Inspirna

Corporate Overview

January 2024

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 currently in Phase 2 development
- Second program abequolixron is a small molecule LXR agonist in Phase 1b/2 development for lung and endometrial 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

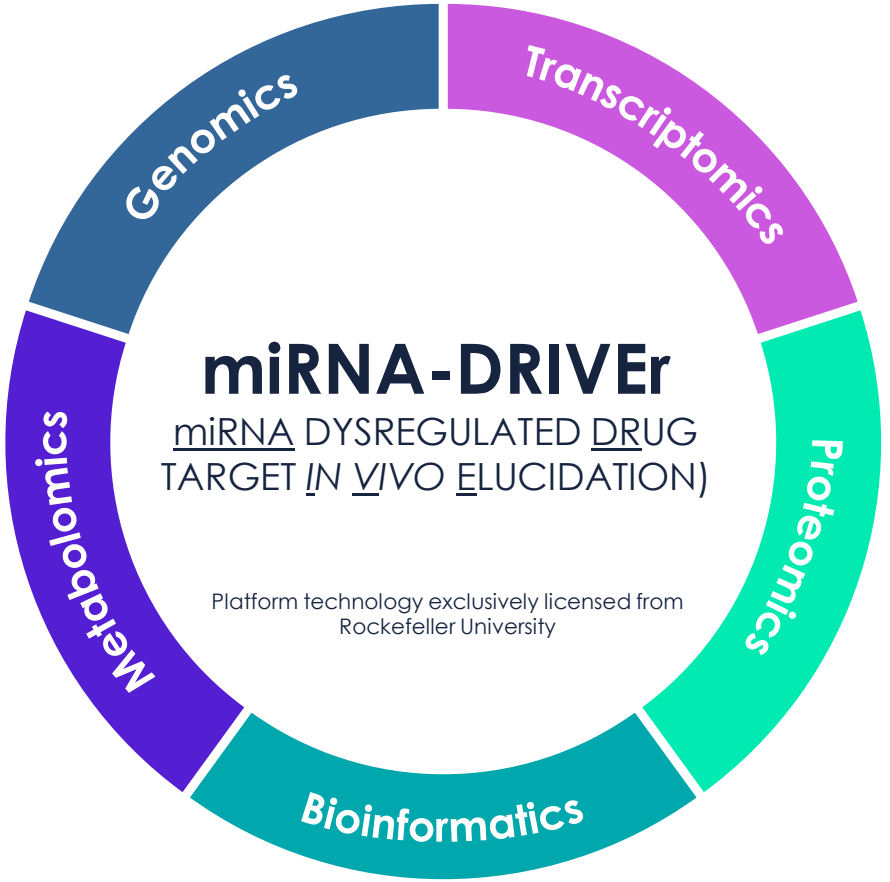
SLC6A8 -Solute Carrier family 6 member 8, LXR- Liver X Receptor, miRNA DRIVEr- miRNA DYSREGULATED DRUG TARGET *IN VIVO* ELUCIDATION

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?

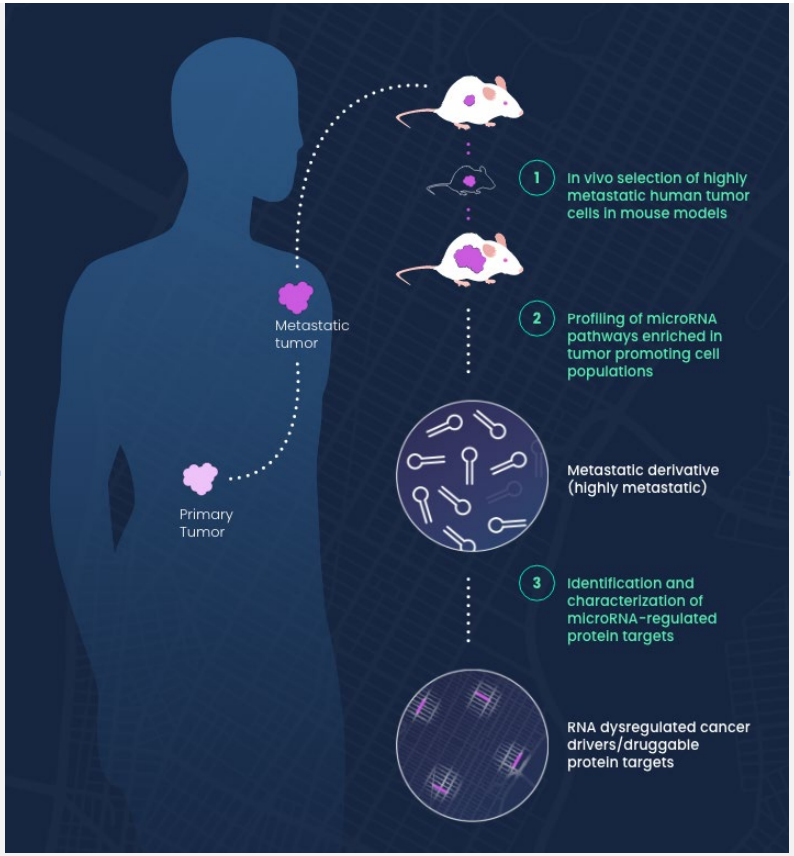
- Micro RNAs (miRNAs)* have important functions in controlling many cell properties:
  - Regulate gene expression at the post-transcriptional level;
  - Modulate cell growth and differentiation;
  - Regulate pathways and networks via coordinated activities;
  - Act as control nodes or hubs in regulatory 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.



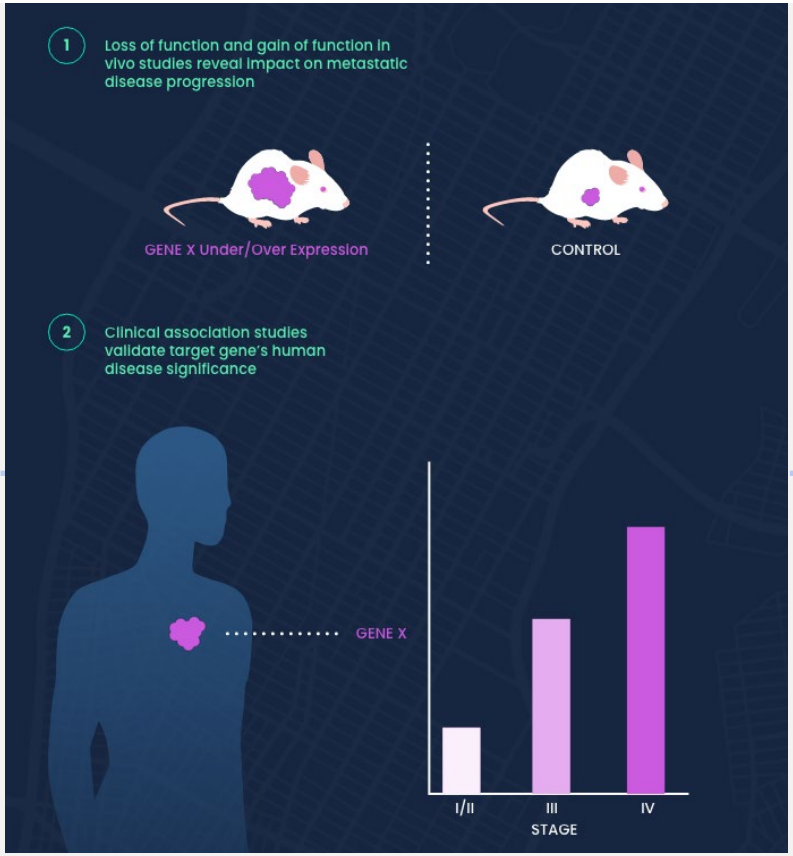
*short, endogenous noncoding RNAs 19–22 nucleotides long

Overview - miRNA-DRIVEr platform
miRNA DYSREGULATED DRUG TARGET *IN VIVO* ELUCIDATION)

RNA-DRIVEr platform reveals actionable RNA-dysregulated cancer targets



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

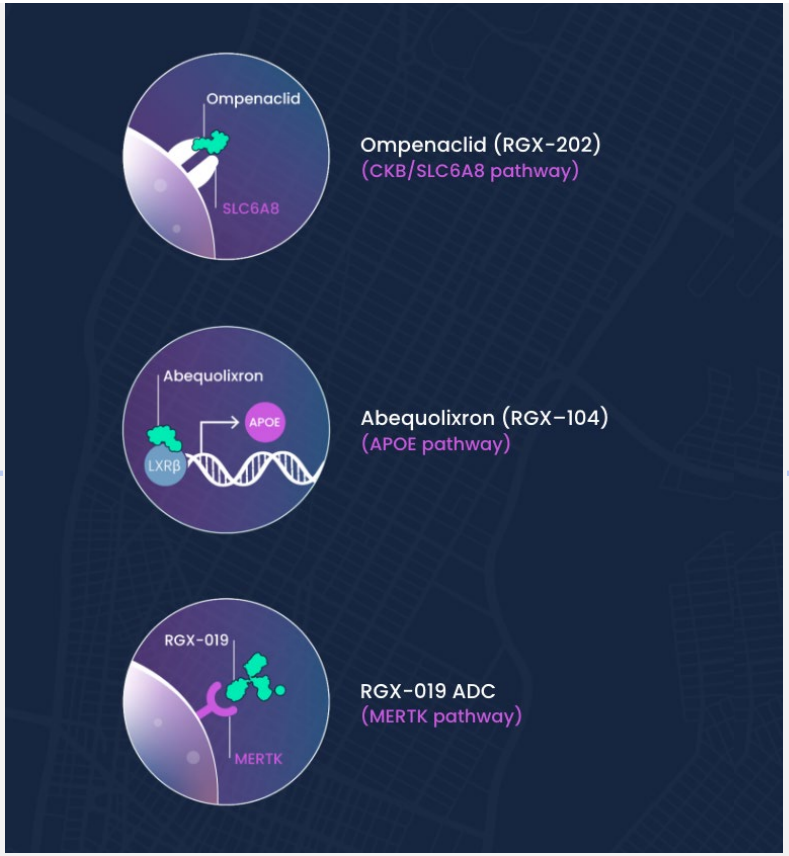


We test and **validate** the role of the discovered gene in driving metastatic disease.

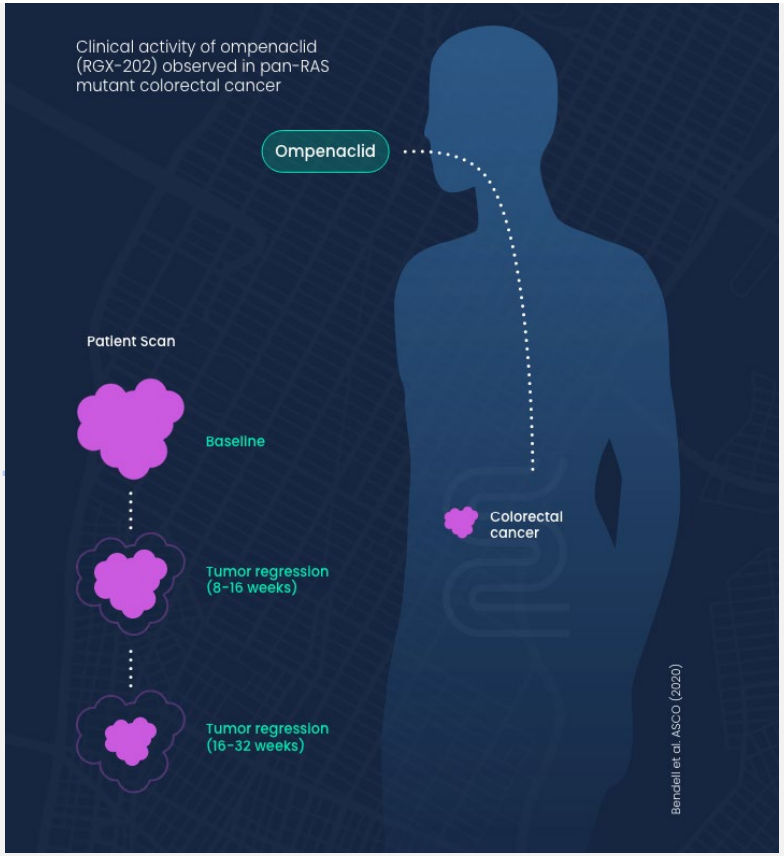
Tavazoie et al. Cell. 2018 Feb 8;172(4):825-840, 2. Loo JM et al. Cell. 2015 Jan 29;160(3):393-406, 3. Pencheva N et al. Cell. 2014 Feb 27;156(5):986-1001, 4. Pencheva N et al. Nat Cell Biol. 2013 Jun;15(6):546-54, 5. Pencheva N et al. Cell. 2012 Nov 21;151(5):1068-82, 6. Png KJ et al. Nature. 2011 Dec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

Overview - miRNA-DRIVEr platform cont…
First-in-class targets discovered and developed

RNA-DRIVEr platform reveals actionable RNA-dysregulated cancer targets (cont'd)



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

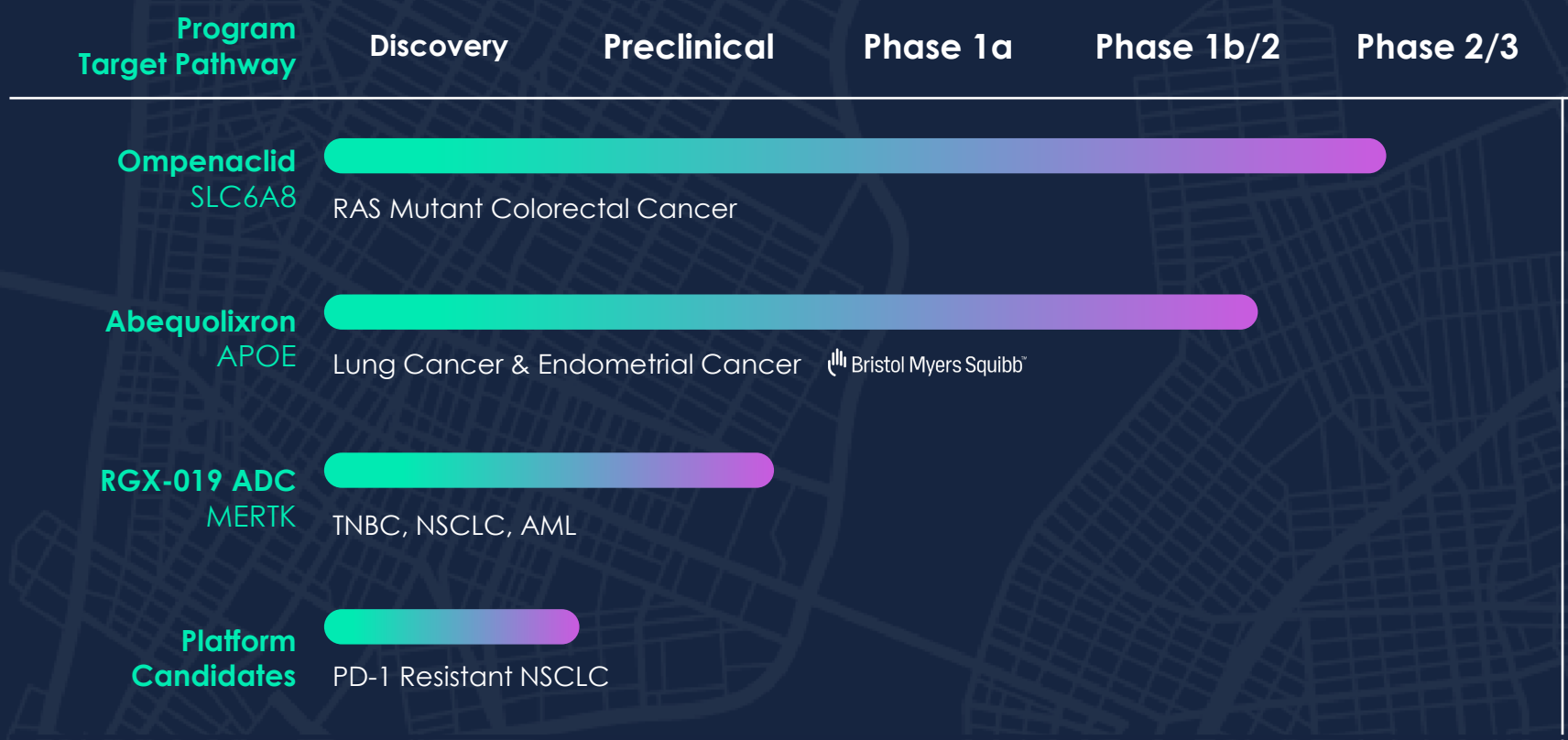


We then transform cancer medicine by leveraging RNA biology driven insights.

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

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

Inspirna's First-in-Class Oncology Pipeline



Key Anticipated Milestones

- Phase 2 RCT interim data in 1H 2025
- Phase 1b/2 follow up data in 2H 2024

- Phase 1b/2 data read-out in 2H 2024

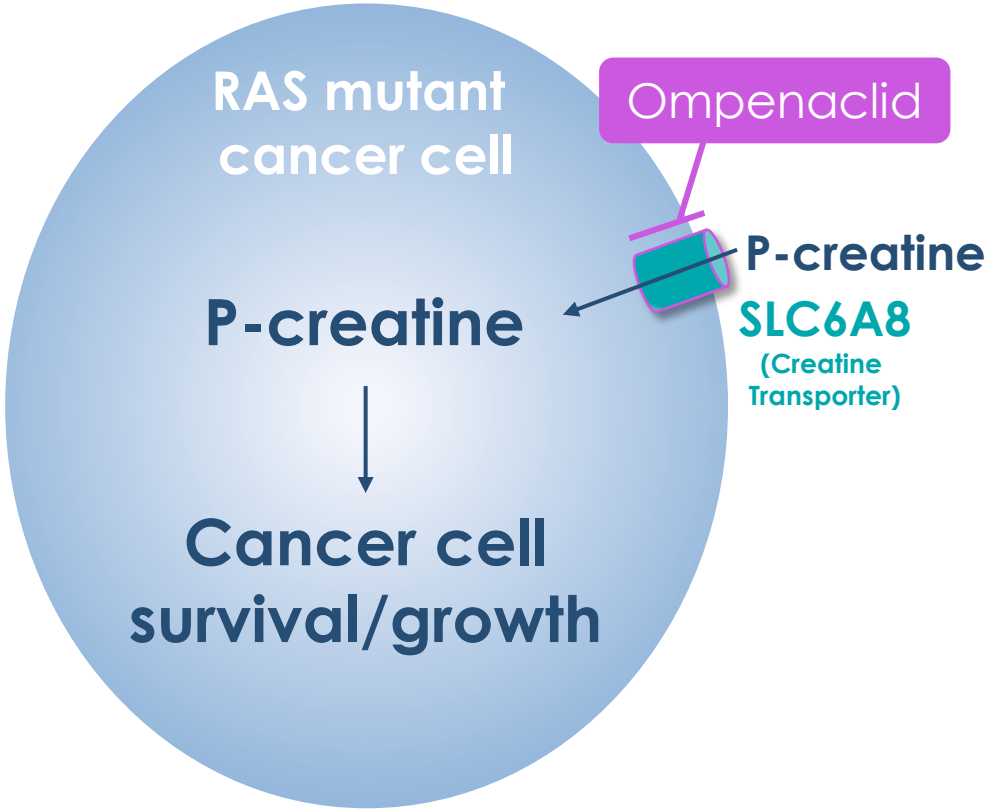
- IND enabling studies to initiate in 2024

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
- Initiated a Phase 2 double blinded randomized controlled trial
- Phase 3 preparation activities underway to enable a global pivotal trial
- Issued global patent protection to 2036 (2040+ if new patents issued)

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

Ompenaclid mechanism of action



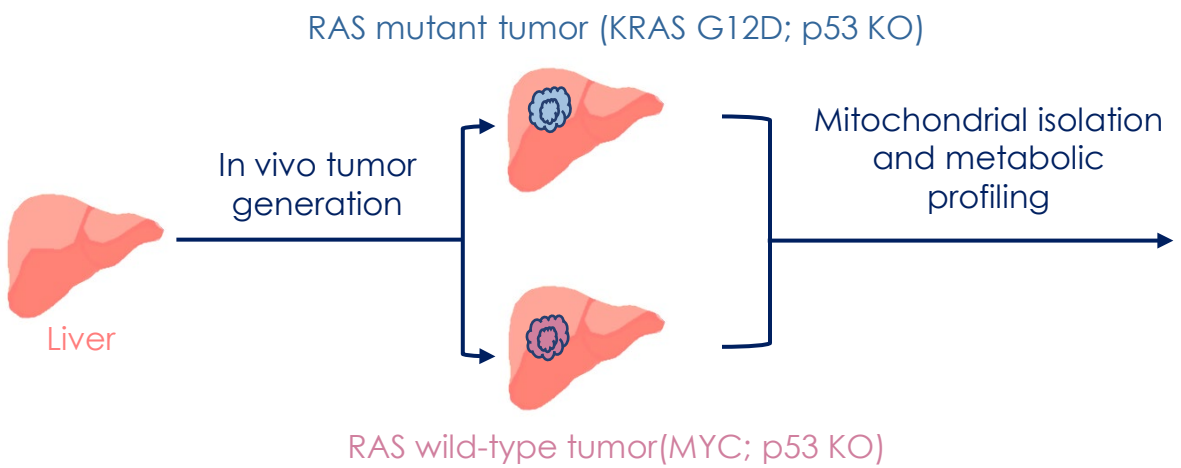
- Ompenaclid is an oral small-molecule competitive inhibitor of the SLC6A8 creatine transporter
- Blockade of SLC6A8 by ompenaclid depletes creatine and phospho-creatine in RAS mutant cancer cells, resulting in impairment of ATP synthesis and induction of apoptotic cancer cell death

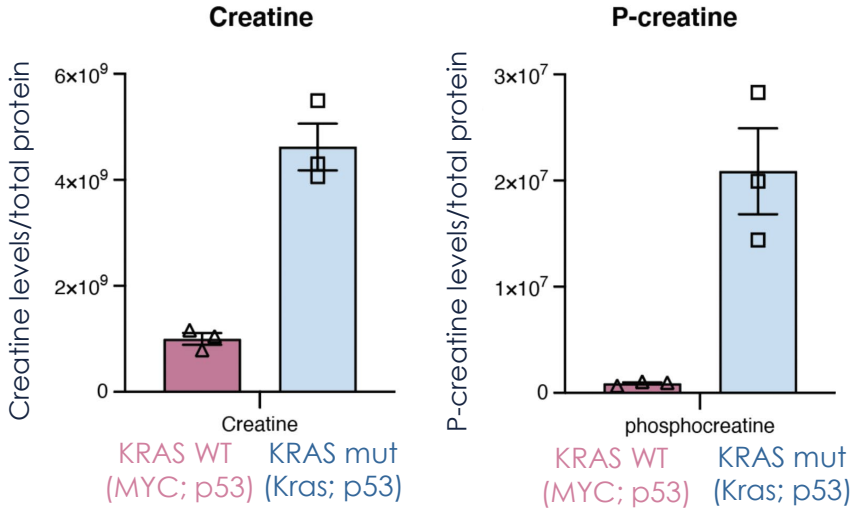
Sources: 1) Kurth et al. *Sci Adv*. 2021 Oct 8;7(41); 2) Loo et al. *Cell*. 2015 Jan 29;160(3):393-406; 3) Bendell et al. ASCO (2020); 4) Kurth et al. AACR (2018)

* 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

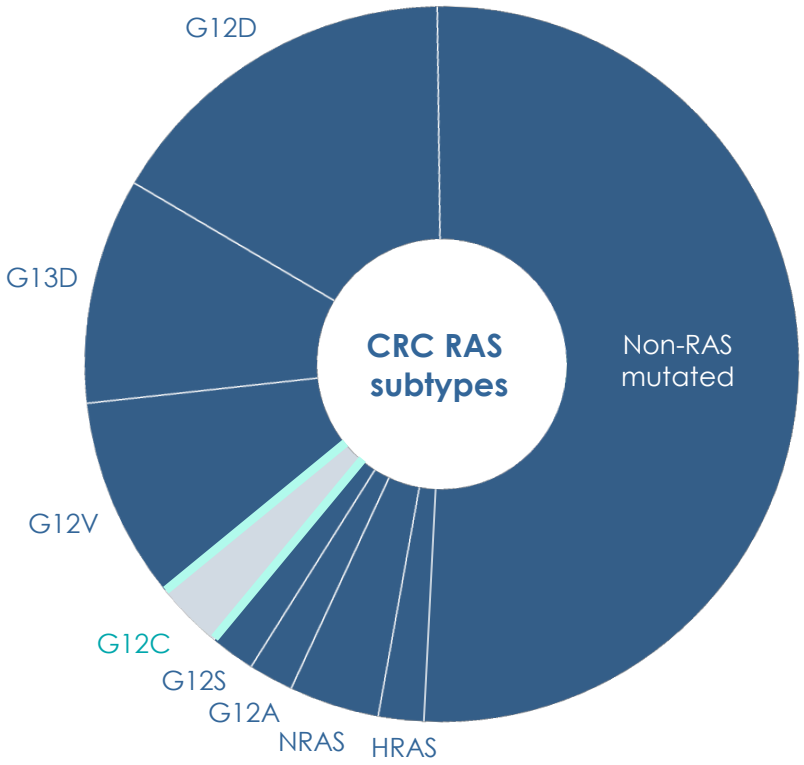
RAS-mutated colorectal cancer (CRC) remains a high-unmet medical need requiring a novel targeted therapy

- 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

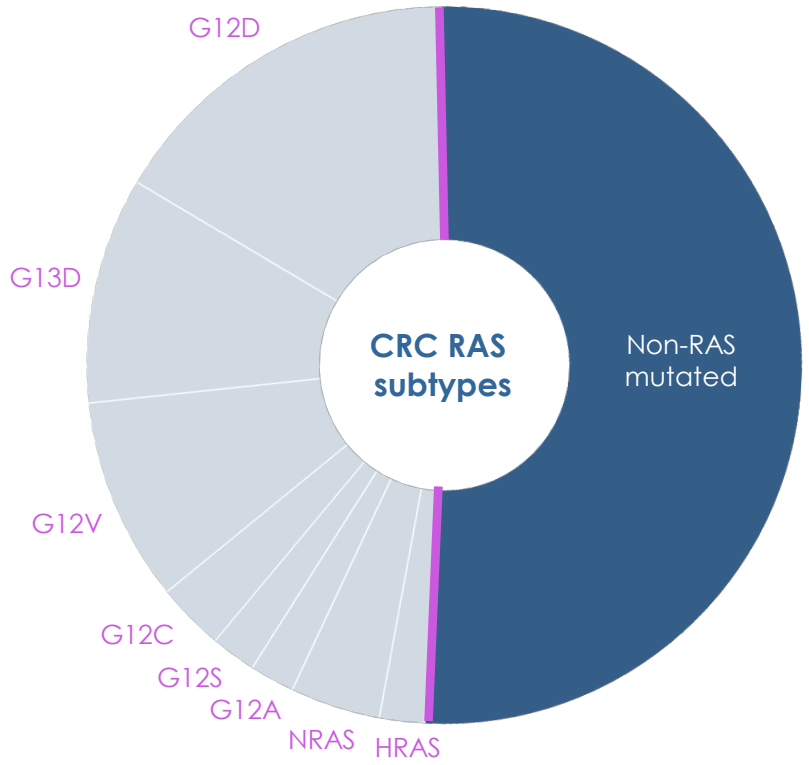
Ompenaclid is a first-in-class clinical stage SLC6A8 inhibitor with activity in pan-RAS-mutated CRC

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

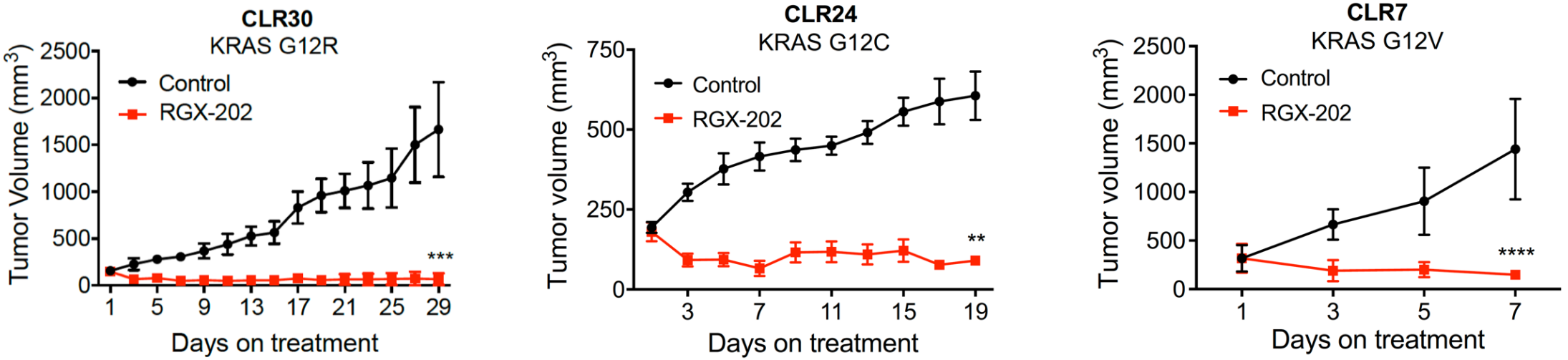
Potentially addressable CRC patient population
(ompenaclid)



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]

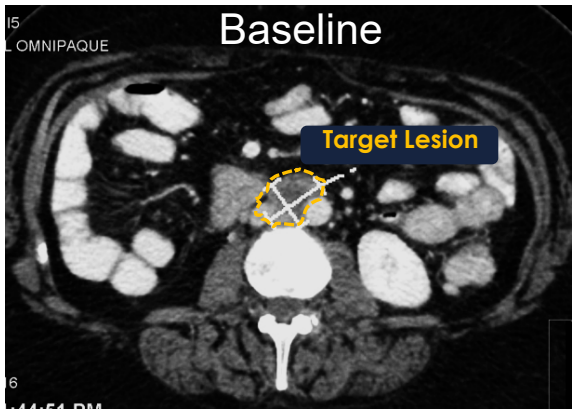
[1]Mayer et al. *N Engl J Med* 2015; 372:1909-1919;
[2]Grothey et al. *Lancet*. 2013;381:303–312

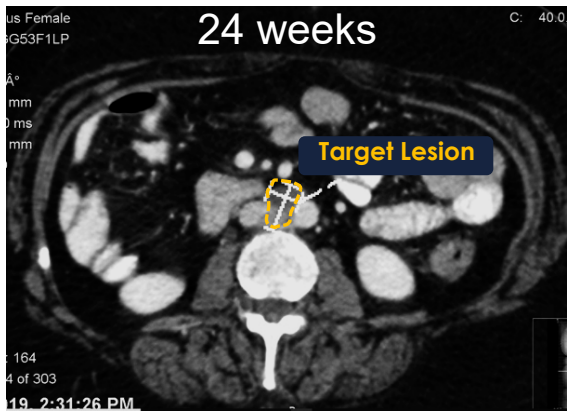
- Safety demonstrated
- Anti-tumor activity observed in KRAS mutant CRC at doses >2400mg BID
- Moved program rapidly into combination 2L setting on strength of preclinical and initial phase 1 monotherapy data

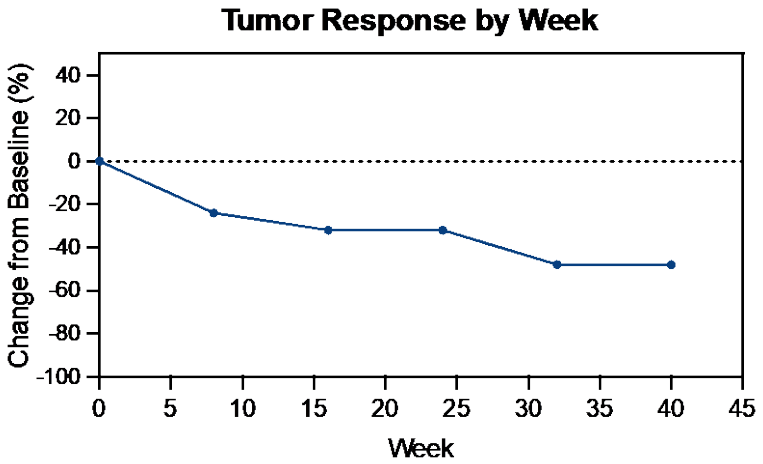
Ompenaclid single agent clinical activity – Phase 1 data presented at ASCO 2020

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

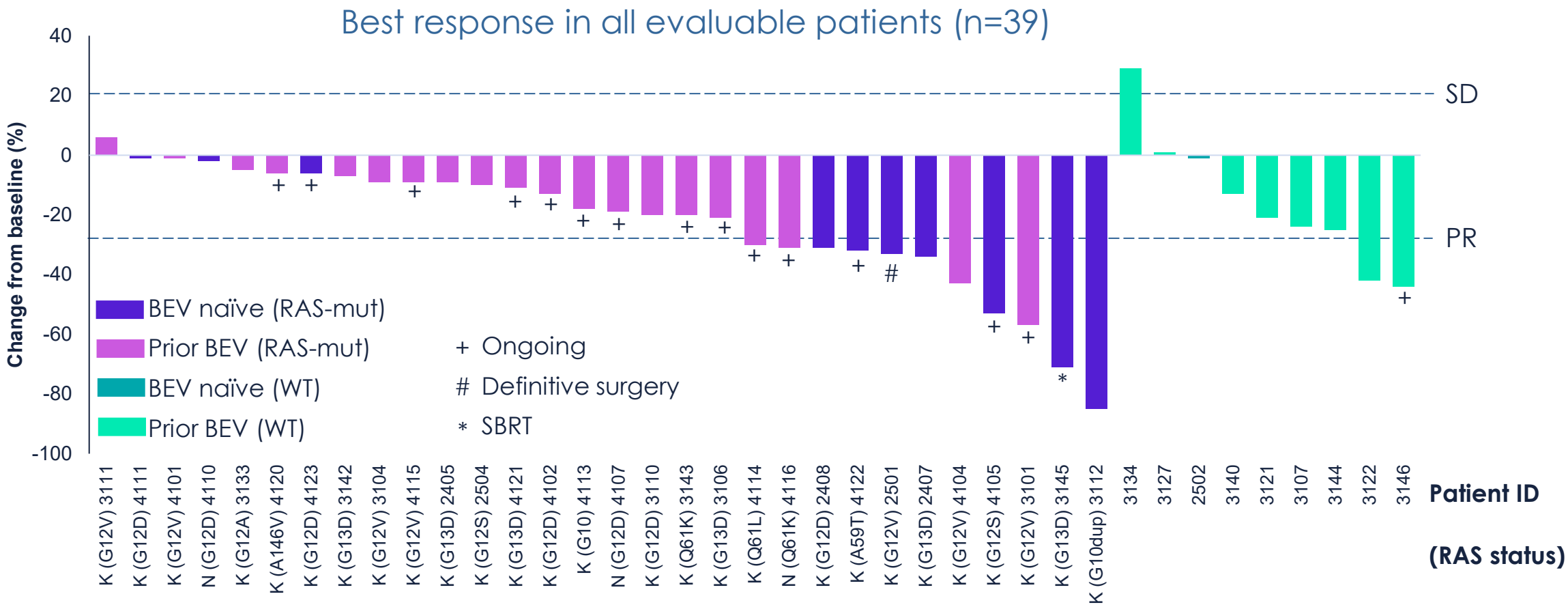
- The most commonly used standard-of-care (SOC) regimen for 2L metastatic CRC is FOLFIRI* + bevacizumab
- The benchmark efficacy data for this regimen in published clinical studies[1-5] are:
  - **Response Rate (ORR): ~15%**
  - **Median PFS: ~6 months**
- Patients with KRAS mutant tumors trend towards 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 – ESMO (October 2023)

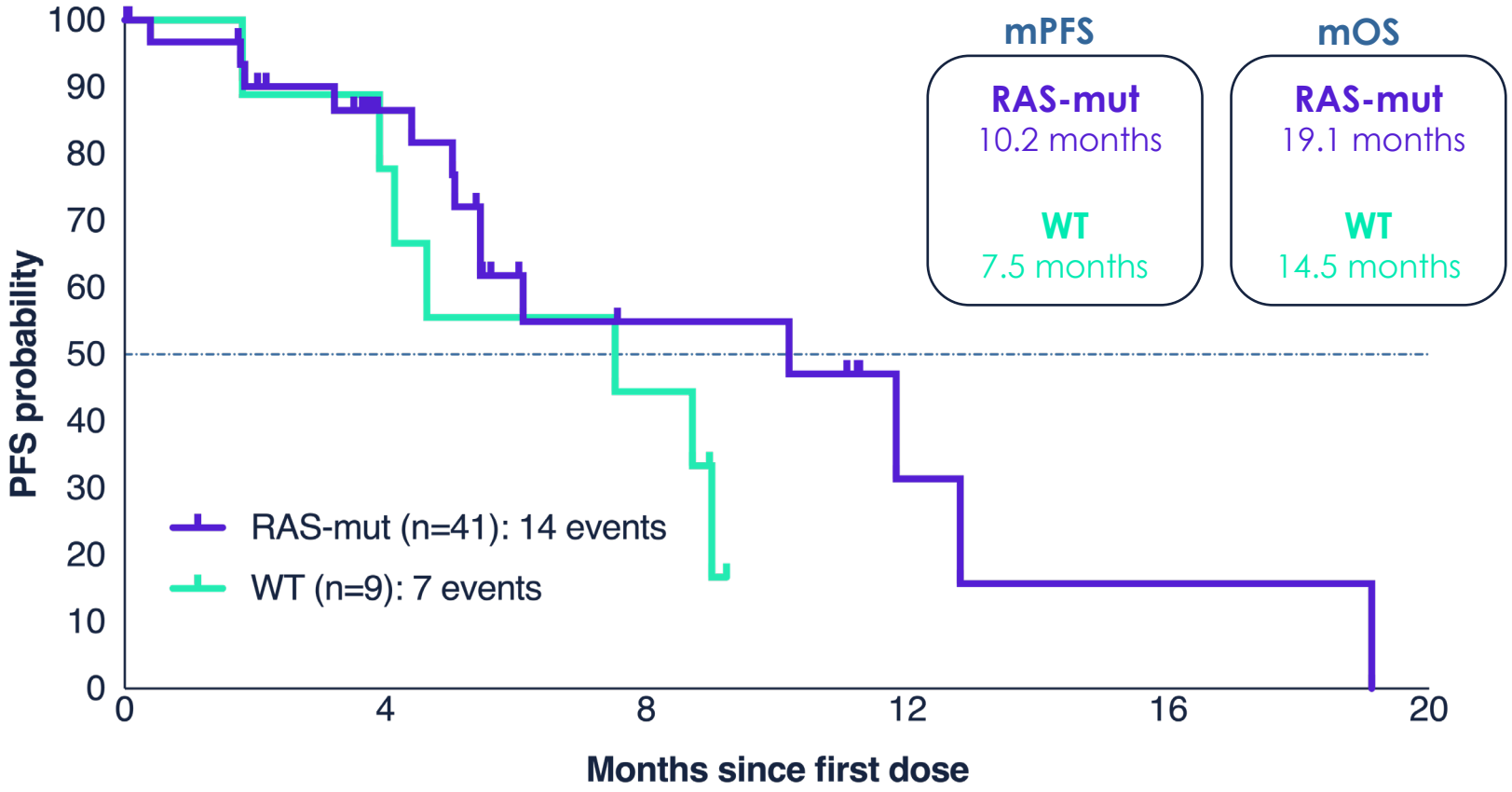
37% ORR, 100% DCR observed in RAS mutant patients



Percentage change from baseline of tumors in evaluable patients. Graph shows patient RAS status (N=NRAS, K=KRAS) and best overall response after ompenaclid + FOLFIRI/BEV treatment. Data cut-off 18 Sep 2023; open database.

PFS in RAS-mut vs WT mCRC – ESMO 2023

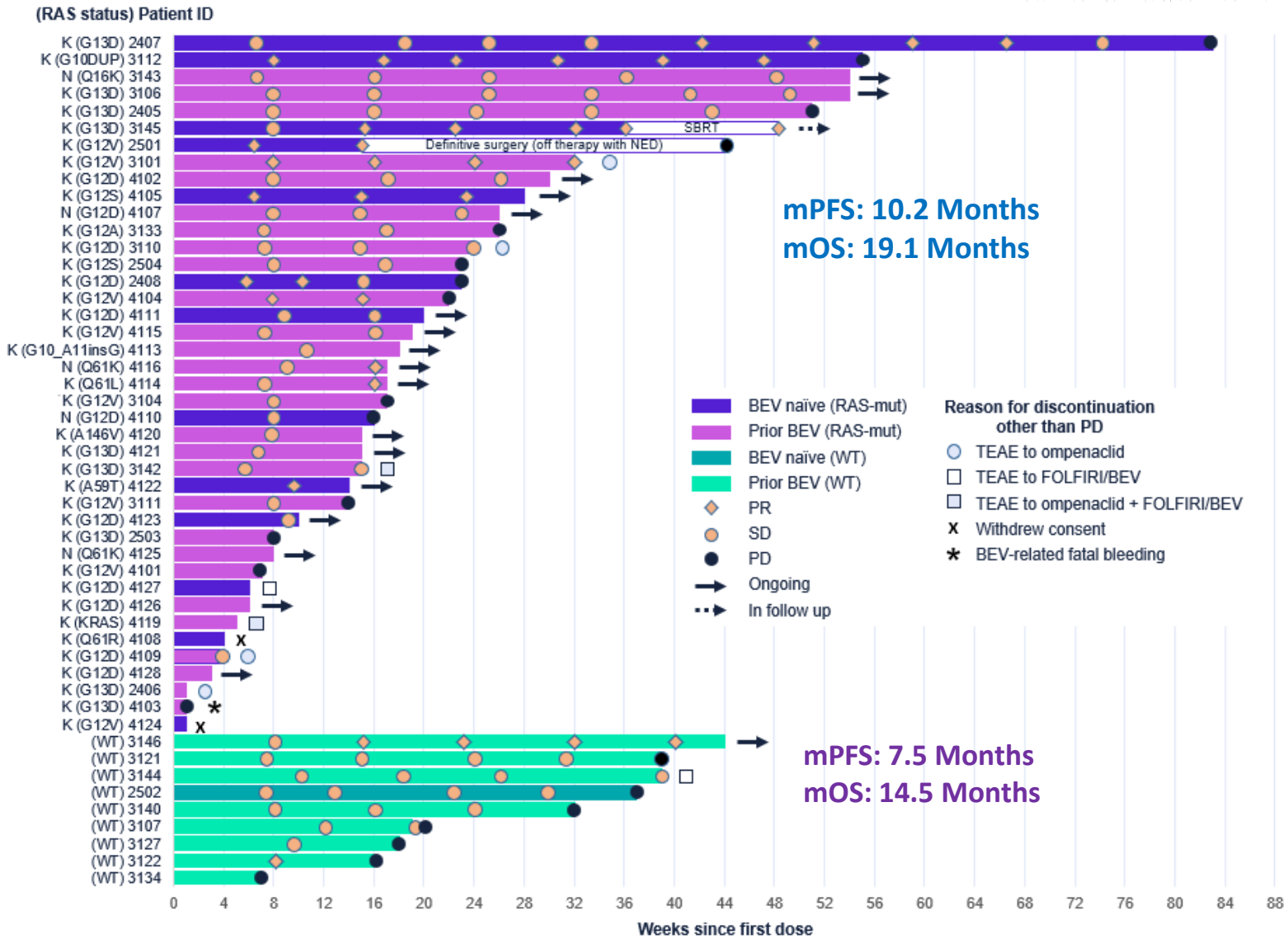
Ompenaclid + SOC increased PFS and OS in RAS-mut vs WT mCRC.



PFS analysis included all patients enrolled (n=50). PFS data are not yet final as of the cut-off date 18 Sep 2023 due to continuing patient follow-up and the limited number of PD events.

Duration of treatment and response in all patients (n=50) – ESMO 2023

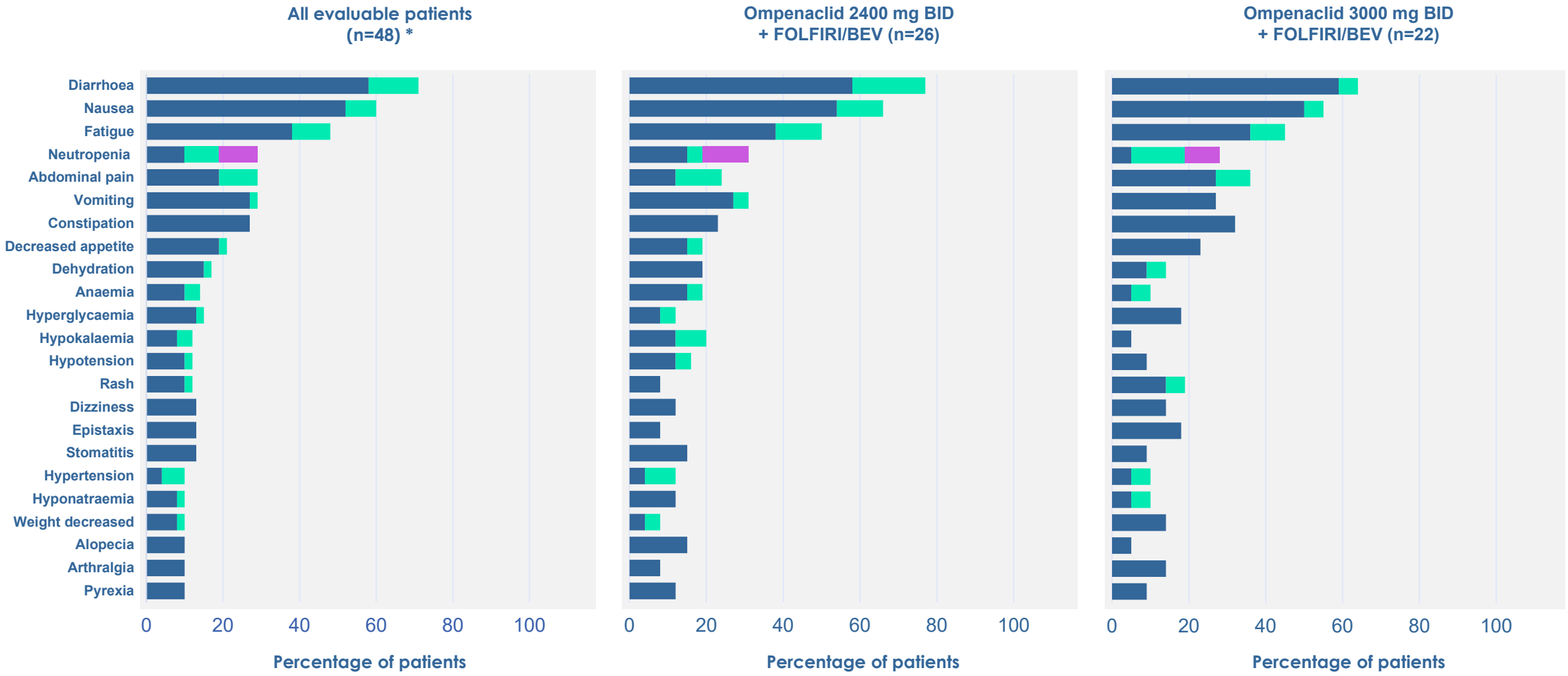
Graph shows patient RAS status (N=NRAS, K=KRAS).
Data cut-off 18 Sep 2023; open database, data subject to change.



Safety summary – ompenaclid + FOLFIRI/ bevacizumab (ESMO 2023)

- 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 (58%) and nausea (52%).
- The most frequent Grade ≥ 3 TEAEs were neutropenia (18%), diarrhea (13%), fatigue (10%) and abdominal pain (10%).
- The only Grade 5 TEAE was 1 patient (2% of total patients) with an intestinal perforation, deemed related to BEV.
- At the evaluated dose levels, ompenaclid added to FOLFIRI/BEV was well tolerated.

Ompenaclid Safety – TEAEs occurring in ≥ 10% of patients (ESMO 2023)



* Includes all patients who have discontinued study treatment and patients with study treatment ongoing who have completed ≥ 1 cycle with AE data entered. Data cut-off 18 Sep 2023.

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)	37% (RAS only)	13%	19%	5%
DCR (WT and RAS)	100% (RAS only)	74%	85%	88%
mPFS (RAS)	**10.2 months**	**5.7 months**	**Unreported**	**5.5 months**
mOS (RAS)	**19.1 months**	**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

^ESMO 2023, Hendifar et al.

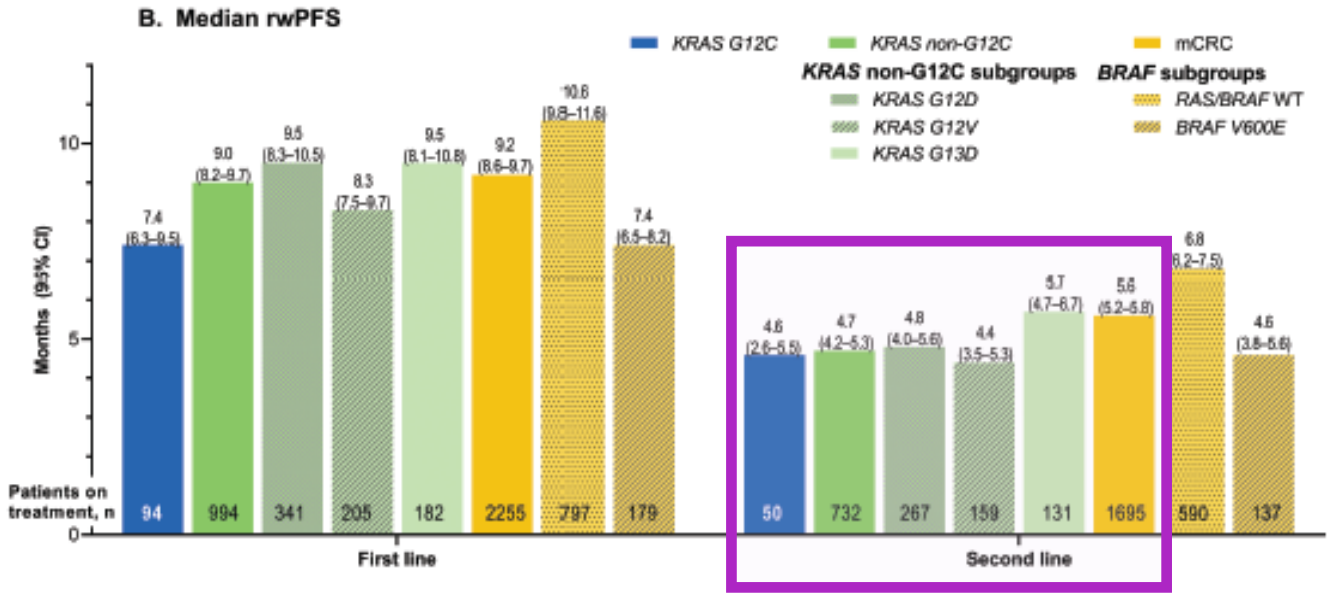
*RAISE Trial: Tabernero et al. *Lancet Oncol*. 2015 May;16(5):499-508
**VELOUR Trial: Van Cutsem et al. *Journal of Clinical Oncology* 2012 30:28, 3499-3506
***ML18147 Trial: Bennouna et al. *Lancet Oncol.* 2013, Jan;14(1):29-37,

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

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

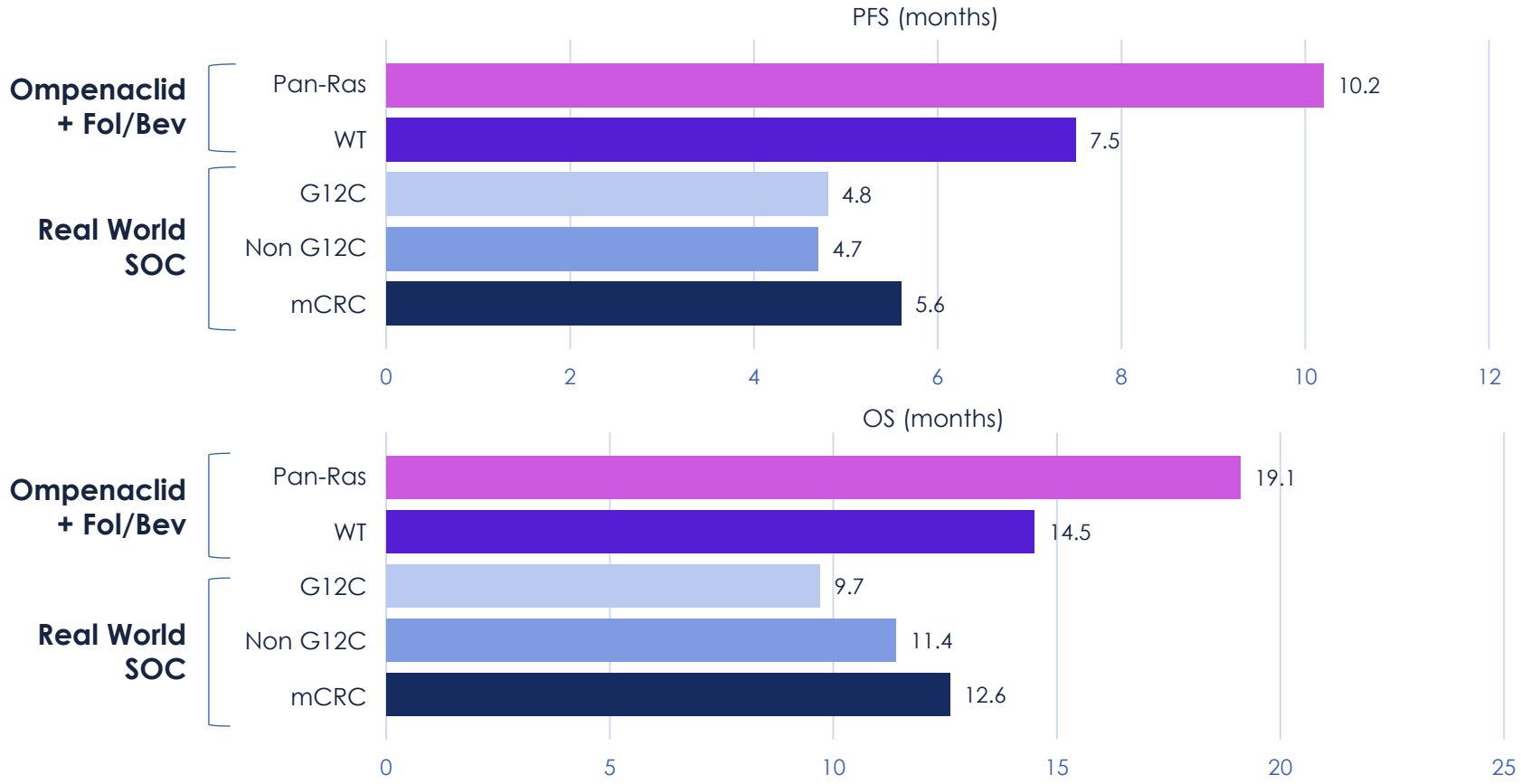


- Median (95% CI) rwPFS in second line settings
  - KRAS G12C cohort – 4.6 (2.6-5.5) months
  - KRAS non-G12C cohort – 4.7 (4.2-5.3) months
  - mCRC overall cohort – 5.6 (5.2-5.8) months

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

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

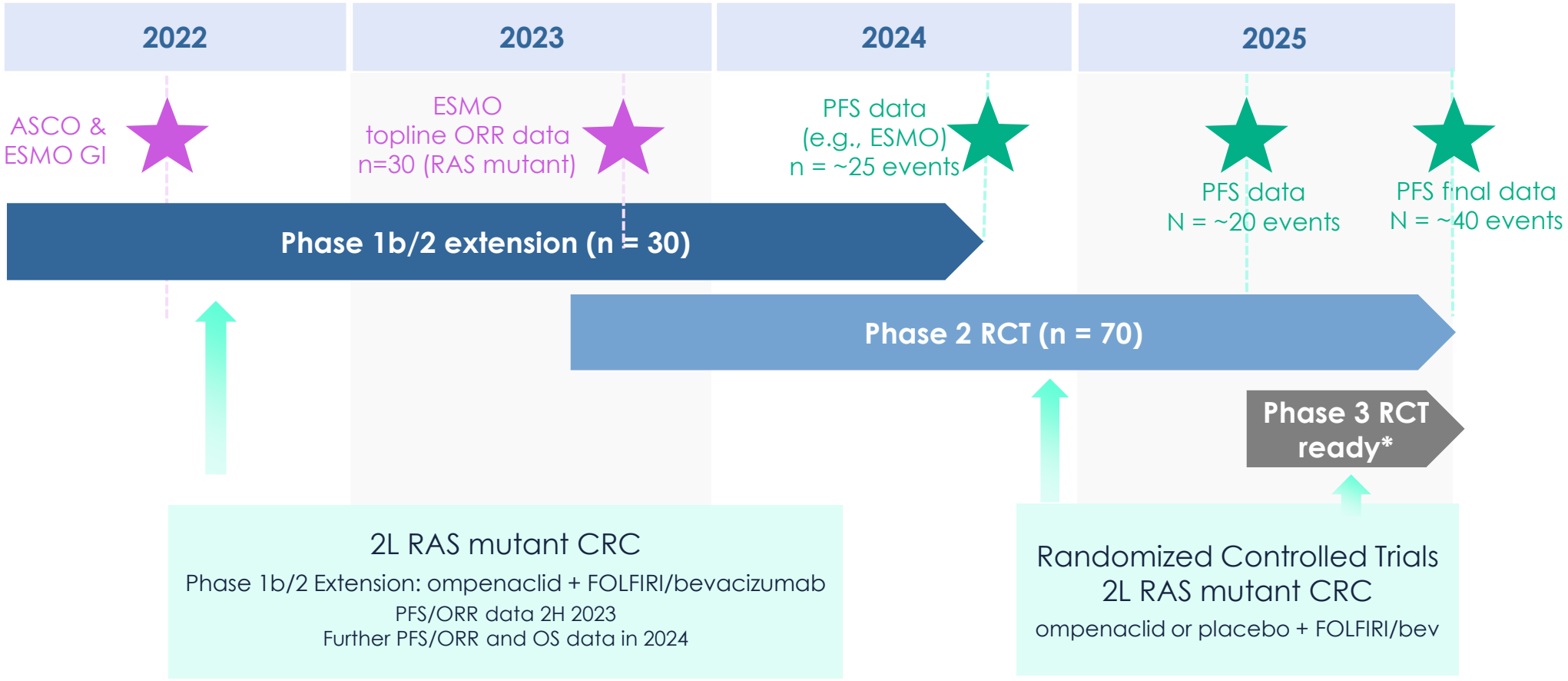
Ompenaclid phase 1b/2 compared to real world data (Fakih et al)



Data cut-off 18 Sep 2023

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

Projected clinical data read-outs for ompenaclid



*Note: clinical trial shown in gray arrow requires additional financing 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

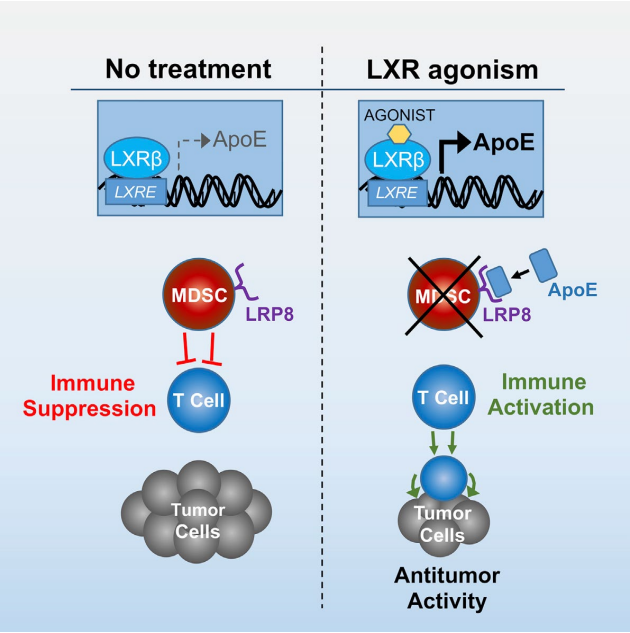
Inspirna Receives $45M Upfront Payment

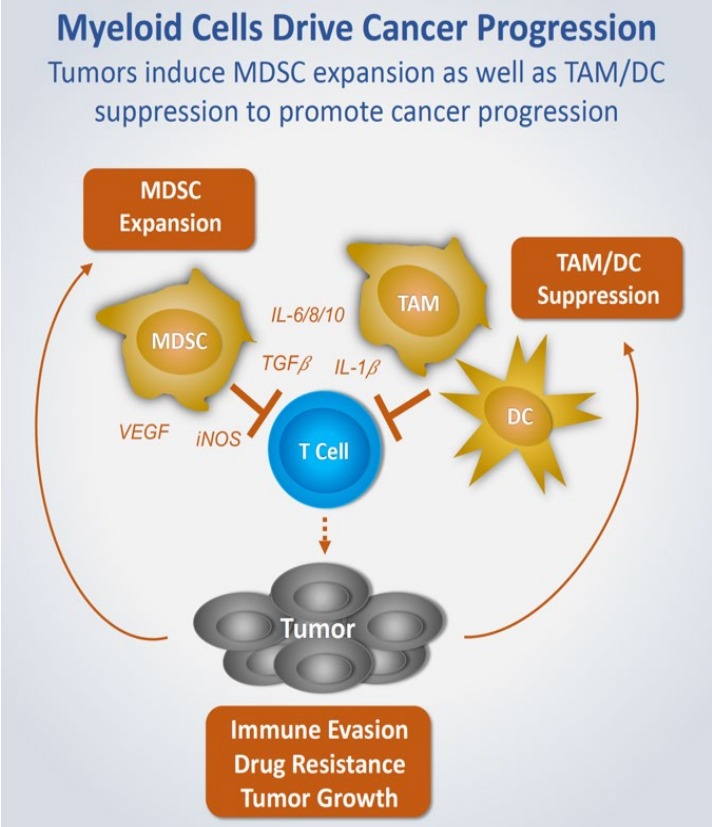
- **Merck KGaA, Darmstadt, Germany receives exclusive license to ompenaclid outside of US**
  - Option to co-develop and co-promote ompenaclid in US
- **Further collaboration focused on follow-on compounds targeting SLC6a8**
  - Inspirna will lead pre-IND activities, and retain co-development/co-commercialization rights in US
- **Inspirna receives $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)

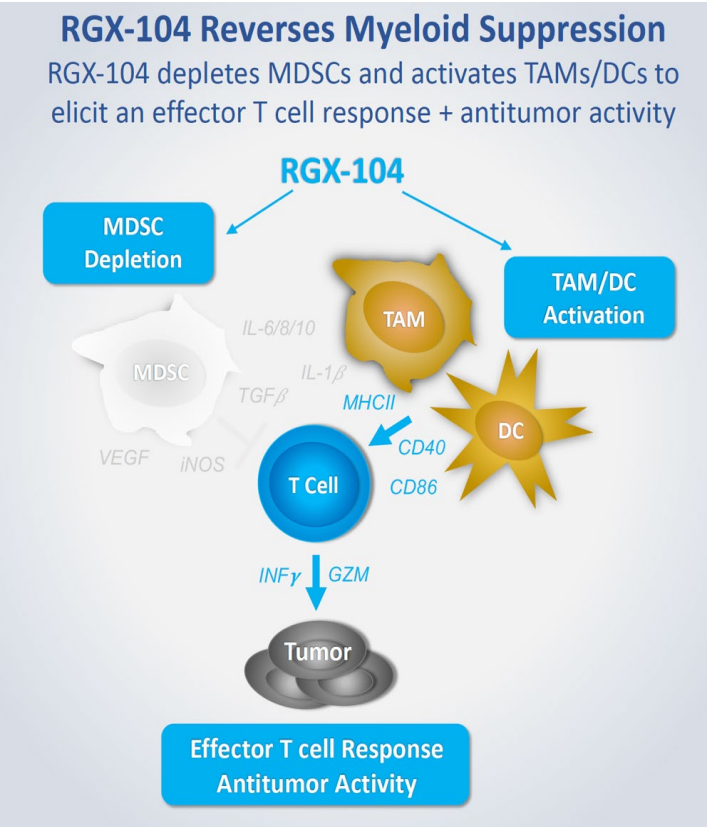
- Potent small molecule LXR beta selective agonist that activates APOE to target immune-suppressive tumor myeloid cells, while also inhibiting tumor angiogenesis
- Monotherapy activity in lung cancer demonstrated during Phase 1
- Clinical activity in combination with docetaxel demonstrated in an ongoing 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
- Ongoing collaboration with Bristol-Myers Squibb to evaluate abequolixron in combination with ipilimumab for the treatment of endometrial cancer
- Issued global patent protection to 2040

RGX-104 is a first-in-class clinically active LXR agonist that reverses myeloid suppression by targeting ApoE





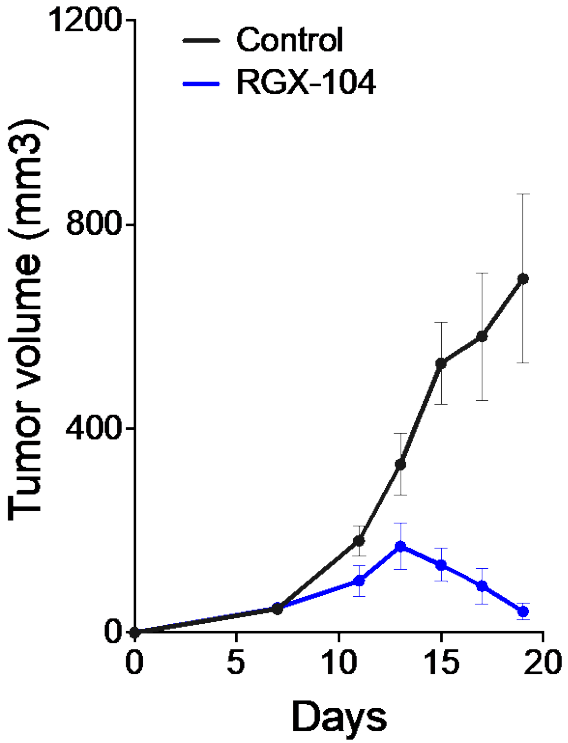
RGX-104



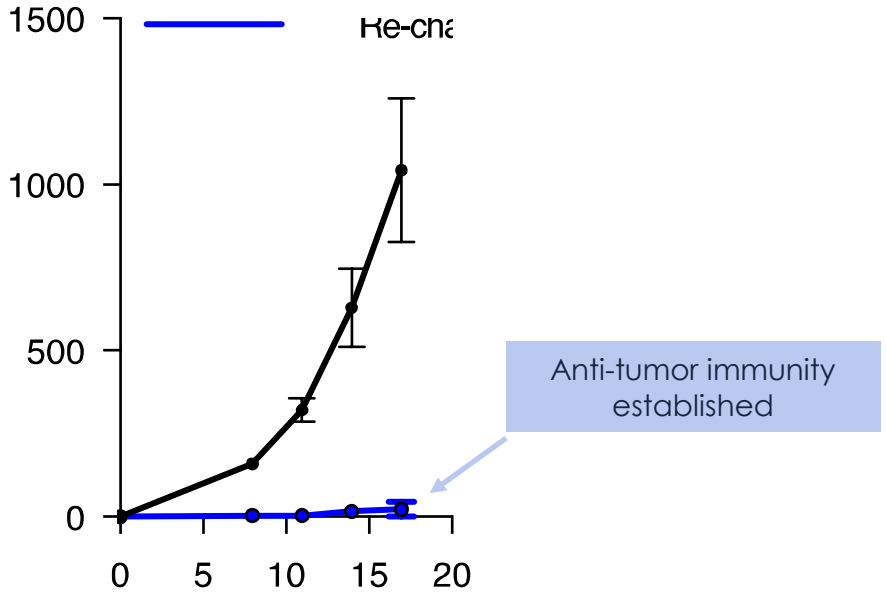
1) Mita M et al. ASCO (2018); 2) Mita M et al. AACR II (2020); 3) Tavazoie M et al. Cell. 2018 Feb 8;172(4):825-840; 4) Lim E et al. AACR I (2020)

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



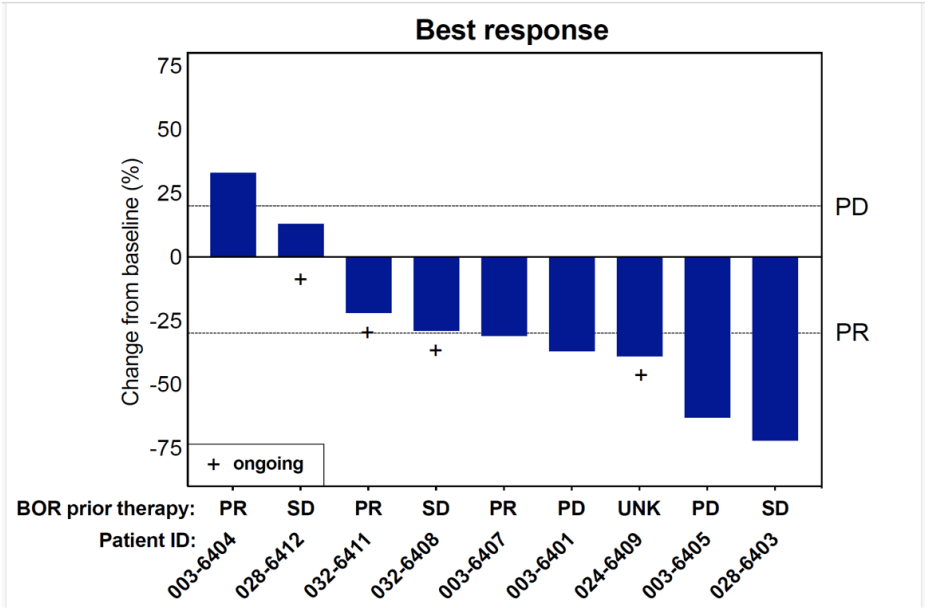
Syngeneic E0771-DR Breast Cancer model
Treatment initiated with RGX-104 (100mg/kg) on Day 3 (left figure)
No treatment administered in re-challenge experiment (right figure)

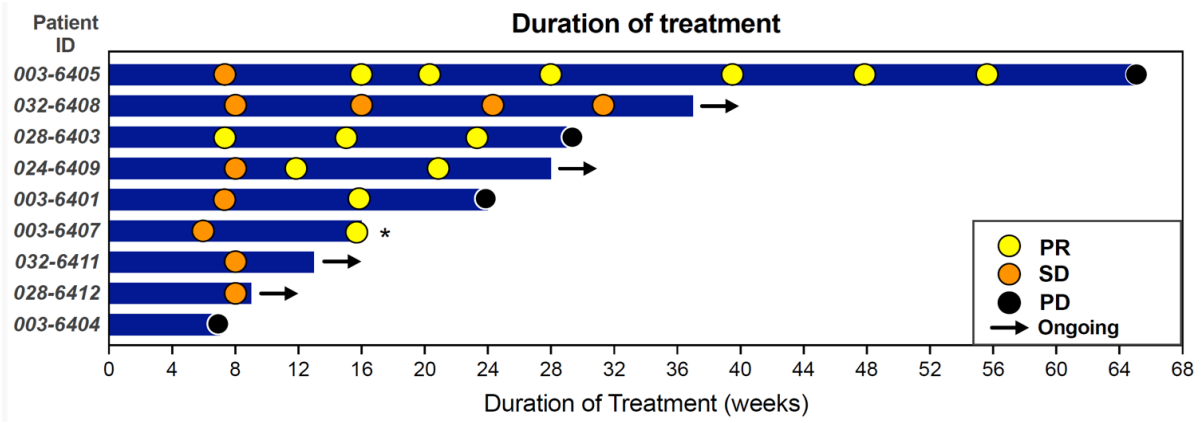
* 1. Tavazoie M et al. Cell. 2018 Feb 8;172(4):825-840, 2. Mita M et al. ASCO (2018), 3. Mita M et al. AACR II (2020), 4. Lim E et al. AACR I (2020), 5. Liang H. Biochem Biophys Res Commun. 2020 Jul 23;528(2):330-335, 6. Wan d et al. J Control Release. 2020 Jan 10;317:43-56

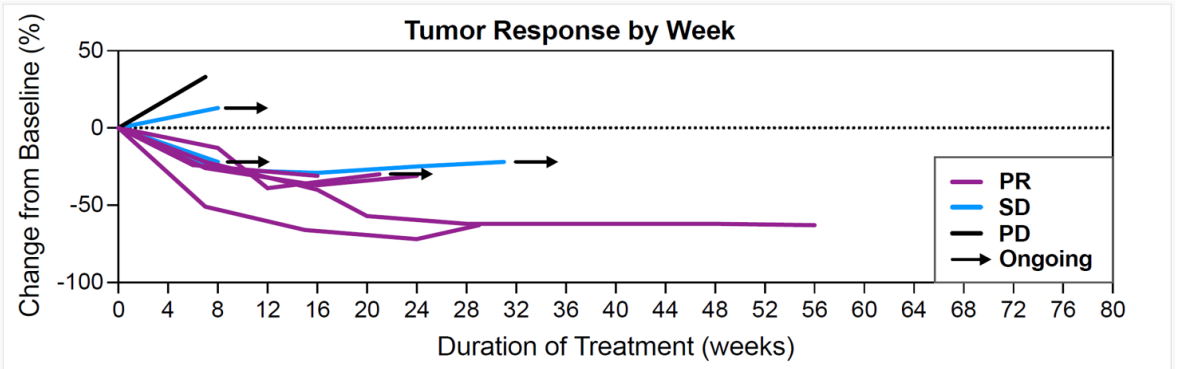
Clinical Activity Observed in 2/3L NSCLC (RGX-104 + docetaxel)

Benchmark Data for 2/3L NSCLC: From Lung-MAP S1800A Trial

- 166 patients randomized to Pembro +Ramucirumab(R) vs SOC (IC of docetaxel+R; docetaxel, pemetrexed, gemcitabine)
- mOS 14.5 mo for P+R vs 11.6 mo for SOC (significant);
- mPFS 4.5 mo for P+R vs 5.2 mo for SOC arm (NS);
- ORR 22% for P+R vs 28% SOC, respectively (NS)



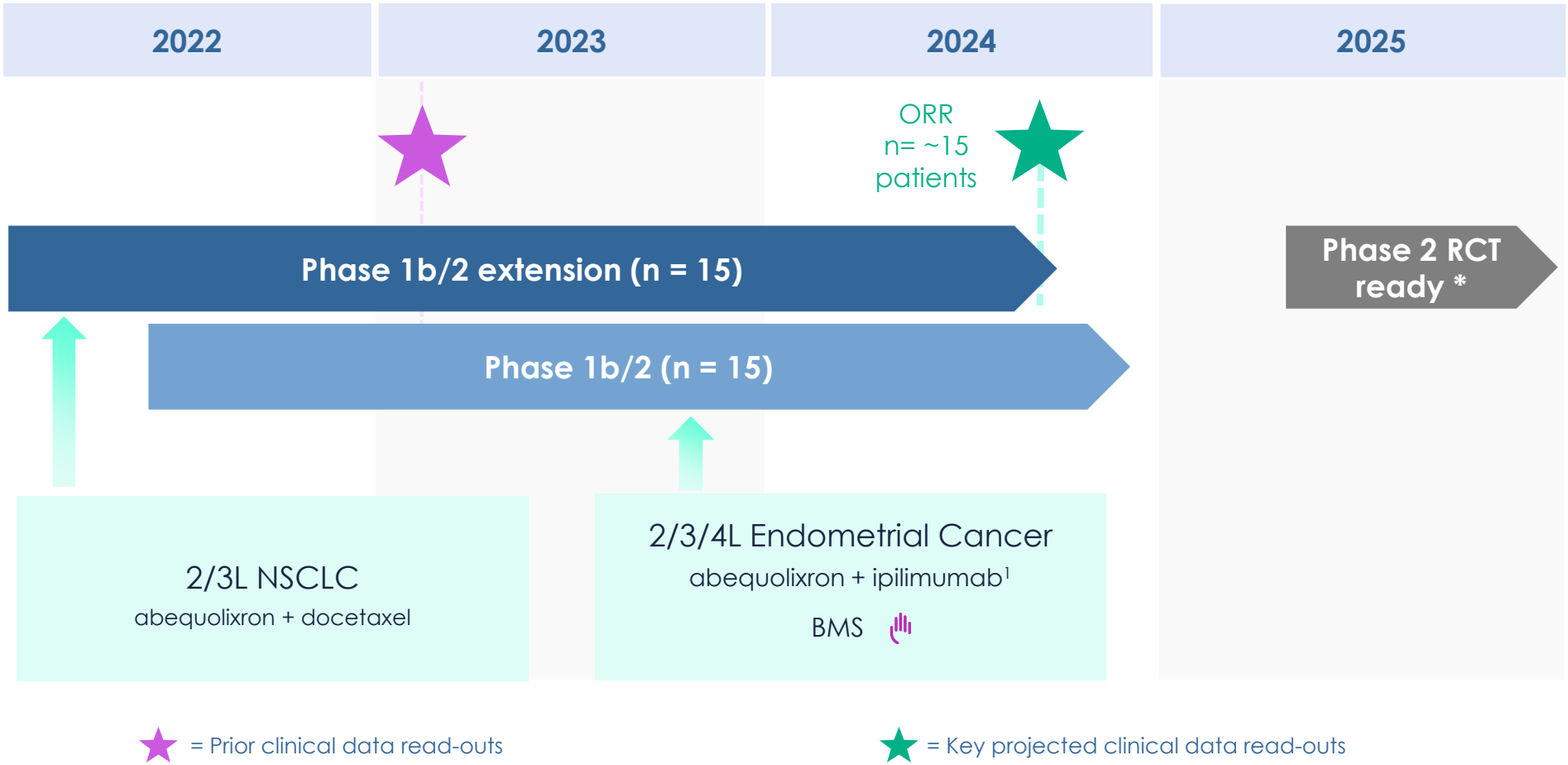




Patients were evaluable for RECIST 1.1 response if they completed at least one treatment cycle and had at least one follow-up scan for RECIST 1.1 assessment. 3 patients were not evaluable for ORR response: 024-6402 – PI decision; 001-6406, baseline scans only and 6 weeks on treatment. 038-6410 – Withdrew consent (1 week on treatment)
*Patient discontinued due to AE's weight loss and persistent nausea. 028-6403 had PD due to CNS lesions

Data cut-off 11/30/2023; Open database, data subject to change

Projected clinical data read-outs for abequolixron



*Indication as well as start date/execution depends on study results as well as additional financing/FDA feedback
[1]Clinical collaboration with Bristol Myers Squibb to investigate abequolixron in combination with ipilimumab in 2/3/4L endometrial cancer setting

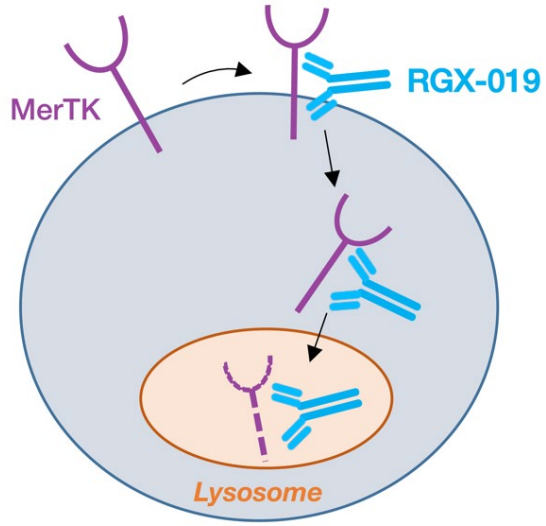
RGX-019 ADC

- Humanized anti-MERTK monoclonal IgG1 antibody being developed as an antibody-drug conjugate (ADC)
- MERTK is over-expressed in several solid and liquid cancers with limited expression on healthy tissues
- RGX-019 ADC binds to a unique epitope that triggers MERTK receptor internalization and partial agonist activity enabling selective delivery of toxic payload to MERTK expressing cancer cells
- Partial agonist activity provides safety advantage over other MERTK targeting agents that have shown retinal toxicity
- Issued composition-of-matter patent protection on RGX-019 ADC (2040 expiration)

RGX-019 ADC is a novel MERTK targeting antibody-drug conjugate

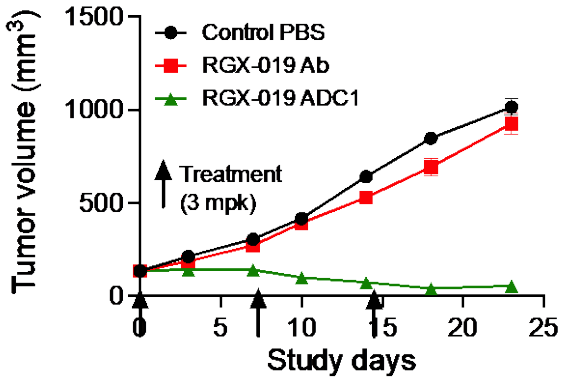
MERTK is selectively over-expressed in multiple solid and liquid cancers

RGX-019 internalizes surface MERTK receptors on cancer cells and immune-suppressive M2 TAMs

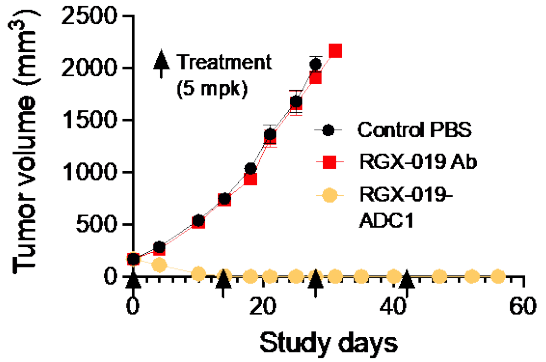


RGX-019 ADC (MMAE) has potent *in vivo* anti-tumor activity in select solid and liquid tumor models

MDA-MB-231 (TNBC)



RPMI8226 (MM)



MDA-MB-231 cells were injected into the 4th mammary fat pad of Athymic nude mice. Treatment with indicated amounts of control (PBS), RGX-019 or RGX-019-ADC started when tumors reached ~150mm3; n=8-10. RPMI cells were injected subcutaneously into NOD-SCID mice,

Sources: 1. Png et al. Nature. Dec 14; 2011.481(7380):190-4, 2. Takeda et al. AACR 2019, 3. Takeda et al. AACR Virtual Meeting (II) 2020, 4. Inspirna unpublished data

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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 next 12-18 months
  - Lead candidate ompenaclid is a novel small molecule that inhibits SLC6A8 with activity in patients with RAS mutant colorectal cancer, currently in Phase 2 development and Phase 3 ready by 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 and endometrial cancer
  - RGX-019 ADC is a novel MERTK targeting antibody-drug conjugate targeting multiple MERTK high tumor types
- Led by an experienced management team, supported by top-tier institutional investors, collaborators and advisors