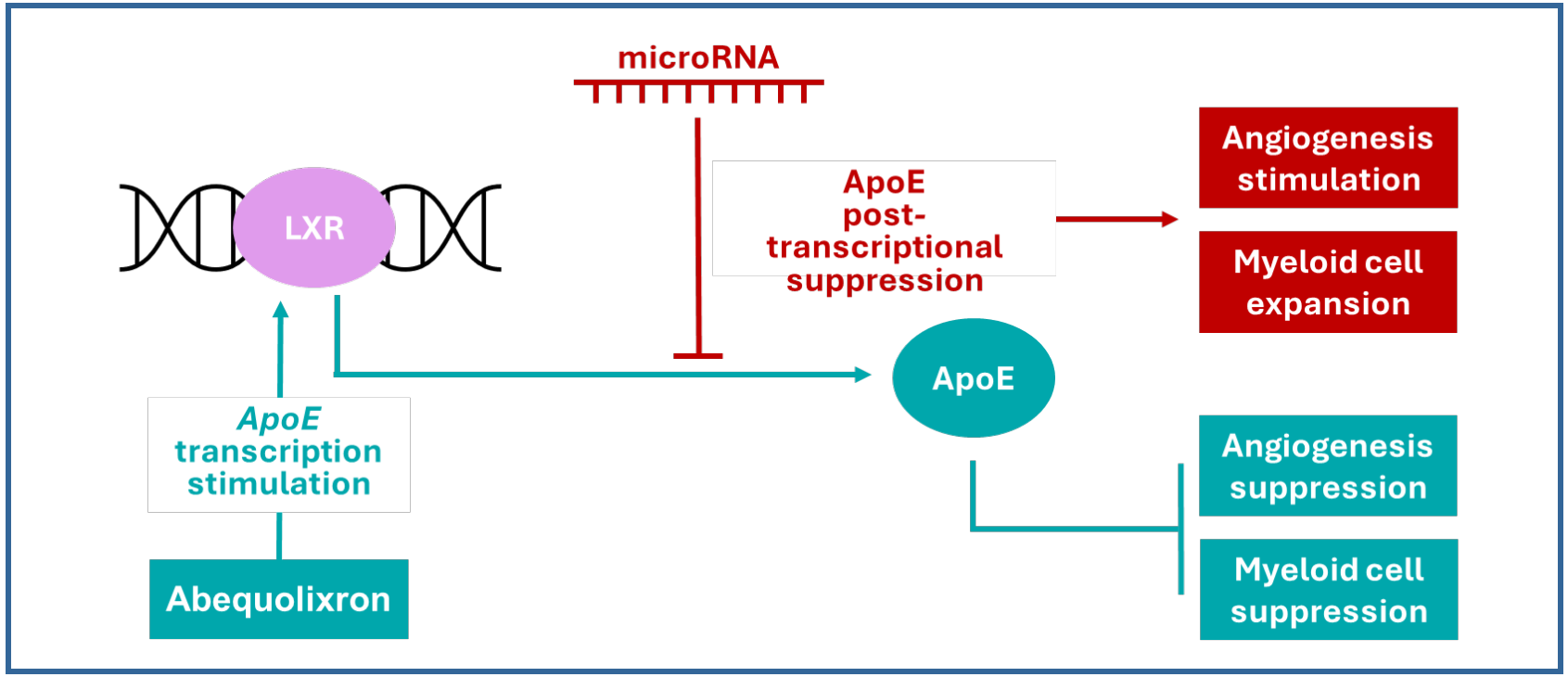
Inspirna Received $45M Upfront Payment

- **Merck KGaA, Darmstadt, Germany received exclusive license to ompenaclid outside of US**
  - Option to co-develop and co-promote ompenaclid in US
- **Further collaboration focused on follow-on compounds targeting SLC6a8**
  - Inspirna will lead pre-IND activities, and retain co-development/co-commercialization rights in US
- **Inspirna received $45M upfront payment**
  - Eligible to receive milestone payments with tiered royalty rates in low teens on net sales outside of US, upon achievement of certain development and sales milestones for ompenaclid
  - Eligible to receive development, regulatory and sales milestone payments for each follow-on compound targeting SLC6A8, along with up to double-digit royalties on net sales outside of US

# Abequolixron
(RGX-104)

- Potent small molecule LXR beta selective agonist that activates APOE to target immune-suppressive tumor myeloid cells, while also inhibiting tumor angiogenesis
- Monotherapy activity in lung cancer demonstrated during Phase 1
- Clinical activity in combination with docetaxel demonstrated in an ongoing Phase 1b/2 clinical trial in patients with non-small cell lung cancer and small-cell lung cancer
- Compound was originally discovered by GlaxoSmithKline for the treatment of cardiovascular disease and exclusively licensed to Inspirna
- Issued global patent protection to 2040

# LXR agonist-induced ApoE creates an anti-tumor microenvironment




- LXR agonism with abequolixron (RGX-104) increases ApoE production
- ApoE inhibits
  - Endothelial migration and angiogenesis
  - Myeloid cell expansion/survival

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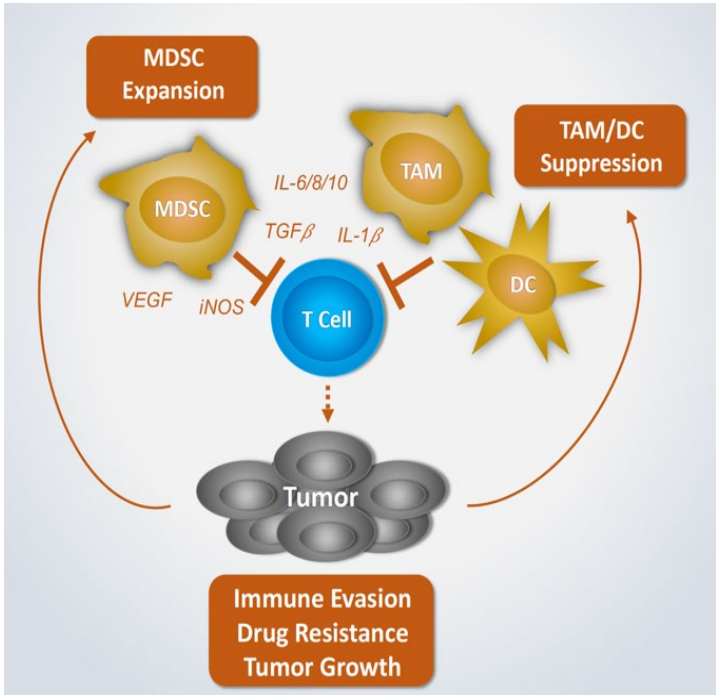
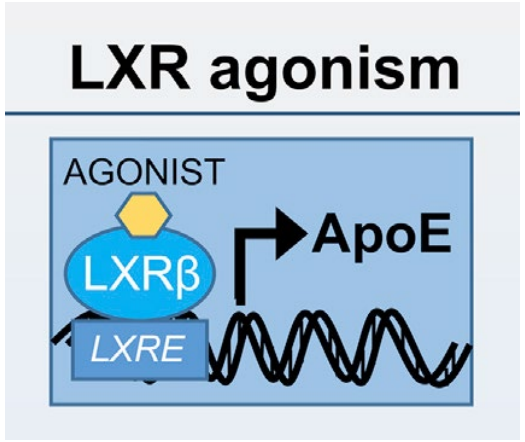
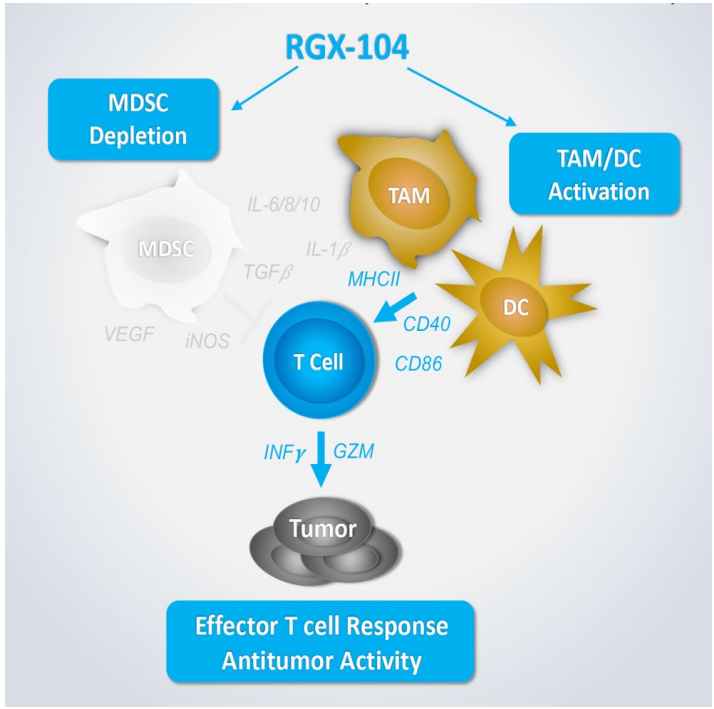
# RGX-104-induced ApoE expression modulates immune system to create an anti-tumor environment




**Pro-tumor environment**
MDSC expansion
TAM/DC suppression

RGX-104



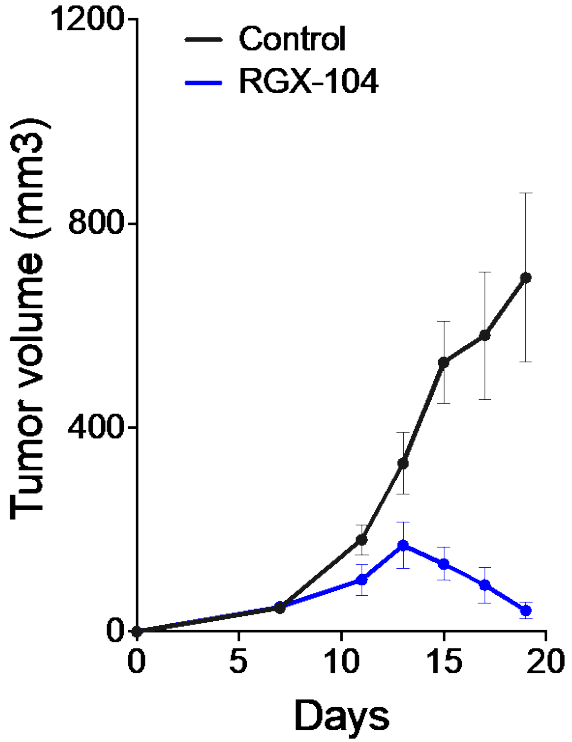




**Anti-tumor environment**
*MDSC suppression*
TAM/DC activation

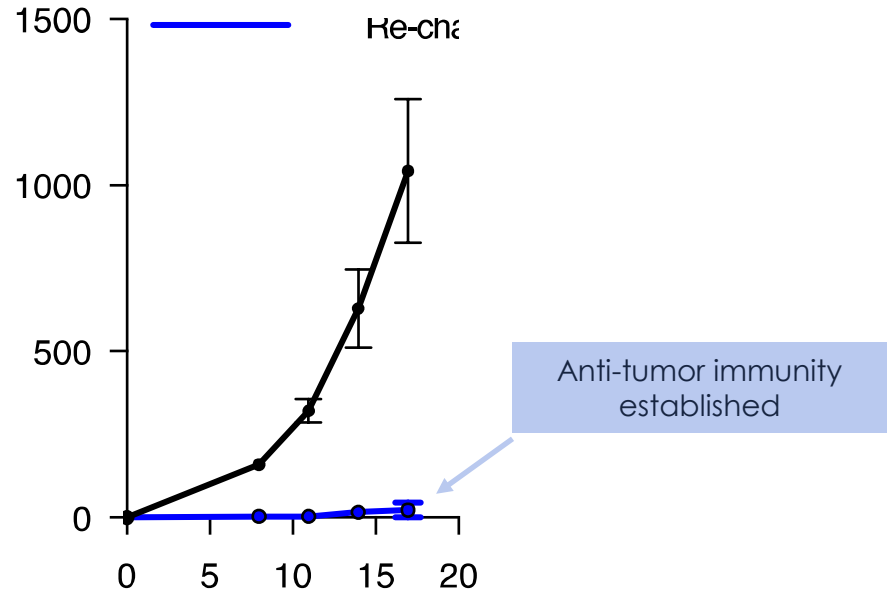
1) Mita M et al. ASCO (2018); 2) Mita M et al. AACR II (2020); 3) Tavazoie M et al. Cell. 2018 Feb 8;172(4):825-840; 4) Lim E et al. AACR I (2020)

# RGX-104 induces anti-tumor immunity in tumor models

## RGX-104 has demonstrated potent immune-modulating and anti-tumor activity in numerous preclinical tumor models spanning different tumor types[1-6]




Mice with complete tumor regressions on RGX-104 treatment were re-challenged with the same cancer cells and failed to grow tumors



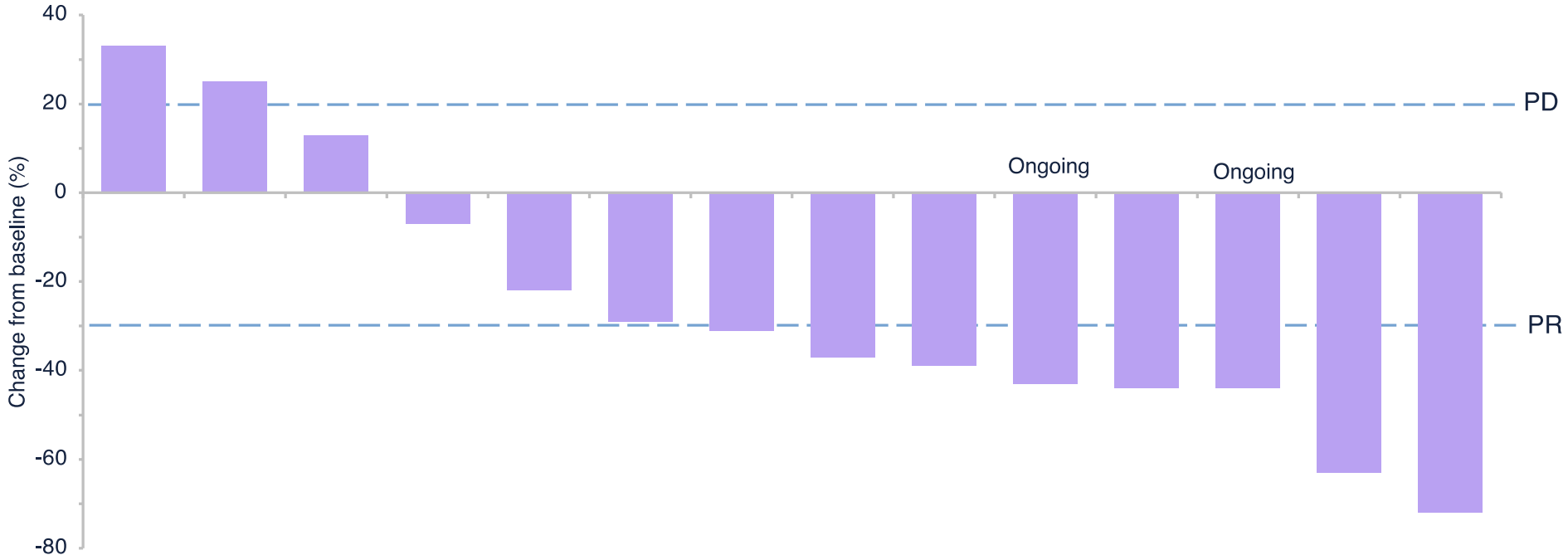

Anti-tumor immunity established

Syngeneic E0771-DR Breast Cancer model
Treatment initiated with RGX-104 (100mg/kg) on Day 3 (left figure)
No treatment administered in re-challenge experiment (right figure)

1) Tavazoie M et al. Cell. 2018 Feb 8;172(4):825-840, 2) Mita M et al. ASCO (2018), 3) Mita M et al. AACR II (2020), 4) Lim E et al. AACR I (2020), 5) Liang H. Biochem Biophys Res Commun. 2020 Jul 23;528(2):330-335, 6) Wan d et al. J Control Release. 2020 Jan 10;317:43-56

# 2/3L NSCLC abequolixron + docetaxel

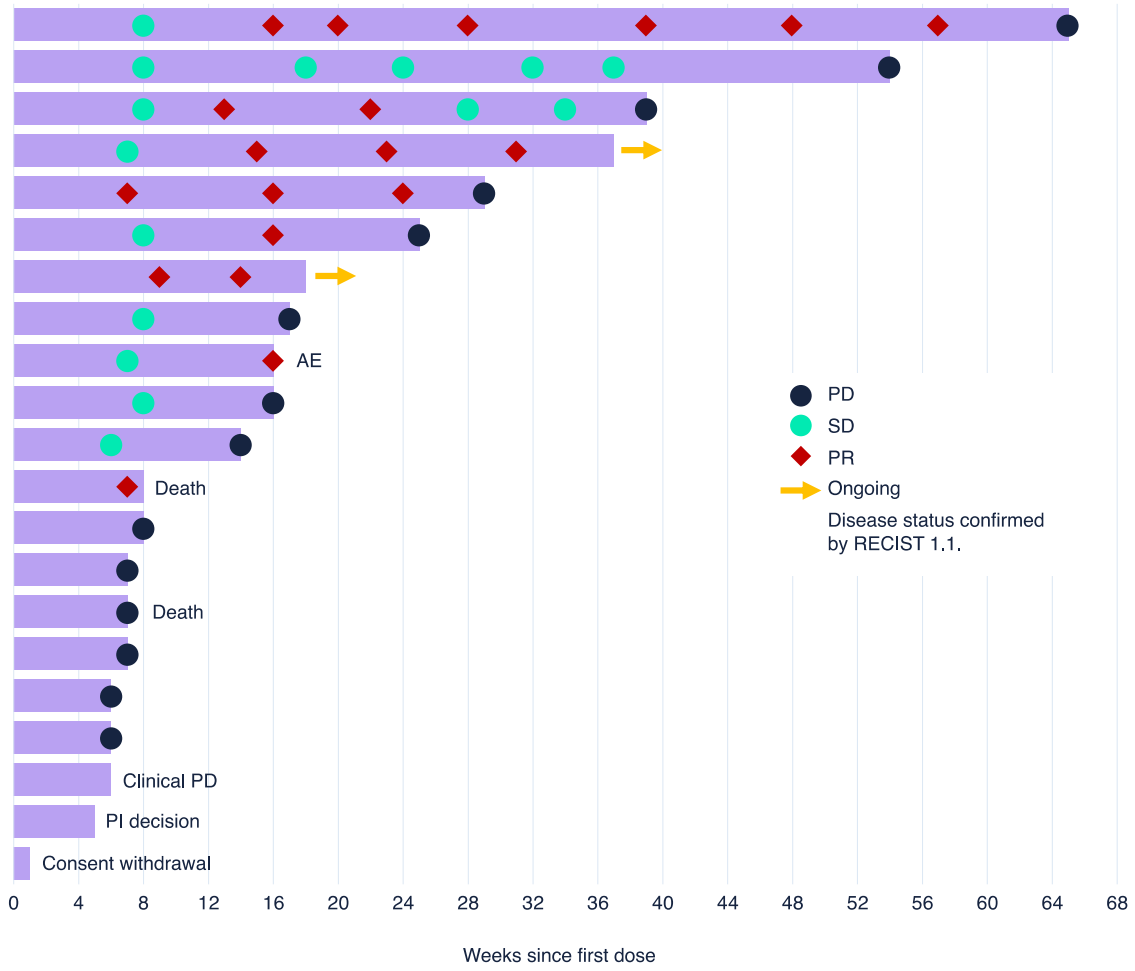
## Best response in all evaluable patients (n=15*)




*The ORR in the ITT population (n=21) was 38% and 53% in the evaluable population (n=15).

One patient (not shown) was evaluable but one of the target lesions was not assessed on the first on-treatment scan. However, the overall response was PD due to non-target PD.

# Duration of treatment response in all patients (n=21)




- Of the 21 subjects, 2 are ongoing, 14 had PD and 5 discontinued for reasons including AEs.
- The mDOR across the 8 patients with PR was 5.8 months.

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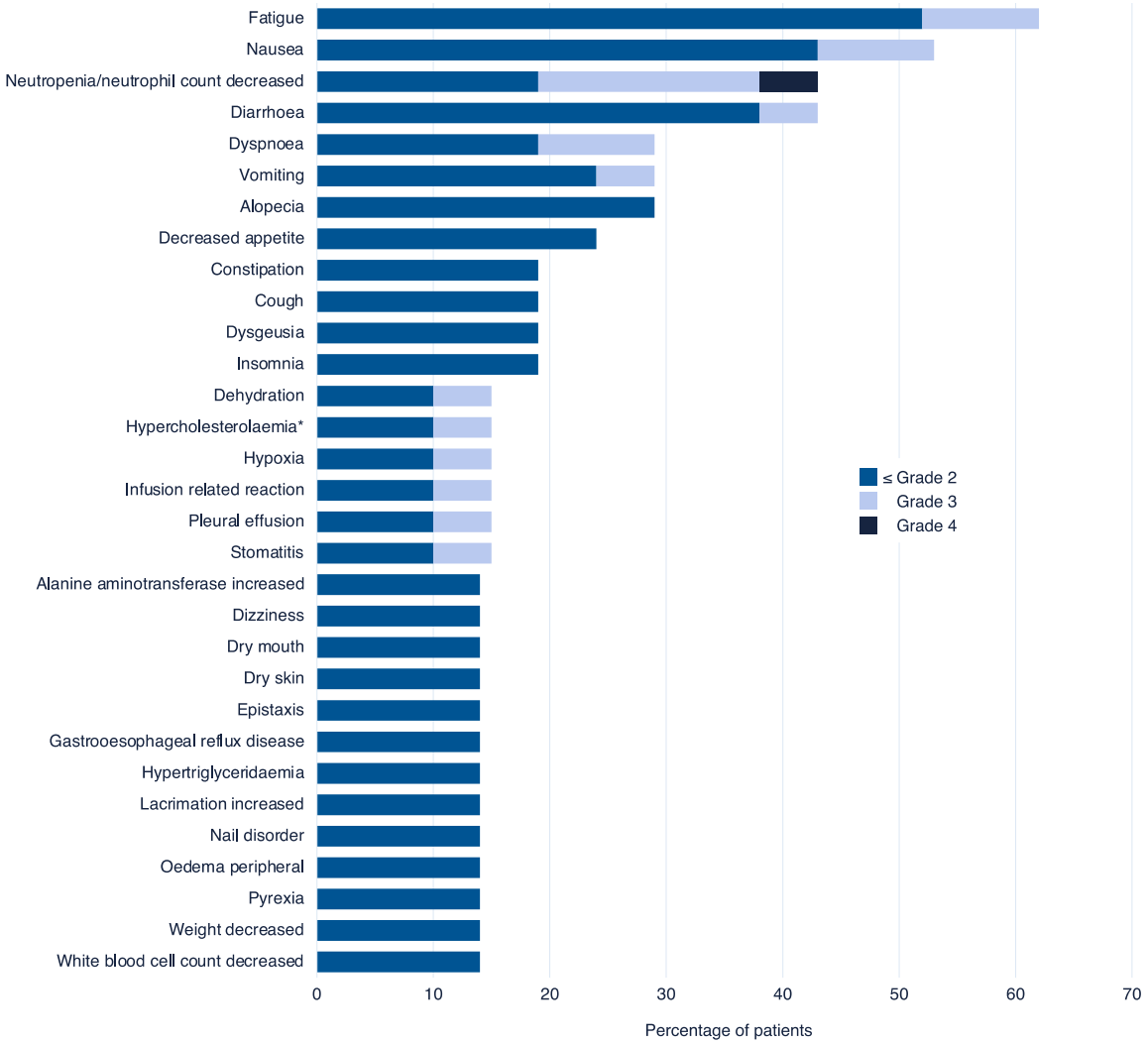
# TEAE in > 10% of all patients (n=21)




- The most common Grade ≤ 2 TEAEs were fatigue (52%), nausea (43%) and diarrhea (38%).
- The most frequent Grade ≥ 3 TEAEs were neutropenia (14%), fatigue (10%), nausea (10%) and dypsnea (10%).

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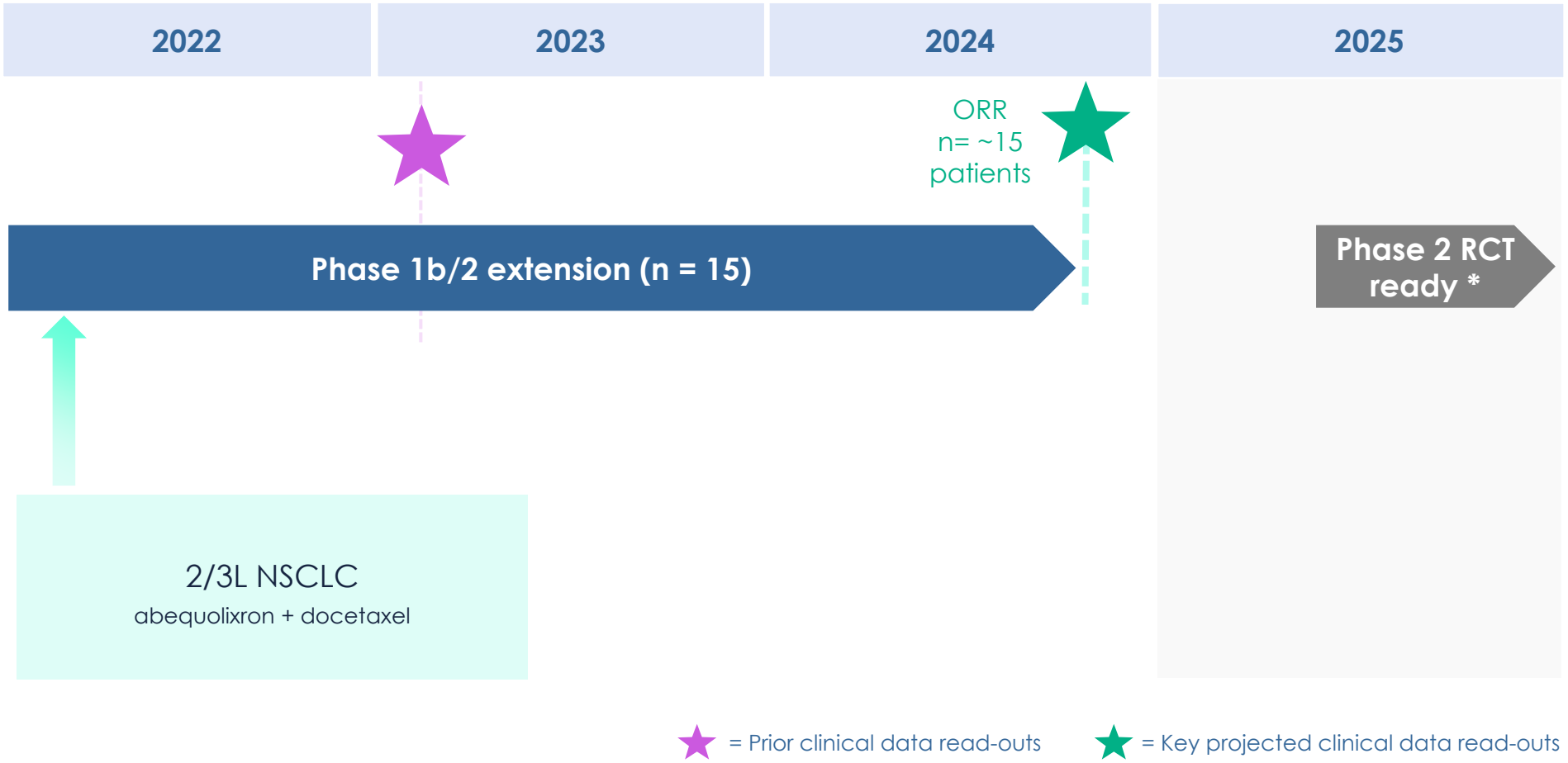
# Projected clinical data read-out for abequolixron




*Phase 2 depends on additional funding and FDA feedback

# Supported by Top-Tier Institutional Investors and Academic/Industry Collaborators

## Investors

SOFINNOVA PARTNERS

novo holdings

PARTNERSHIP FUND for New York City

SIXTY DEGREE CAPITAL

LEPU MEDICAL

SANDS CAPITAL

OCEANPINE

WuXi AppTec

Dreavent Life Sciences Ecosystems

ALEXANDRIA

VIVO CAPITAL

HealthVentures

Exor Seeds

## Collaborators

**Merck KGaA, Darmstadt, Germany**

SCIENTIA · PRO · BONO · HUMANI · GENERIS
The Rockefeller University
1901

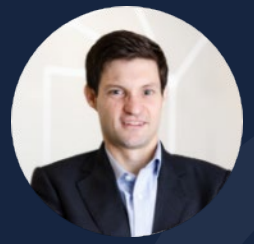
THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

# Management Team

Track record of advancing transformative science & innovative therapies

**Usman "Oz" Azam, MD**
Chief Executive Officer

Novartis; TMUNITY; Empyrean



**David M. Darst, MBA**
COO/ CFO & Co-Founder

Apellis; POTENTIA; OrbiMed

**Karim Benhadji, MD**
Chief Medical Officer

Taiho Oncology; Eli Lilly

**Anne Assmus, PhD**
Head of Business Development

Affimed; Morphosys

**Nayomi Thomas**
Associate Director of HR & Legal

JP Morgan Chase; Viagogo

**Darren Wong, PhD**
SVP of Translational Medicine

Exarta Therapeutics; Concert Pharmaceuticals; Medivation

**Steve Wald, MS**
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Epizyme; Sepracor

**Kimberly Hoffman**
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MERCK; Pfizer; Northern Biologics

**Isabel Kurth, PhD**
SVP, Research

ISREC Foundation Recherche Cancer; The Rockefeller University

**Osamu Takahashi, MD, PhD**
VP, Clinical Development

Taiho Oncology; Gilead Sciences; AstraZeneca

**Corey Sohmer, MBA**
VP, Finance & Accounting

Actinium Pharmaceuticals; Cyclacel

# Advisors and Board of Directors

Deep experience in oncology drug development

## Scientific and Clinical Advisors

**Masoud Tavazoie, MD, PhD**
(Inspirna co-founder)

- Former Inspirna CEO

**Sohail Tavazoie, MD, PhD**
(Inspirna co-founder)

- Professor & Director of Metastasis Center, Rockefeller University; oncologist, MSKCC
- President, American Society of Clinical Investigation

**Jean-Pierre Bizzari, MD**

- Former Executive VP & Global Head of Oncology, Celgene
- Current Board Director, Compugen, Transgene, Halozyme

**Josep Tabernero, MD, PhD**

- Head of Medical Oncology at Vall d'Hebron University Hospital
- Former President of ESMO

**Alan Venook, MD**

- Head of GI Oncology Program, UCSF

**Hossein Borghaei, DO, MS**

- Head of Thoracic Oncology, Fox Chase Cancer Center

**Yelena Janjigian, MD**

- Chief, GI Oncology Division, MSKCC

**Antoni Ribas, MD PhD**

- Professor, UCLA; Director of Tumor Immunology

**Siavash Kurdistani, MD**

- Chair of Biological Chemistry, UCLA; Associate Director, Gene Regulation

## Board of Directors

**Dieter Weinand, MS (Chairman)**

- Former CEO, Pharmaceuticals Division at Bayer AG
- Current Chairman of Replimune, Mnemo

**Usman "Oz" Azam, MD**

- CEO, Inspirna

**Peter Van Vlasselaer, PhD**

- Former CEO of ARMO, iPierian, Arresto, Avidia

**Eric Rowinsky, MD**

- Director of Biogen; Former CMO, ImClone Systems

**Antoine Papiernik, MBA (investor)**

- Managing Director, Sofinnova Partners

**Raymond Camahort, PhD (investor)**

- Partner, Novo Ventures

**Michael Ginder, CFA (investor)**

- Research Analyst, Sands Capital

**Priscilla Sugianto, MD (investor)**

- Principal, Vivo Capital

**Jue Pu, MS (investor)**

- Director of Investments, Lepu Medical

# Summary

- Inspirna is a clinical stage biotechnology company headquartered in New York City that is developing first-in-class novel therapeutics to treat cancer types of high-unmet medical need
- Our miRNA-DRIVEr* platform has delivered novel targets that are now being evaluated in tumor types that present significant commercial opportunities with multiple clinical data catalysts in the near future.
  - Lead candidate ompenaclid is a novel small molecule that inhibits SLC6A8 with activity in patients with RAS mutant colorectal cancer, with Phase 2 randomized data in 1H 2025, and Phase 3 ready by end of 2025
  - Second program abequolixron is a potent small molecule LXR beta selective agonist that activates APOE currently in Phase 1b/2 development for lung cancer
- Led by an experienced management team, supported by top-tier institutional investors, collaborators and advisors



* **miRNA-DRIVEr** - **miRNA** DYSREGULATED **DR**UG TARGET *I*N *VI*VO **E**LUCIDATION