The DARPin® Difference: Offering Patients a New Dimension of Protein Therapeutics

R&D Day Molecular Partners
New York
November 9, 2017

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Speakers & Agenda

Introduction & Moderation
Patrick Amstutz, PhD
Chief Executive Officer

Clinical Pipeline – Oncology
Andreas Harstrick, MD
Chief Medical Officer

Multiple Myeloma
Angelo Vacca, MD
University of Bari, Medical School

Non-Small Cell Lung Cancer
Kate Gold, MD
UCSD Moores Cancer Center

HER2+ Cancers
Richard Baird, MD, PhD
University of Cambridge

DARPin® Difference & Immuno-Oncology
Michael Stumpp, PhD
Chief Scientific Officer

Ophthalmology – Abicipar
Baldo Scassellati Sforzolini, MD, PhD, MBA
SVP, Clinical Development, Allergan

Question and Answer Session
All

Key Messages for Today

- Successful transition from DARPin® platform into clinical product company
- Key value inflection points ahead:
  - MP0250 (2x Phase 2) and MP0274 (Phase 1) in oncology
  - Abicipar (Phase 3 data) in ophthalmology
- MP0310 selected as 1st development candidate from our I/O DARPin® toolbox
- Financed beyond 2019, capturing key value inflection points
- Keep on forward integrating towards late-stage development and the market

Making the DARPin® Difference Reality for Patients

Patrick Amstutz, CEO

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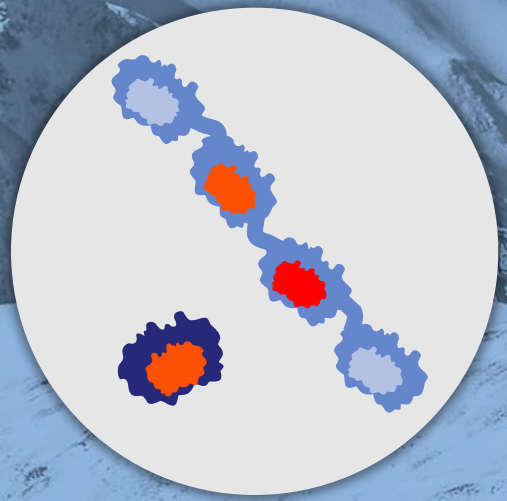
Molecular Partners – Opening a New Chapter

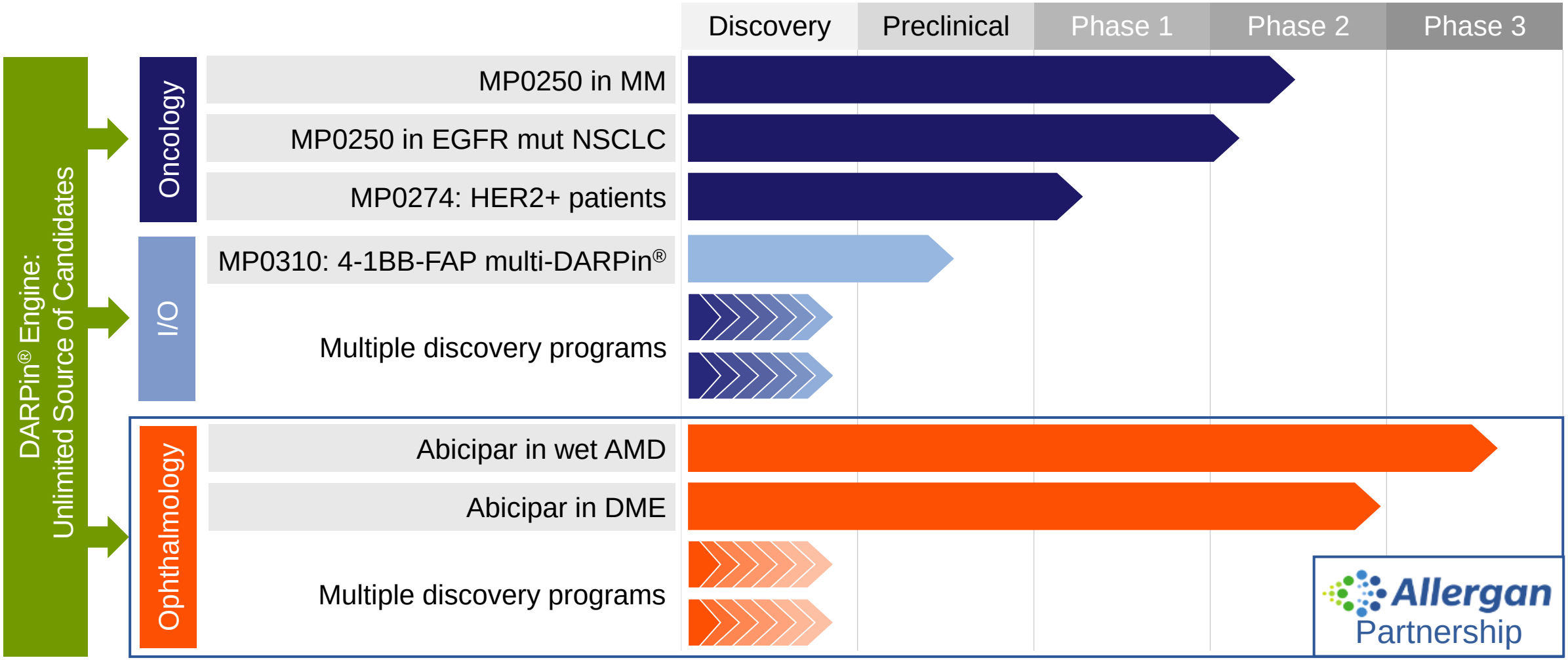
DARPin® platform company

Clinical product company

Fully integrated biotech in oncology



Balanced and Robust Portfolio



AMD, age-related macular degeneration; DME, diabetic macular edema; MM, multiple myeloma; NSCLC, non-small cell lung cancer.

Turning the DARPin® Differentiation into Patient Outcome – Our Target Profiles

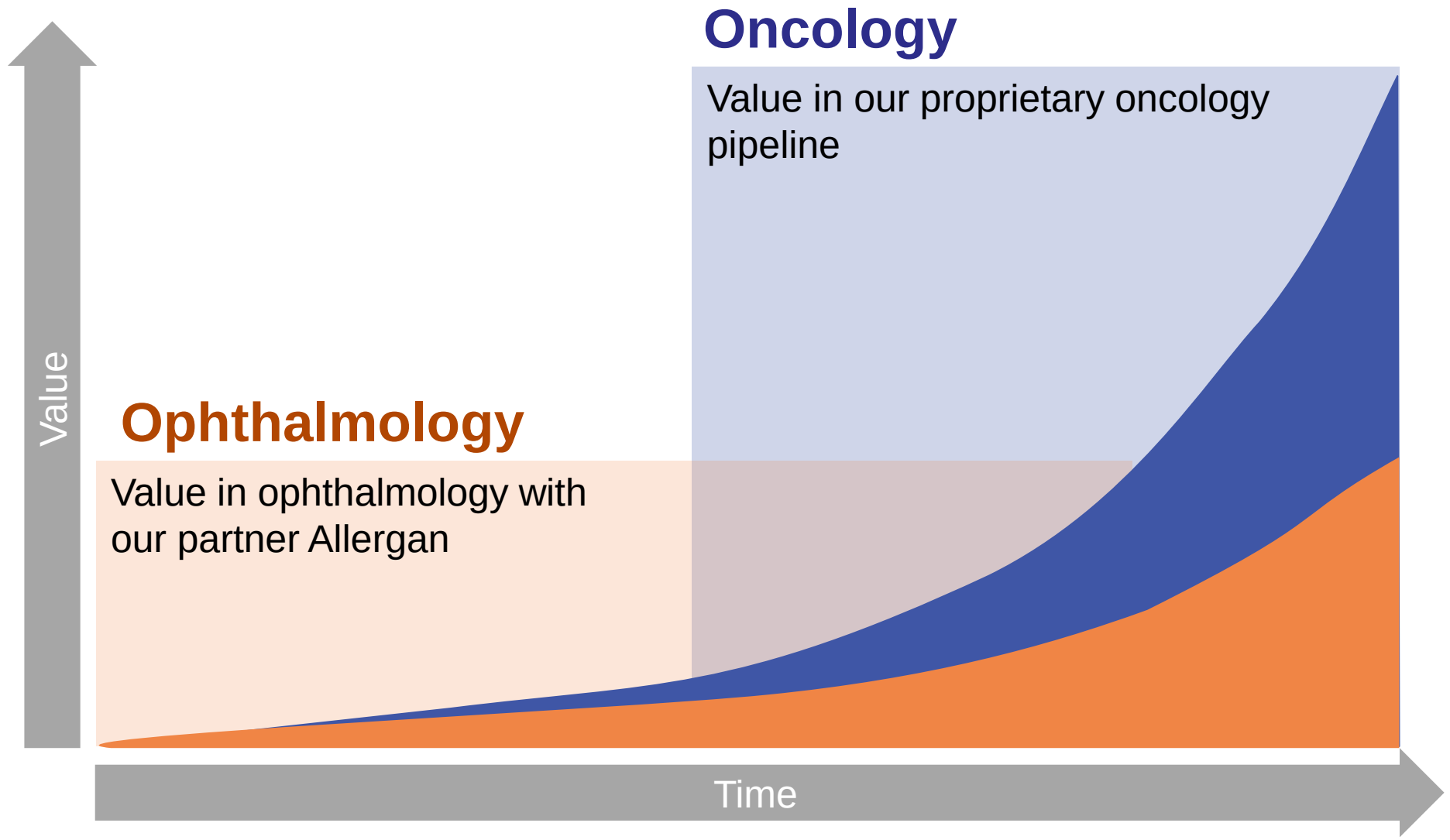
Pre-Clinical	Phase 1	Phase 2	Phase 3
MP0310	**MP0274**	**MP0250**	**Abicipar**
		NSCLC	Wet AMD
		MM	
Tumor-restricted activity (switch) to avoid dose-limiting side effects	Molecular handcuff inducing cell death in HER2+ cancer cells	Blocking 2 key escape pathways in parallel	Long-acting VEGF inhibitor in the eye
Opening a new therapeutic window for combinations	Activity in patients no longer benefiting from approved antibodies	Restore activity of drugs to which cancer has become resistant in MM and NSCLC	Non-inferiority to competition with less frequent ocular injections

Multiple Value Inflection Points Ahead

Funding beyond 2019

	2018	2019	2020
Abicipar	Wet AMD: 1-y Ph 3 efficacy DME: Ph 3 expected start		Wet AMD: expected launch in 2020
MP0250	MM: initial efficacy NSCLC: initial safety	MM: efficacy NSCLC: initial efficacy	NSCLC: efficacy
MP0274	Initial safety Initial efficacy	Efficacy	
MP0310	Preclinical data	FIH	

Ready to Capture Value Beyond Ophthalmology



Clinical Oncology Pipeline

Andreas Harstrick, CMO

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MP0250: A First-in-Class Bi-Specific DARPin® Molecule

MP0250

- First bi-specific biologic blocking VEGF and HGF

- VEGF and HGF/cMET are key escape pathways to SOC treatments
- This escape has been described for liquid and solid tumors
- Blocking the escape pathways may restore activity of SOC drugs

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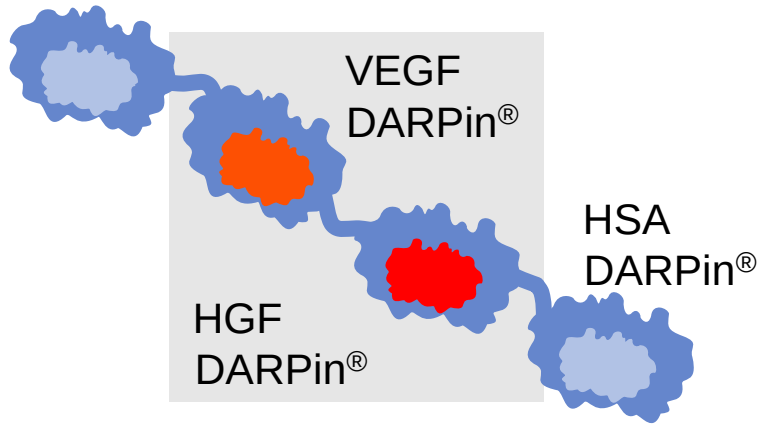
- Our choice of indications
  - Multiple myeloma (MM)
  - EGFR mutated non-small cell lung cancer (NSCLC)
- Potential in additional indications

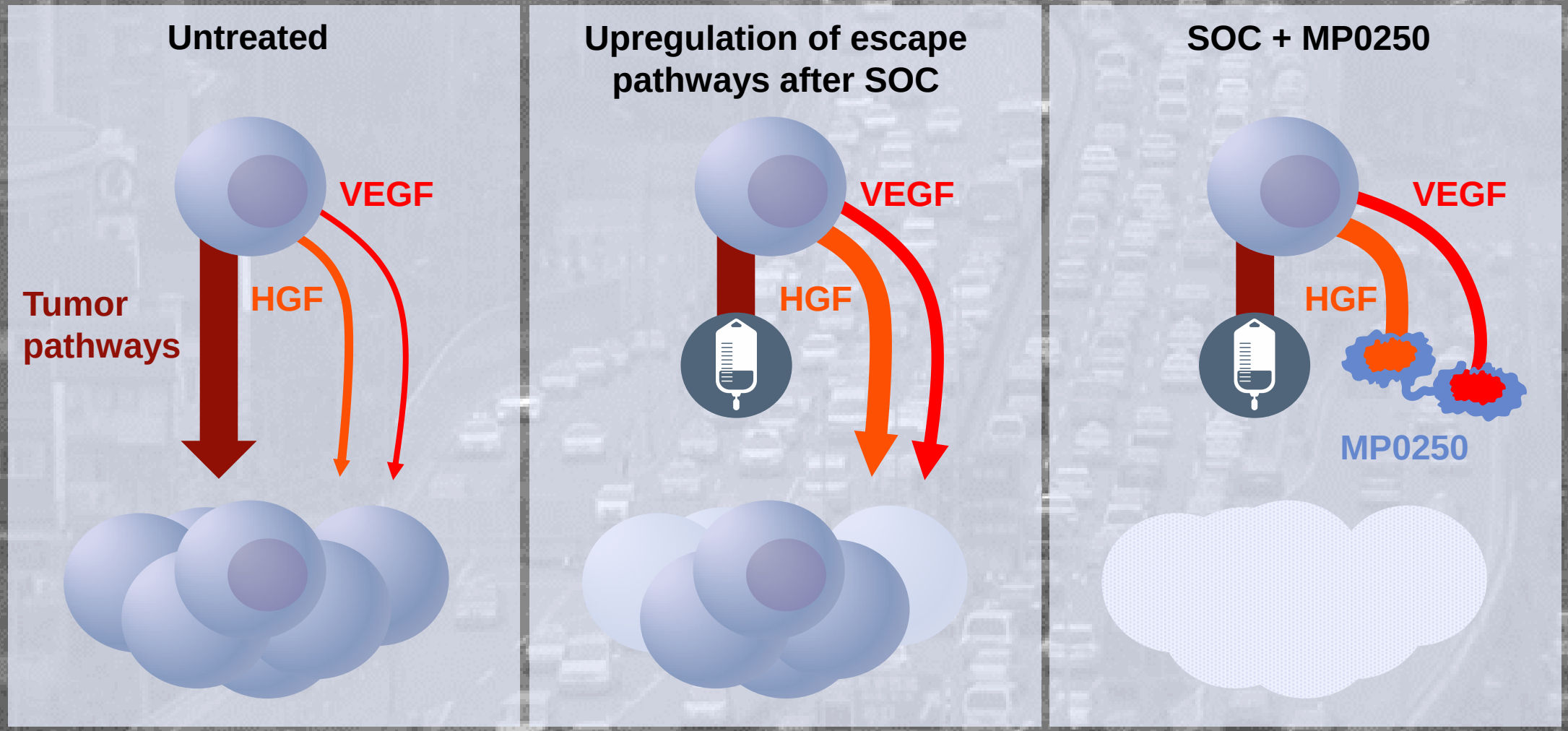


- Fully owned by Molecular Partners – IP protection at least until 2036

SOC, standard of care; HSA, human serum albumin

MP0250 Blocks Two Tumor Escape Pathways

MP0250

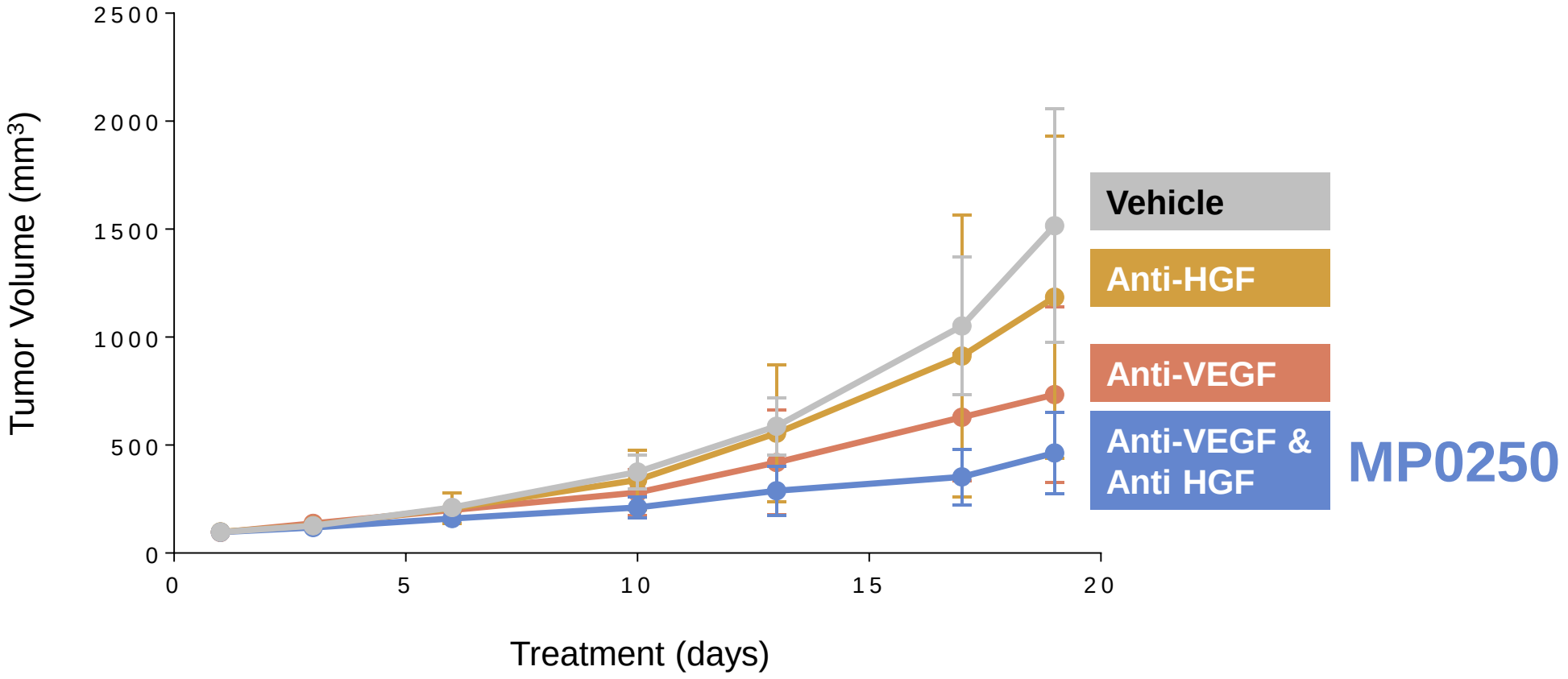


Combined HGF and VEGF Inhibition Leads to Synergistic Effect in Mouse Model

MP0250

Tumor Growth Inhibition

Syngeneic colorectal cancer model* (MC38)



*Syngeneic model: HGF-cMet axis fully functional – murine tumor grown in mouse strain of origin.

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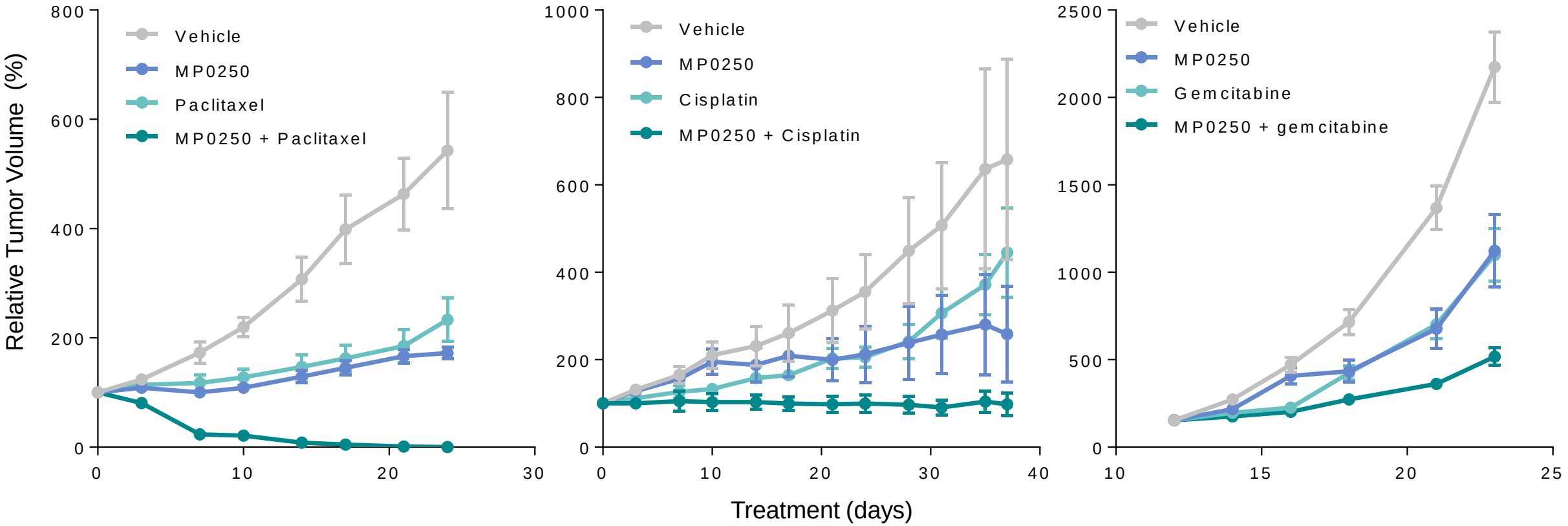
MP0250 can be Combined with Many Agents Across Different Tumors to Increase Their Potency

MP0250

Gastric cancer PDX model
GXA3027

Head & Neck cancer PDX model
HNXF1905

Pancreatic cancer tumor model
KP4



PDX model, patient-derived xenograft mouse model

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MP0250 Can Be Dosed Safely, Conveniently and Shows Clear Signs of Efficacy in Phase 1 Study

MP0250

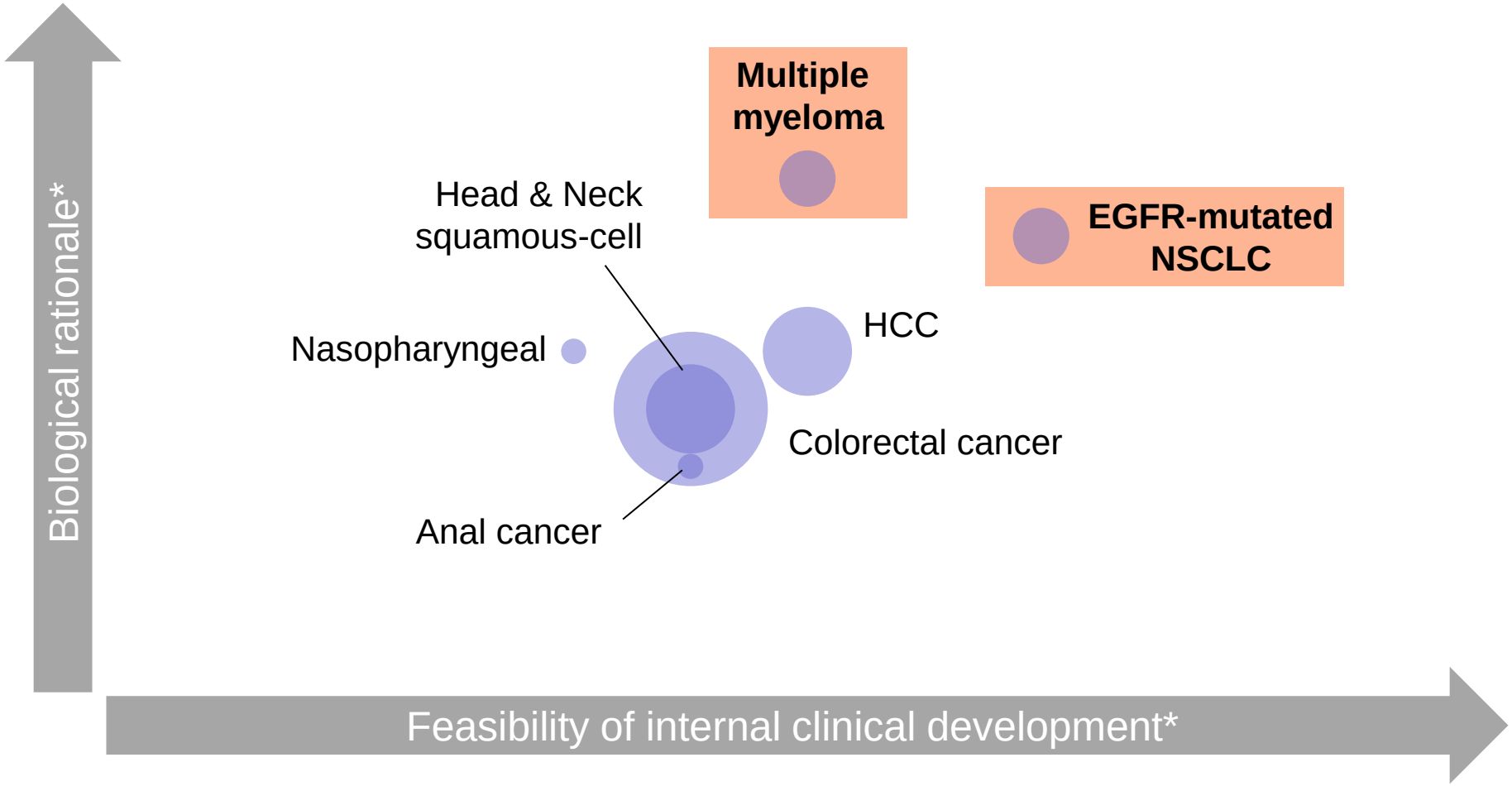
Dosing* Convenient, flexible administration	**Exposure** Repeated dosing resulted in good exposure	**Safety** Well tolerated	**Efficacy** Clear signs of antitumor efficacy
▪ Infusions well tolerated ▪ Dosing every 2 or 3 weeks possible ▪ Systemic half-life: ~2 weeks	▪ Sustained drug exposure throughout treatment periods (max. to date >12 months) ▪ Only 1/40 patients developed a relevant titer of ADAs (>10 fold above background)	▪ Most common AE was hypertension, generally well controlled with standard medication: AEs were as expected for a VEGF inhibitor	▪ 2 patients showed significant reduction in tumor volume ▪ Treatment duration was ≥3 mo in 18 patients (40%) and ≥6 mo in 4 patients (10%)

These first-in-human data give confidence that DARPin® therapies can be developed in general for systemic administration.

* 1- and 3-h infusion q2wk at doses ≤8 mg/kg or q3wk at 12 mg/kg; 1-h and 3 h infusion well tolerated
ADA, anti-drug antibody; AE, adverse event.
**Study details can be found on clinicaltrials.gov using the identifier: NCT02194426.

Our Indications for Phase 2: MM and NSCLC

MP0250

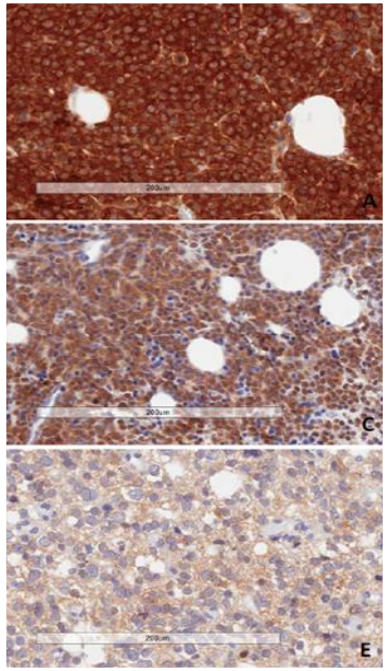


Bubble size indicates estimated relative market potential (incidences; source: Datamonitor).
*Based on internal assessment on speed to market and complexity of development program.
Potential of gastric cancer, renal cancer and other cancers under evaluation.

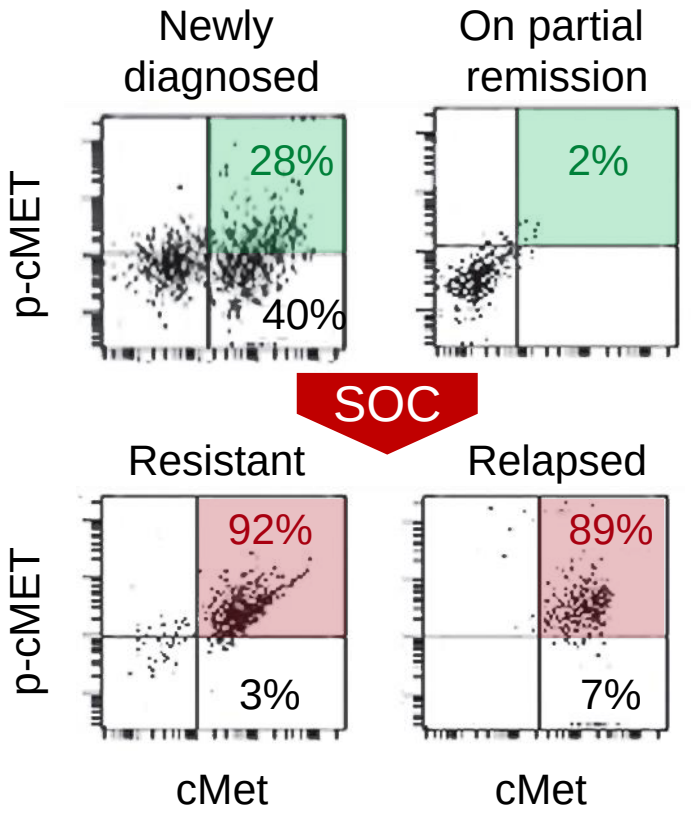
HGF & VEGF Rationale in MM is Supported by Clinical Data

MP0250

Bone marrow of 8 MM patients sampled for HGF expression levels:

HGF score		Patients
3+		6/8
2+		2/8
1+		0/8

HGF receptor activation[1] dynamics

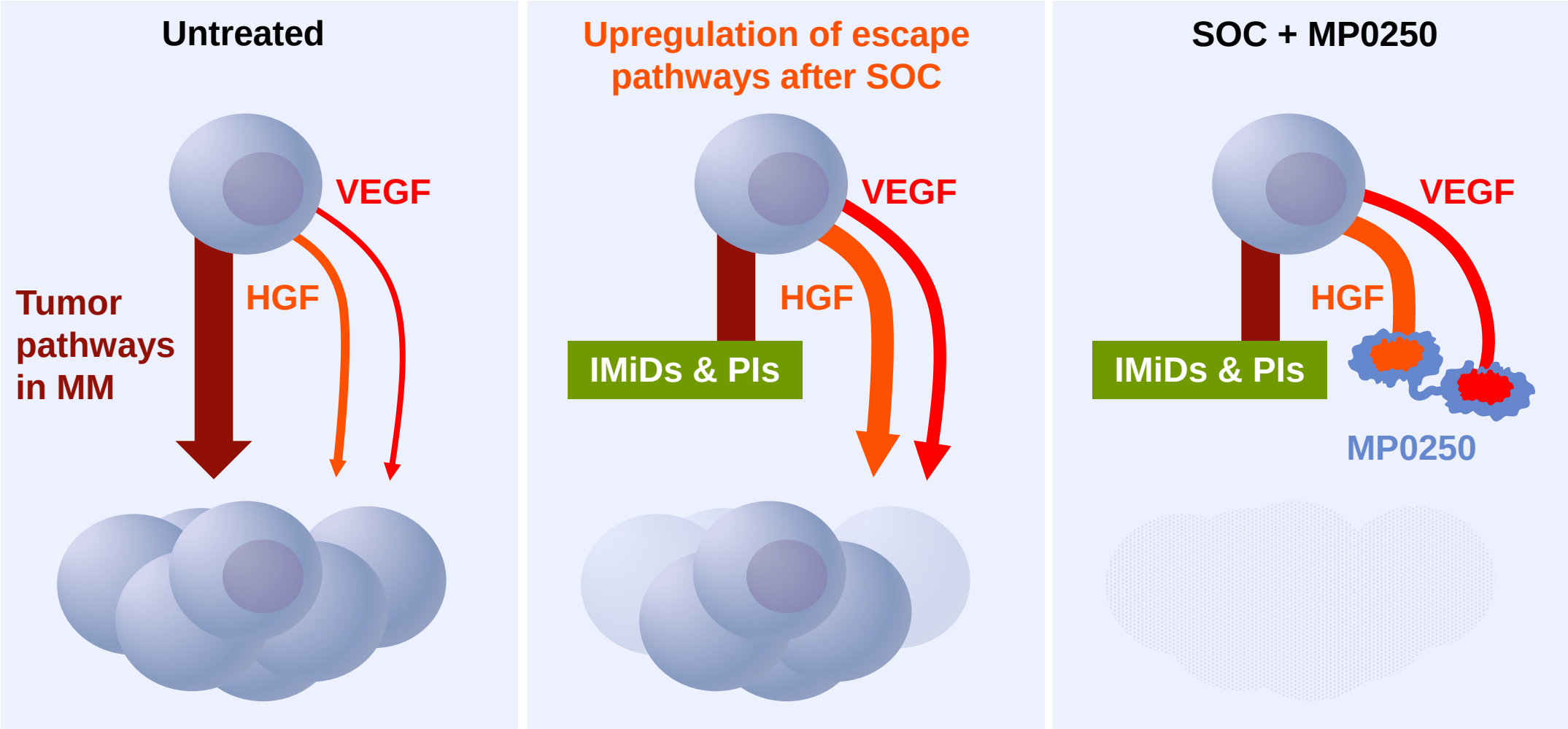


VEGF rationale: A small MM study of bevacizumab (Avastin®) + bortezomib (Velcade®) demonstrated benefit over bortezomib alone[2]

1. Moschetta M, et al. Clin Cancer Res 2013;19:4371-82; 2. White D, et al. Cancer 2013;119:339-47.

MP0250 Rationale: Reverse Resistance in MM

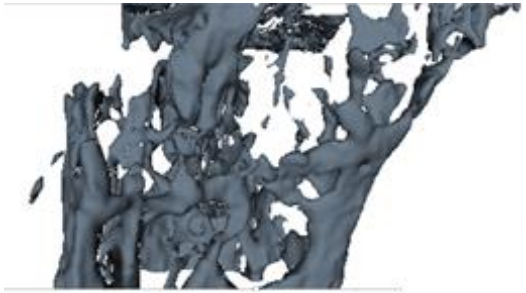
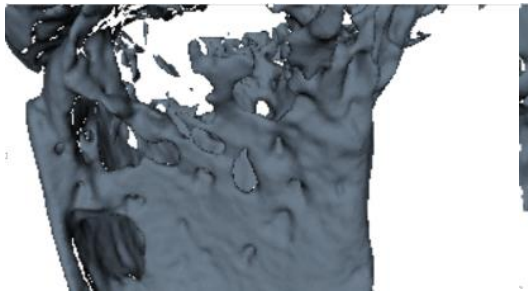
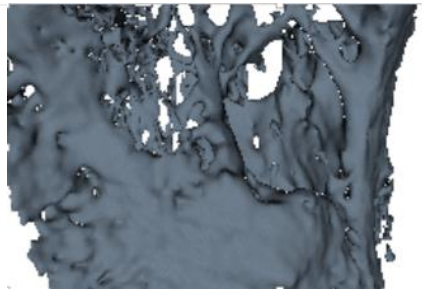
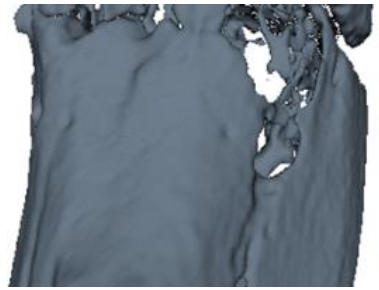
MP0250



IMiD, immunomodulatory drug; PI, proteosome inhibitor.

MP0250 Combination with Bortezomib (Velcade) Results in Superior Efficacy in Mouse Model

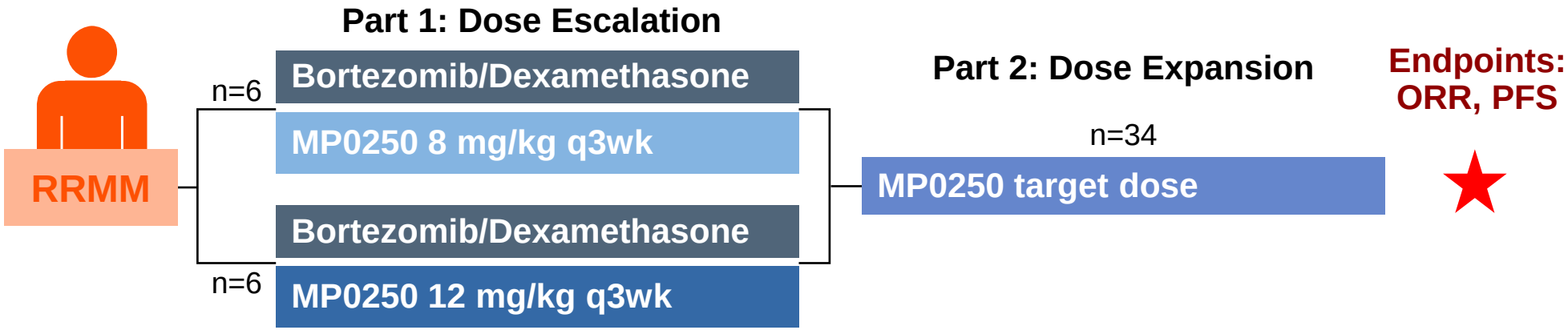
MP0250

	Vehicle	Velcade	MP0250	Velcade + MP0250
Bone morphology				
Muscle invasion				

Xenograft model: H929.

MP0250 Phase 2 Study in MM

MP0250



- Phase 2 open-label, single-arm, multicenter study of MP0250 + bortezomib + dexamethasone in patients with refractory and relapsed multiple myeloma (RRMM)
- Study population: MM patients who have received ≥2 lines of therapy, including bortezomib and an IMiD, and have shown no response to most recent therapy or progressed ≤60 days after most recent therapy
- Study status: actively recruiting for Part 1
- Next readouts: Initial safety 2017 / Initial efficacy 2018

*Study details can be found on clinicaltrials.gov using the identifier: NCT03136653.

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Current Treatment Strategies in MM

MP0250

- Multiple myeloma is the second most common blood cancer
- Patients are elderly (median 68 years)
- Global market value ~USD 9bn, expected to reach >12bn by 2021 (6% CAGR)*
- Despite new drugs there is no cure and patients eventually progress on treatment

Stem Cell Transplant

I/O approaches (CAR-Ts – BCMA)

Backbone

PIs (proteasome inhibitors)
Velcade (Bortezomib)
Kyprolis (Carfilzomib)
Ninlaro (Ixazomib)

IMiDs
Revlimid (Lenalidomide)
Pomalyst (Pomalidomide)

Antibodies
Darzalex (Daratumumab)
Empliciti (Elotuzumab)

Later-line treatments

Farydak

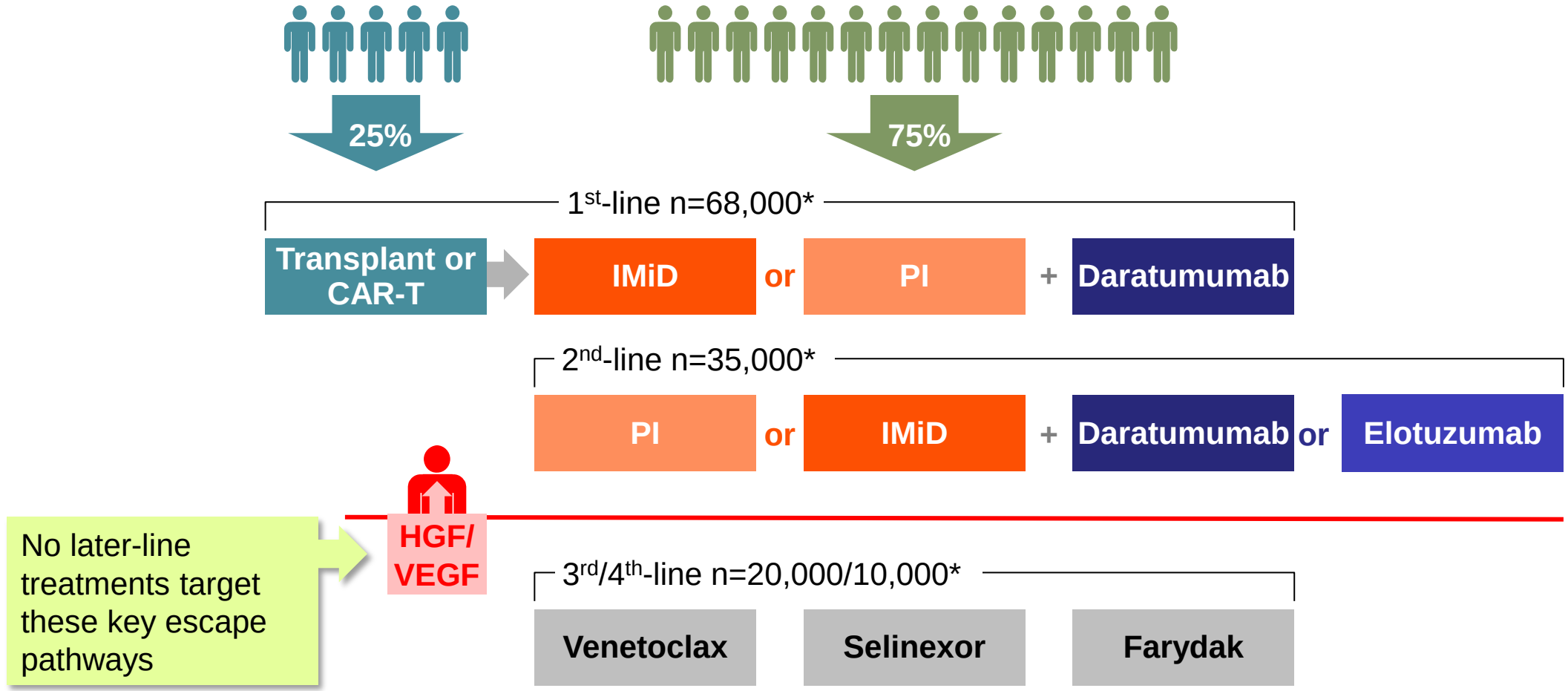
Venetoclax

Selinexor

*Including US/5EU/JP. Datamonitor.

Unique Potential of MP0250 in MM

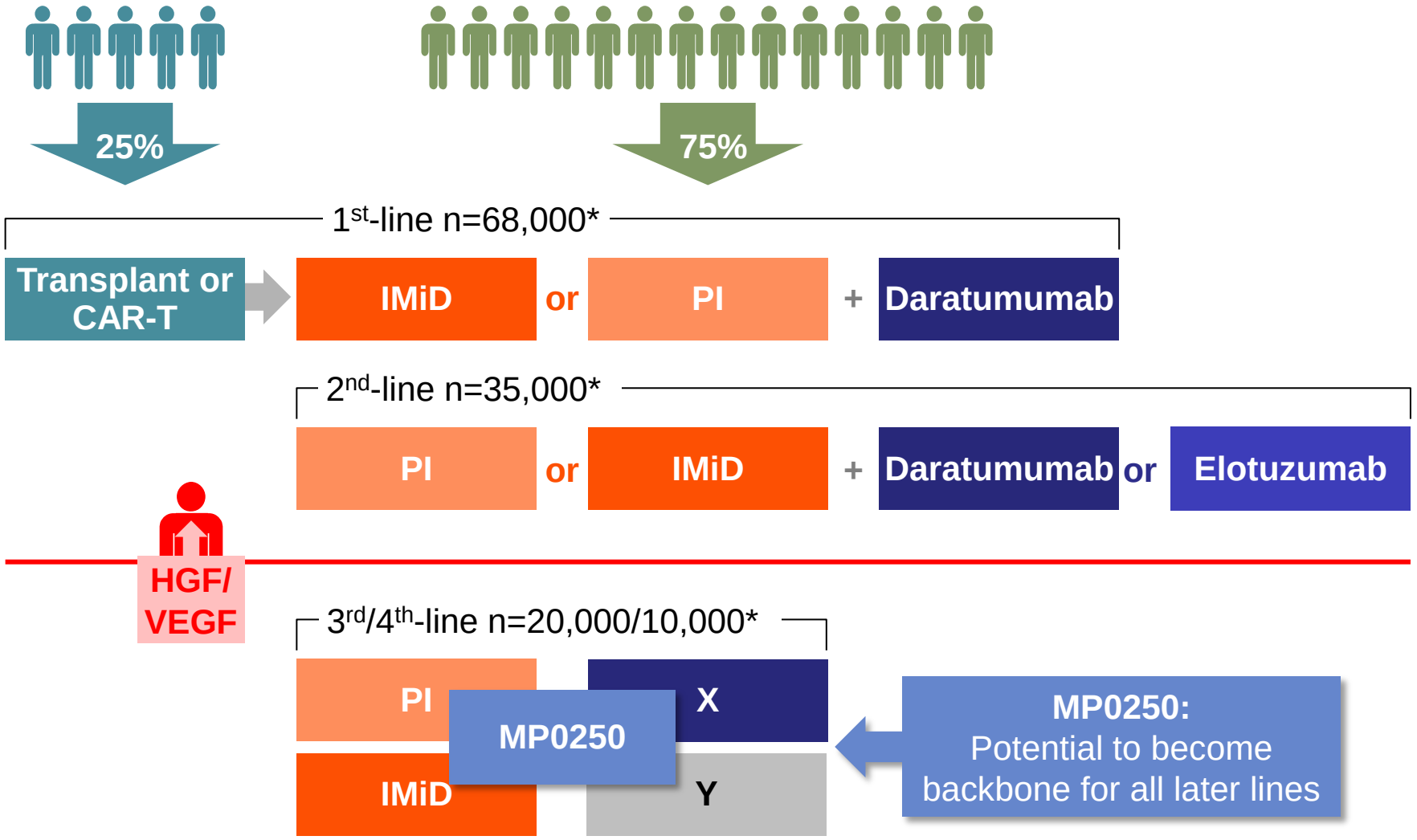
MP0250



*Including US/5EU/JP. Datamonitor.

Target Product Profile of MP0250 in MM

MP0250



*Including US/5EU/JP. Datamonitor.

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EGFR mut NSCLC – High Medical Need

MP0250

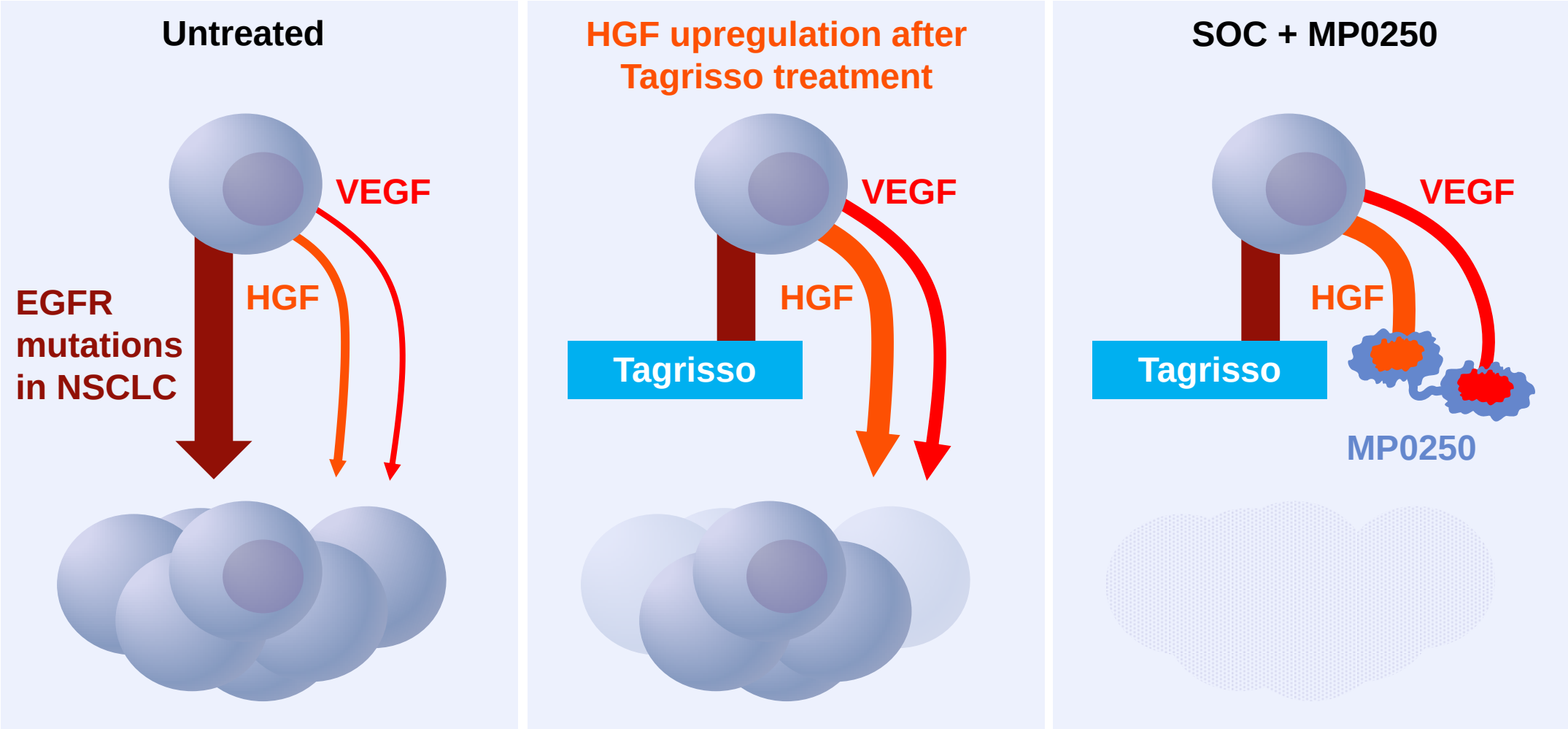
- NSCLC is leading cause of cancer death
- Activating EGFR mutations are found in up to 10% of Western and up to 50% of Asian NSCLC[1]
- 60% of EGFR mut NSCLC patients who failed 1st gen. TKI (Iressa, Tarceva) develop a 2nd, resistance-conferring mutation in EGFR (T790M)[1]
- Tagrisso (osimertinib) is the only approved agent to treat T790M+ NSCLC

- Activation of c-MET pathway appears to be significantly involved in acquired resistance to Tagrisso
  - Savolitinib (c-MET TKI) plus Tagrisso produced 28% response in patients who failed Tagrisso and showed MET amplification
  - c-MET pathway validated in this subset of patients
  - c-MET amplification (15%) is a subgroup of c-MET/HGF upregulation (60–80%)[2]

TKI, tyrosine kinase inhibitor.
1. Including US/5EU/JP. Datamonitor; 2. Spigel R, et al. J Clin Onc 2017;35:412-20.

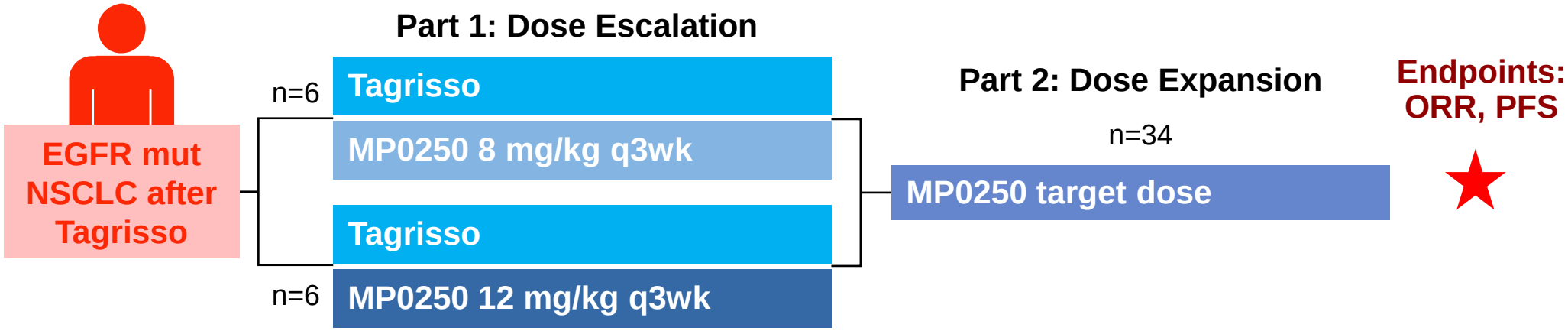
MP0250 Rationale in NSCLC

MP0250



MP0250 Phase 2 Study in NSCLC

MP0250



- Status: FDA approval Sep 2017 – 1st oncology DARPin® drug candidate in US
- On track to dose 1st patient in Q1 2018
- Next readouts: initial safety in 2018 & initial efficacy 2019

*The study details can be found on clinicaltrials.gov when available.

Unique Potential of MP0250 in EGFR mut NSCLC

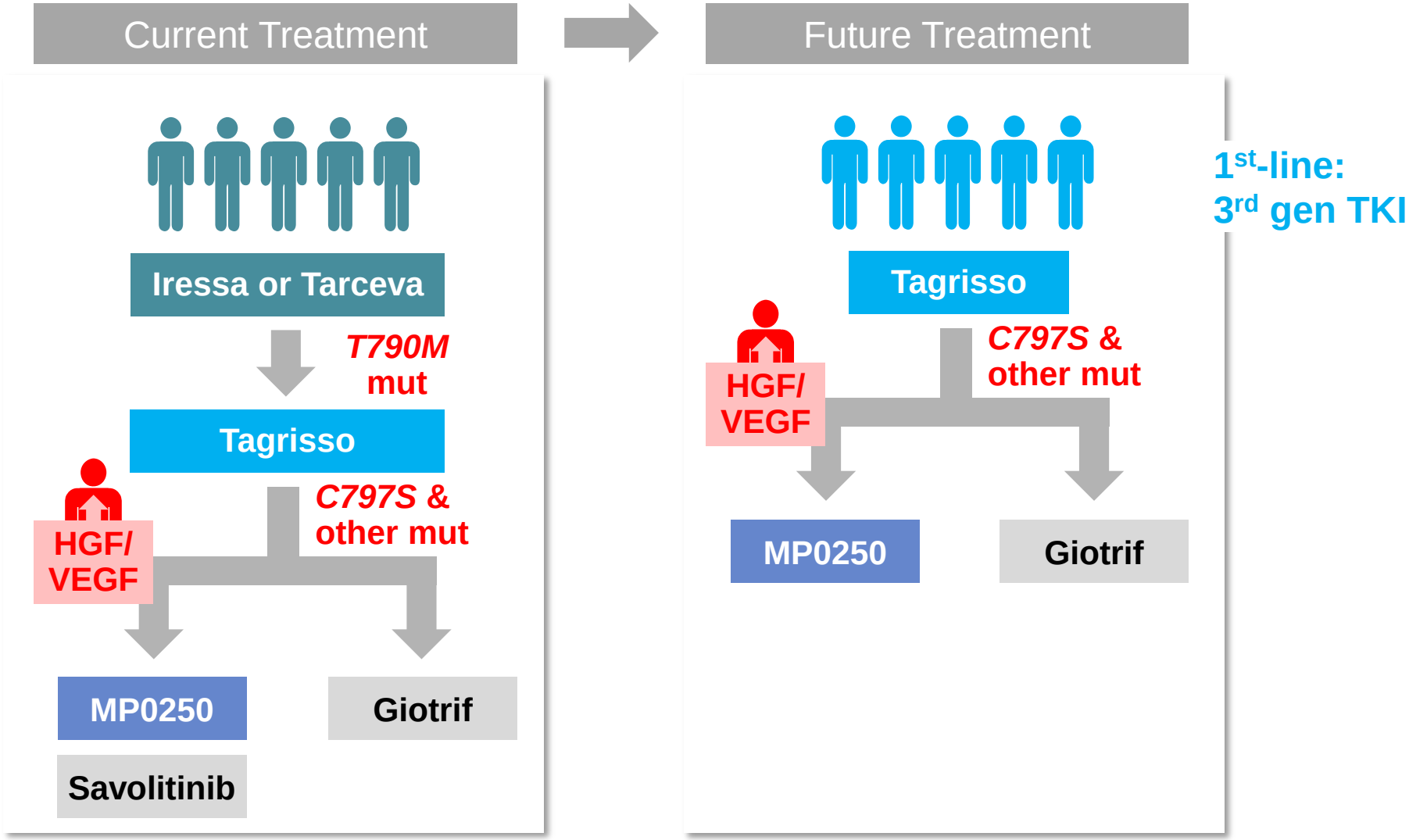
MP0250

Treatment Line
(# of patients today[1])

1st-line:
1st gen TKI
n=200,000

2nd-line:
3rd gen TKI
n=130,000

Refractory
n=70,000



[1] Including US/5EU/JP. Datamonitor.

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MP0274: Killing HER2+ Cells with New MoA

MP0274

- Allosteric inhibitor of Her2 blocks Her2- and Her3-mediated signaling and induces apoptosis

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- Current antibody-based treatments do not cure advanced Her2+ tumors
- Our new MoA may help patients not adequately responding to current therapies

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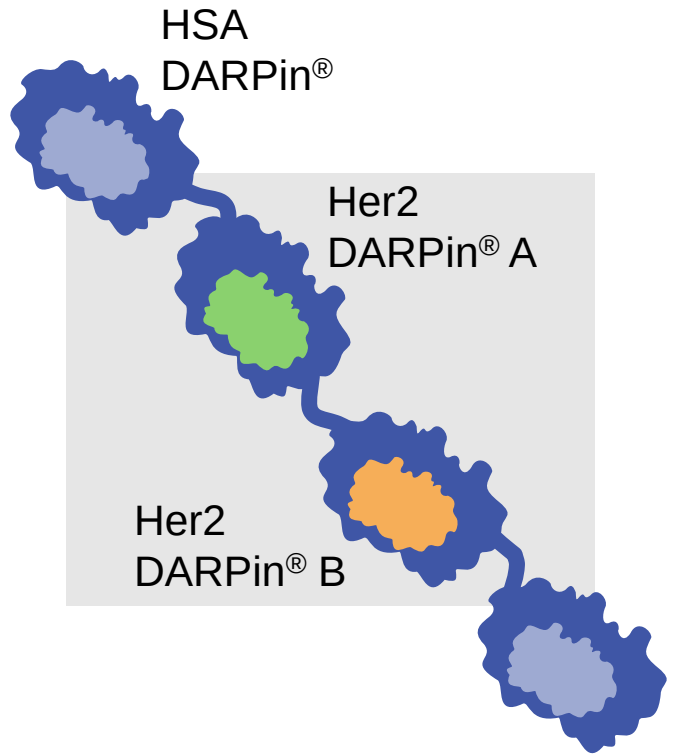
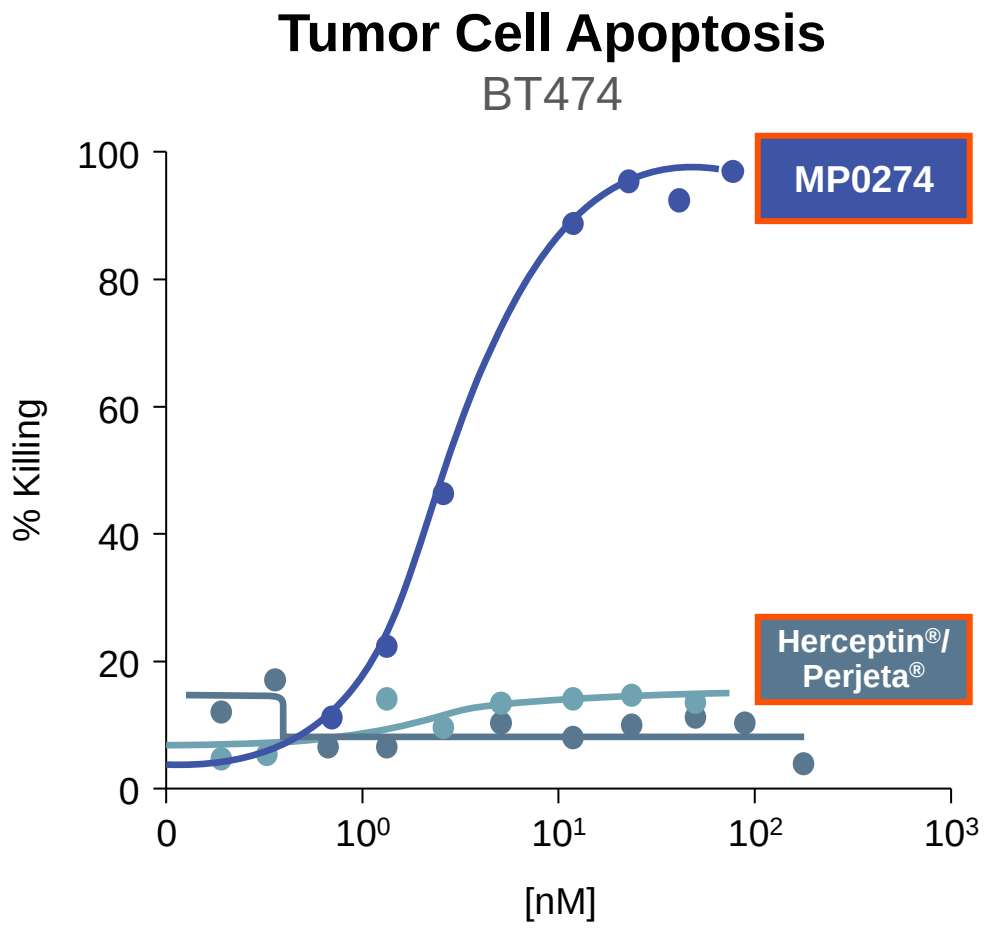
- Ongoing Phase 1 in Her2 positive tumor patients progressing on SOC

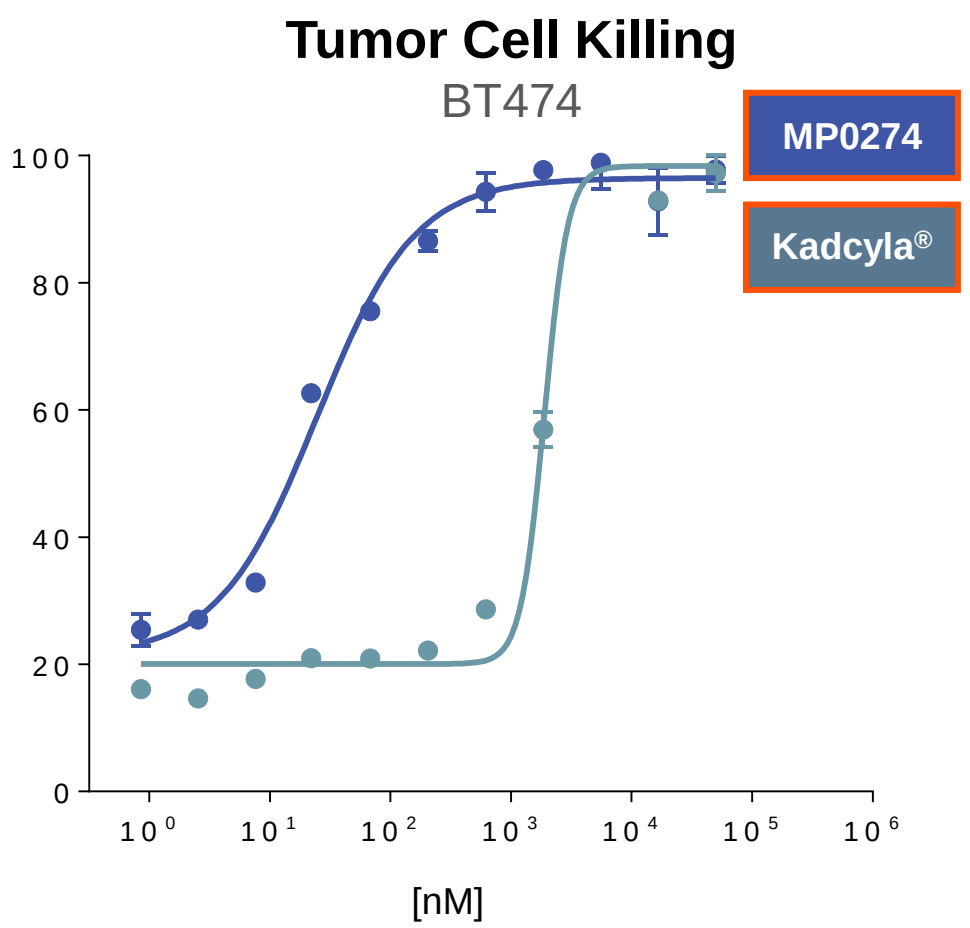
- Fully owned by Molecular Partners – IP protection until at least 2037



MP0274 is a Highly Potent Tumor Cell Killer

MP0274





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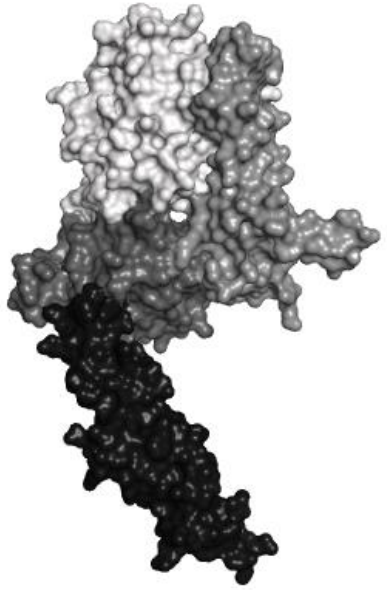
MP0274 Forces Her2 in Conformational Deadlock Leading to Cell Death

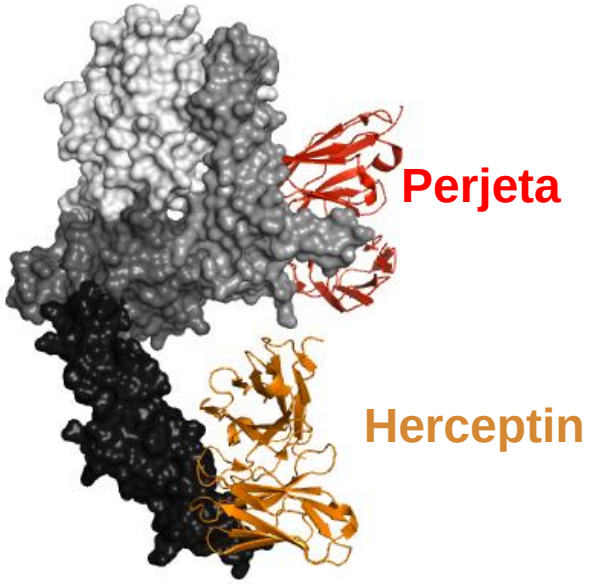
MP0274

Her2 EC-Domain



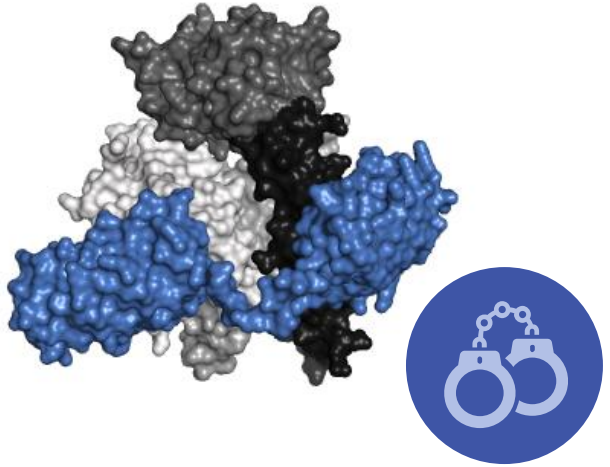
Her2 has several «active sites» with conformational flexibility

Trastuzumab & Pertuzumab



Herceptin and Perjeta block 2 distinct Her2 functions

MP0274 Bi-paratopic DARPin®

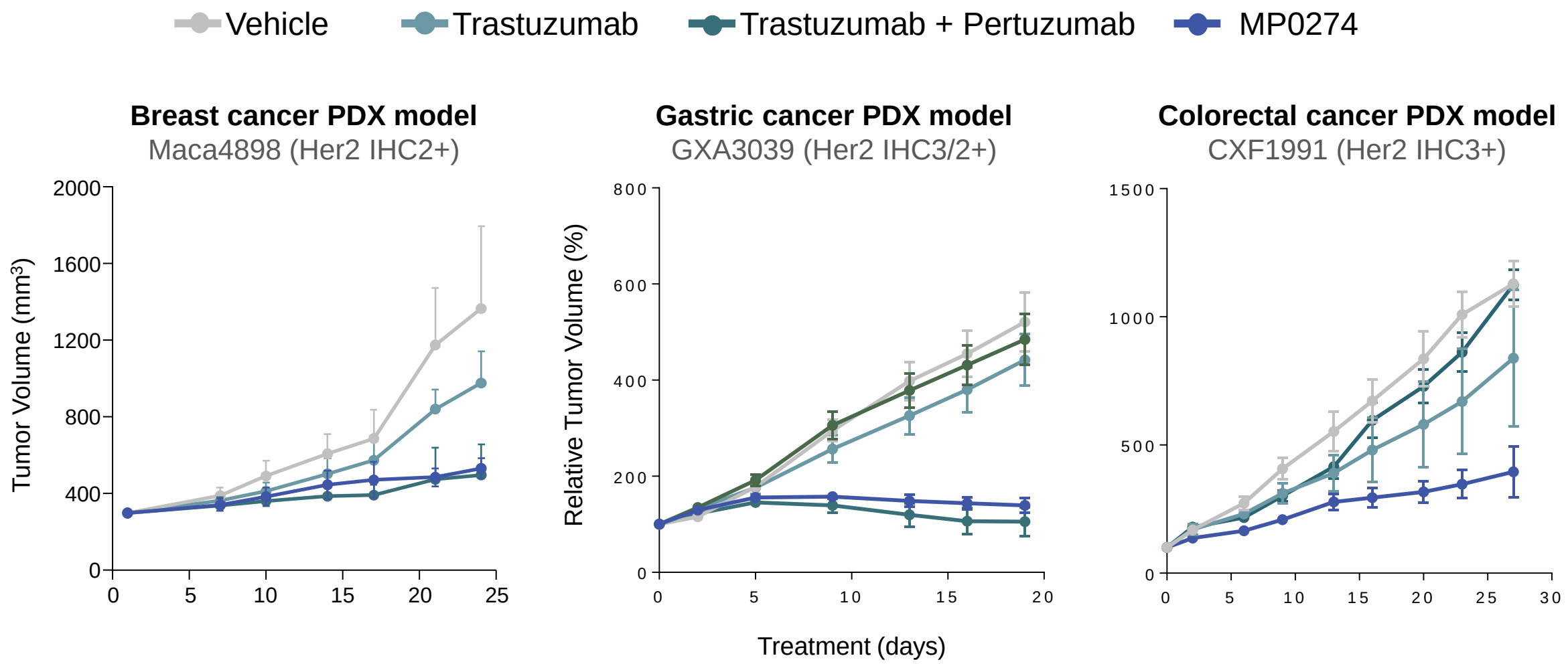


MP0274 handcuffs Her2 into a fully inactive conformation*, acting as a broad-range allosteric inhibitor

EC: extracellular; *model
* Model picture

MP0274 Efficacy in PDX Mouse Models

MP0274



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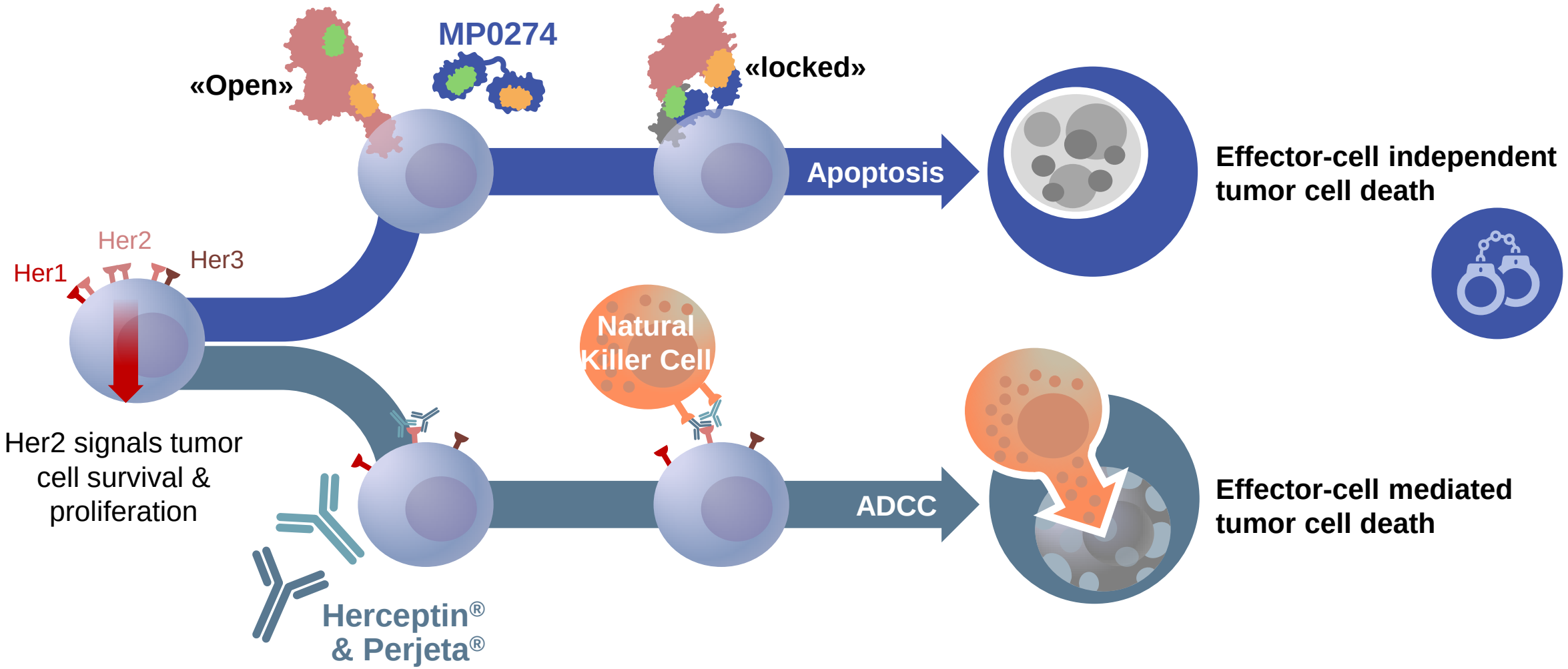
Direct Induction of Tumor Cell Death with MP0274

MP0274



ADCC, antibody-dependent cellular cytotoxicity

MP0274: Phase 1 Study in Her2+ Cancer Patients

MP0274

- Phase 1, first-in-human, single-arm, multi-center, open-label, repeated-dose, dose escalation study to assess safety, tolerability and pharmacokinetics of MP0274 in patients with advanced HER2-positive solid tumors with expansion cohort at recommended dose to confirm safety and to assess preliminary efficacy
- Primary objective: to assess the safety and tolerability of MP0274 in patients with HER2-positive solid tumors who have progressed after standard therapy for advanced disease
- Study treatment:
  - Dose Escalation (Part A): 4 dose groups (4 x 3-6 patients), starting dose 4 mg/kg q3wk
  - Dose Expansion (Part B): at recommended dose: 26 patients (total of up to 32 patients at target dose)
- Status: 3 sites open for recruitment (Germany, Switzerland, UK); 1st patients dosed; 2 additional sites planned for dose expansion
- Next readouts: initial safety data expected 1st half of 2018; initial efficacy data towards end of 2018
- Value proposition: completely new mode of action: induction of apoptosis in HER2 addicted cancer cells independent of ADCC compared to approved therapies

*Study details can be found on clinicaltrials.gov using the identifier: NCT03084926.

Clinical Perspective

Angelo Vacca, University of Bari
Kate Gold, UCSD
Richard Baird, University of Cambridge

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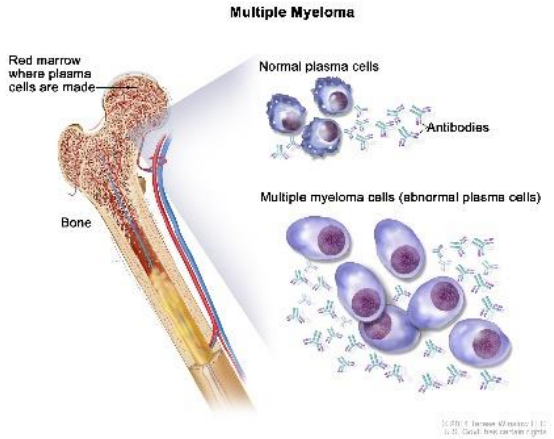
DARPIN® MP0250 IN MULTIPLE MYELOMA

Prof. Angelo Vacca
Clinica Medica "G. Baccelli"
Università degli Studi di Bari, Bari (Italy)

MYELOMA CLINICAL CHARACTERISTICS

- Cancer of the plasma cells.
- 10% of all hematological malignancies.[1]
- Myeloma is most frequent in people aged 65 to 74 (median 69) years.
- MM is sensitive to a variety of cytotoxic drugs, including newer proteasome inhibitors (Pis) and immunomodulating drugs (IMiDs) both as initial treatment and at relapse.
- Although responses are typically durable, MM is not curable with current approaches.
- 5-year survival rate: 40-50%.[2]

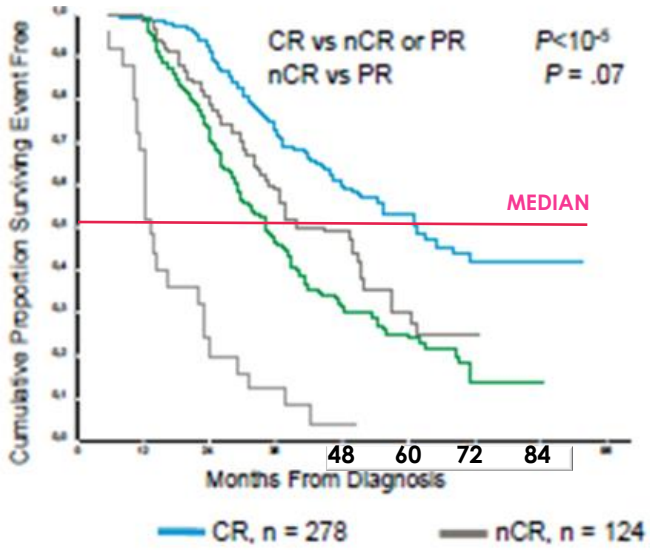
MM accounts for 1% of all cancer and 10% of all hematologic malignancies, with approximately 86,000 new cases of MM occurring annually worldwide.



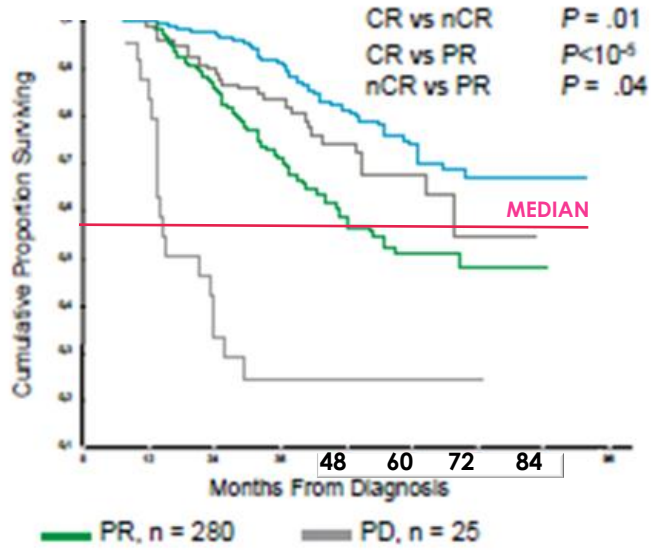
1) Moreau P et al. Ann Oncol. 2013 Oct;24 Suppl6:vi133-7. Steliarova-FoucherE et al. European Network of Cancer Registries, International Agency for Research on Cancer. Available fromhttp://eco.iarc.fr, accessed on 19/Nov/2015.
2) 2) Cancer Research UK, www.cancerresearchuk.org/health-professional/cancer-statistics/statistics-by-cancer-type/myeloma/survival/heading-Zero, Accessed 19/Nov/2015.

WHAT IS THE GOAL OF TREATMENT IN MM? COMPLETE RESPONSE AS A SURROGATE MARKER FOR SURVIVAL

EVENT-FREE SURVIVAL (EFS)



OVERALL SURVIVAL (OS)

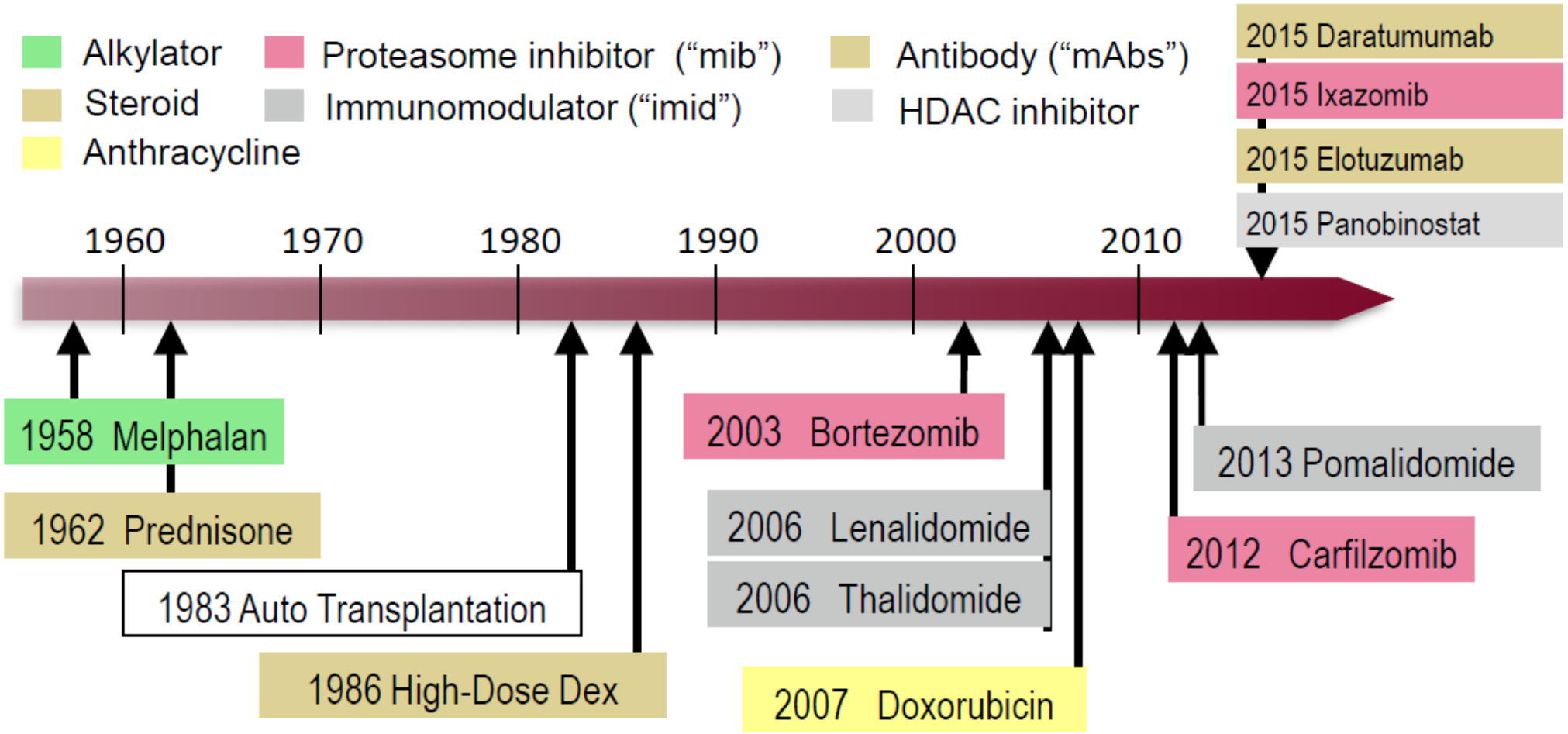


	CR	nCR	PR	PD
Median EFS, months	61	40	34	13
Median OS, months	NR	NR	61	15

CR = complete response
nCR = near complete response
PR = partial response

Lahuerta JJ, et al. J Clin Oncol. 2008;26:5775–82.

APPROVED TREATMENT OPTIONS 2017



Auto = autologous; Dex = dexametasone.

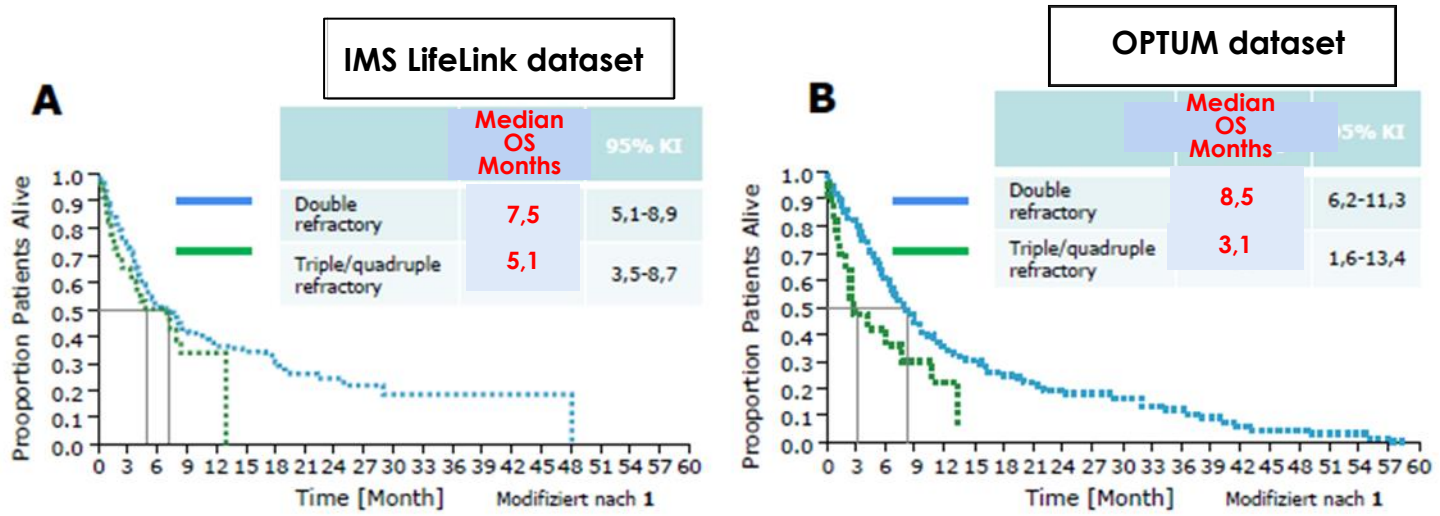
http://www.ema.europa.eu/ema/index. Diagram adapted from Munshi NC.Hematology2008:297.

UNMET NEEDS IN MULTIPLE MYELOMA

Overall Survival in Double and Triple/Quadruple Refractory Patients and Patients with ≥3 Prior Lines of Therapy (Including a PI and an IMiD)

Recent review of real world data found outcomes remain poor for patients with relapsed MM despite the approval of carfilzomib and pomalidomide

Median OS for triple/quadruple refractory patients was just 154 days (~5 months)

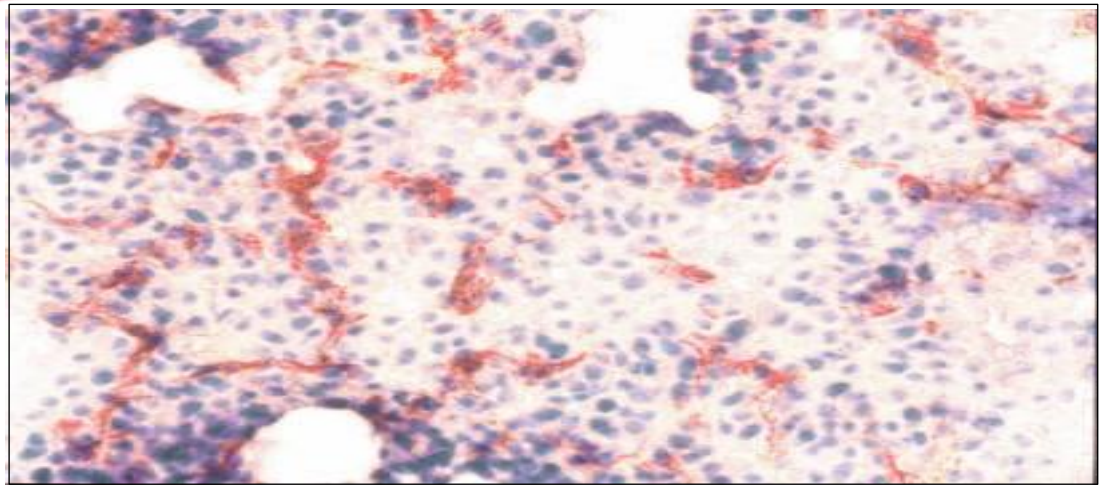


Usmani iS. et al. ASH, 2015; Abstract 4498.

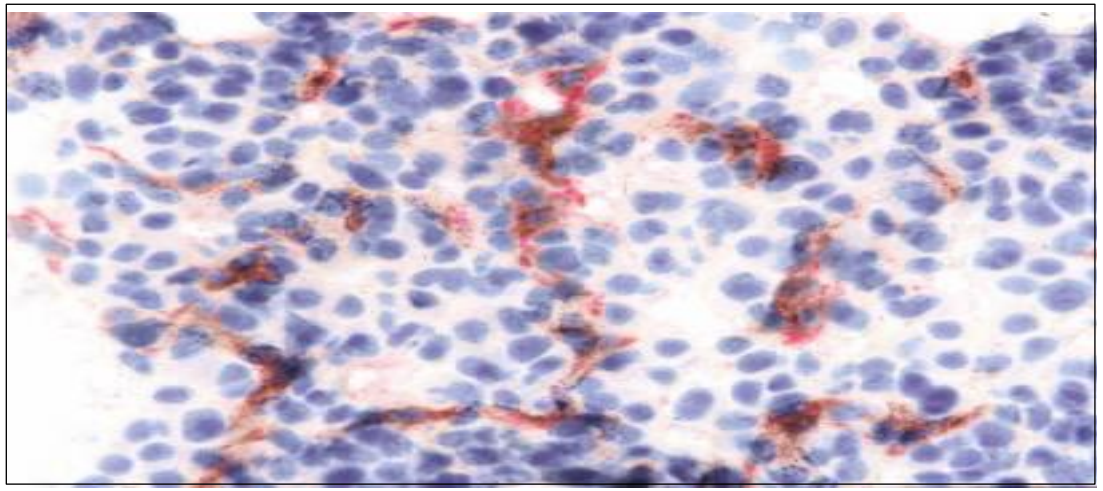
PI = proteasome inhibitor
IMiD = immunomodulatory drug

BONE MARROW ANGIOGENESIS IN PATIENTS WITH ACTIVE MULTIPLE MYELOMA

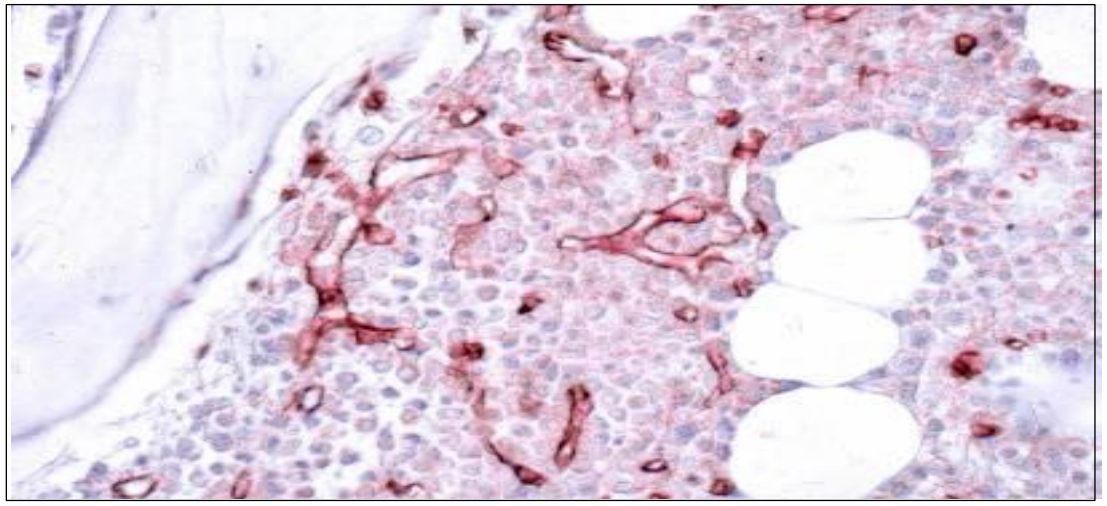
Megafield



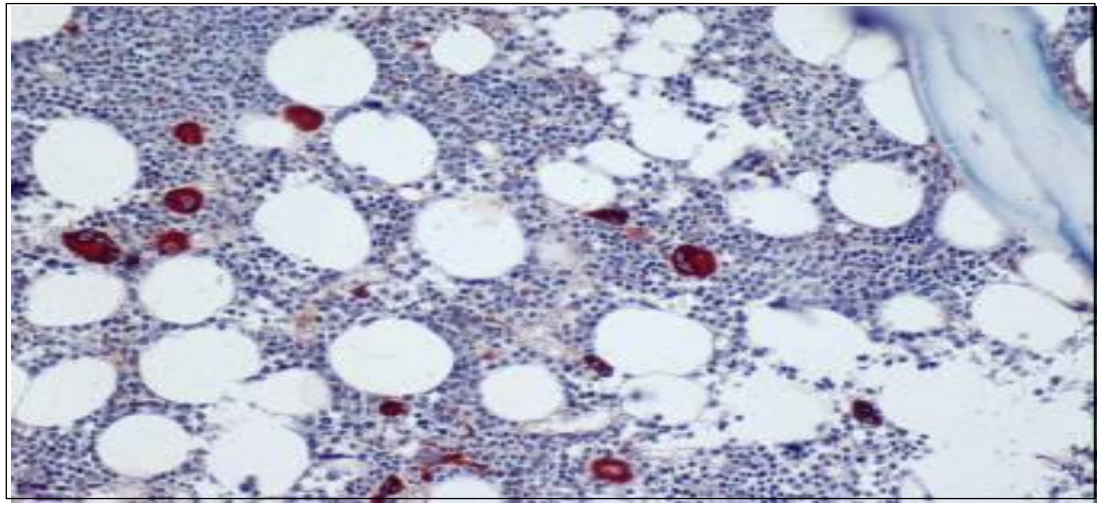
Single or clustered endothelial cells



Vessel arborizations

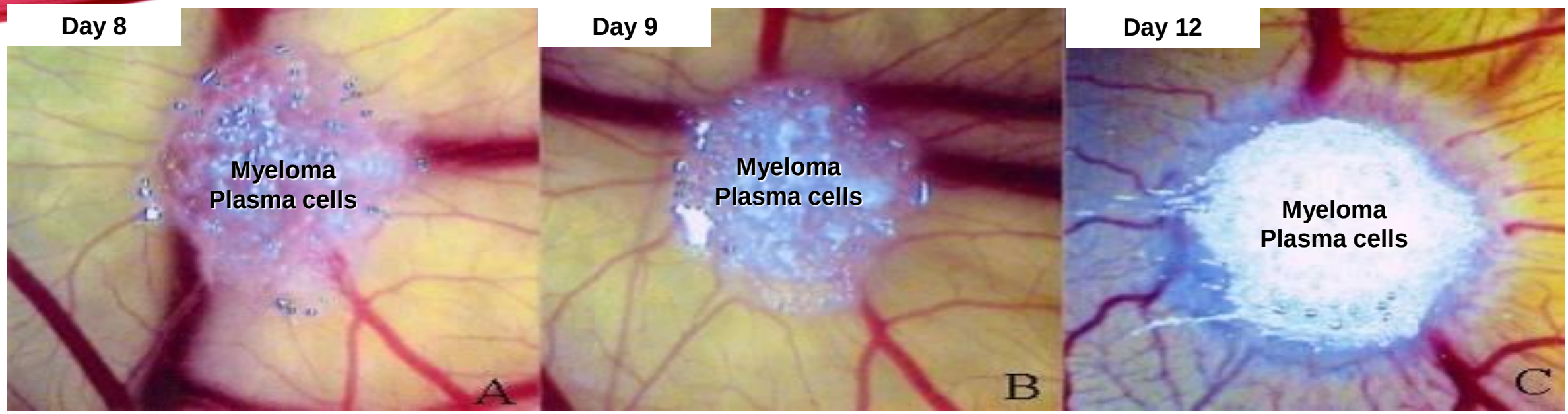


MGUS: no vessels



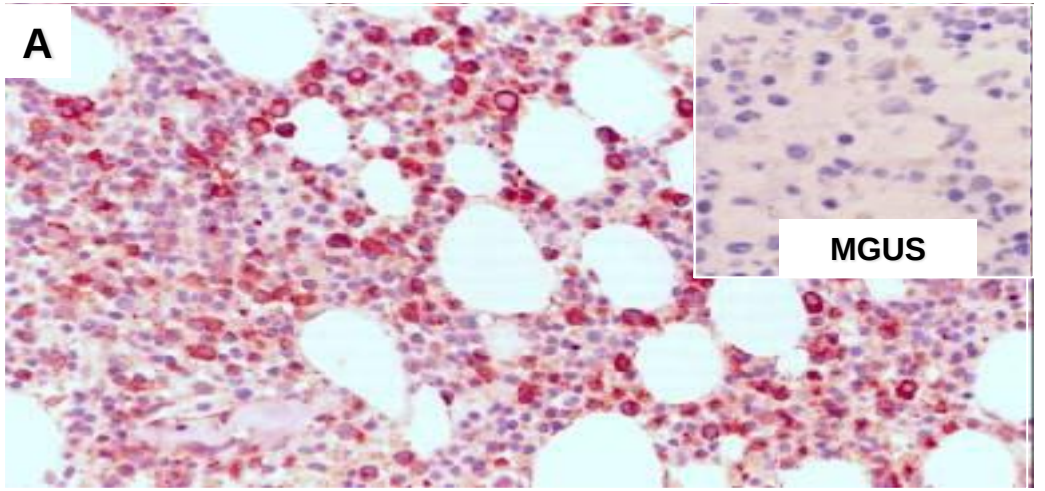
Vacca et al, Br J Haematol 1994

TIME-COURSE OF ANGIOGENESIS INDUCTION BY MYELOMA PLASMA CELLS IN THE *IN VIVO* CAM-SPONGE ASSAY

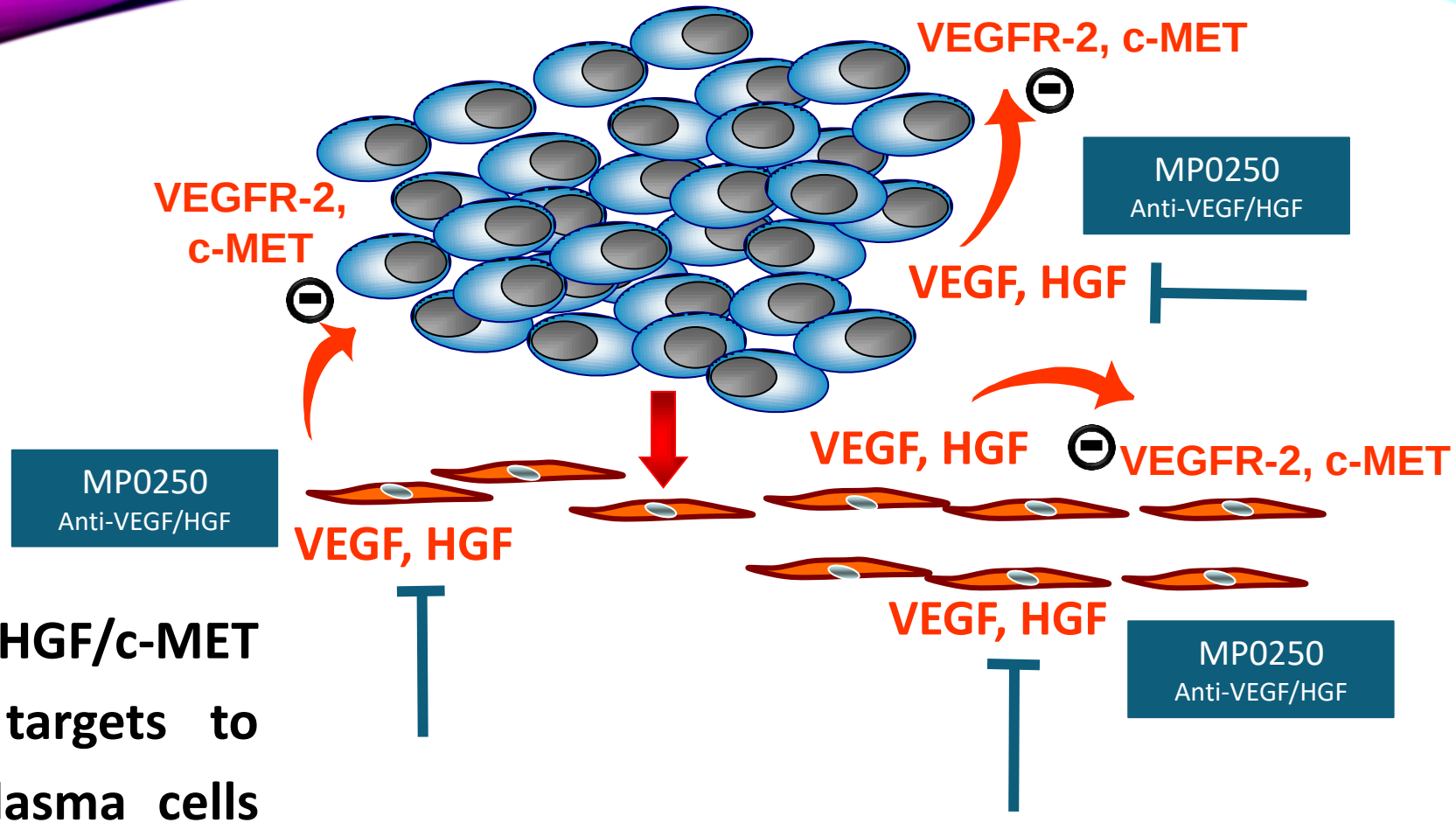


Transformation into a myeloma plasma cells results in aberrant or increase in angiogenic factors **(VEGF and HGF)** promoting bone marrow angiogenesis.

VEGF-A+ myeloma plasma cells



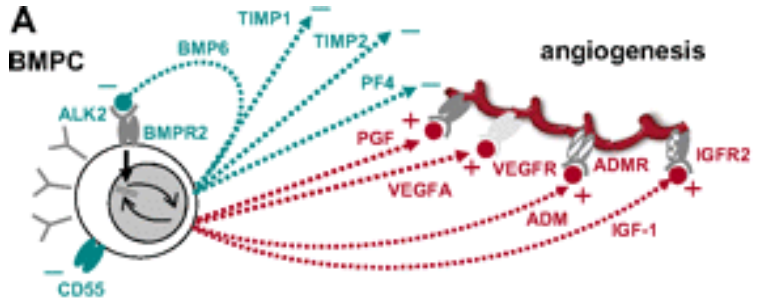
Vacca et al. Blood 93: 3064-73, 1999; Ribatti - Vacca. Leukemia 21: 44-52, 2007;
Hose et al. Blood 114:128-143, 2009b

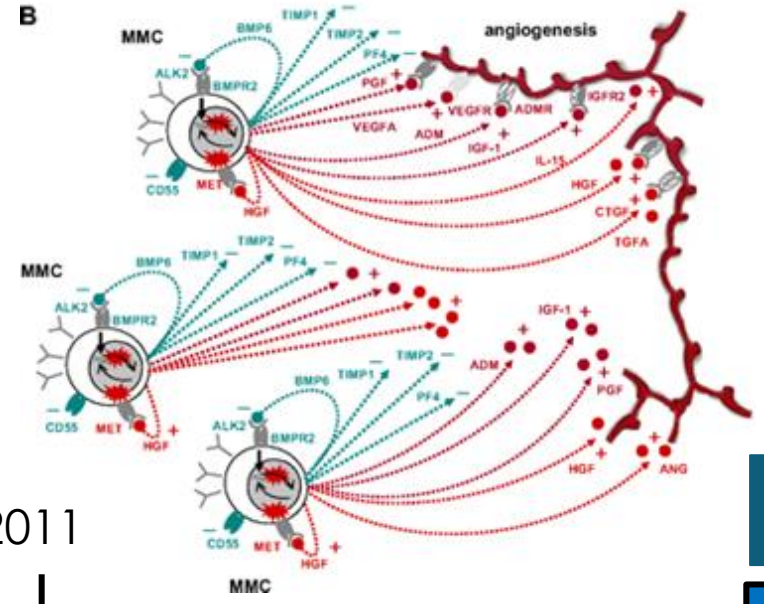


VEGF/VEGFR-2 and HGF/c-MET pathways are appealing targets to simultaneously hit MM plasma cells and endothelial cells: MP0250 as the newest drug

[1]Rampa et al. 2014; [2]Ferrucci et al. 2014; [3]Gambella et al. 2015; [4]Ria et al. 2011; [5]Derksen et al. 2003; [6]Moschetta et al. 2013

ONE ANTIANGIOGENIC COMPOUND FITS ALL





1980 — Discovery — VEGF

2006 — Solid Tumours — Bevacizumab With or Without Thalidomide in Patients With Relapsed/ Resistant MM

2009 — HGF

2011 — Multiple Myeloma — Bevacizumab + Bortezomib Bevacizumab + Lenalidomide In Relapsed/ Resistant MM

MP0250
Anti-VEGF/HGF

VEGF and HGF

- **Inhibition of angiogenesis in BM constrains plasma cell expansion**
- **Inhibition of plasma cell adhesion to BM makes MM cells sensitive to antitumor agents and inhibits survival**
- **Stabilization of bone homeostasis constrains tumor survival and reduces bone pain**
- **Inhibition of plasma cell migration and growth**

Hose et al. Blood 114:128-143, 2009b.

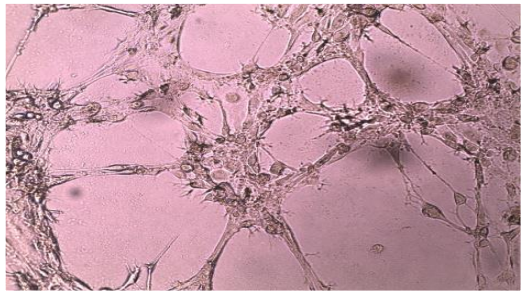
Prof. K. Vanderkerken, Dr. K. Vermeil, Brussels

Drs. L. Rao, D. Giannico, my lab

SIMULTANEOUS INHIBITION OF BOTH VEGFR-2 AND c-MET AS THE STRONGEST ANTIANGIOGENIC SYSTEM IN MYELOMA

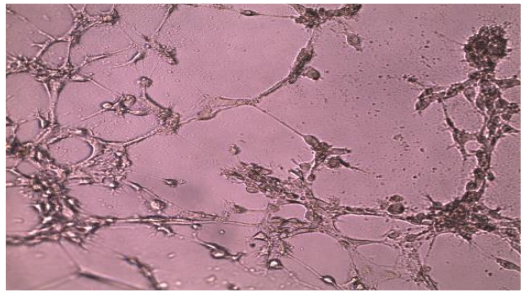
How to overcome drug resistance in patients with multiple myeloma ?

SFM (serum free medium)



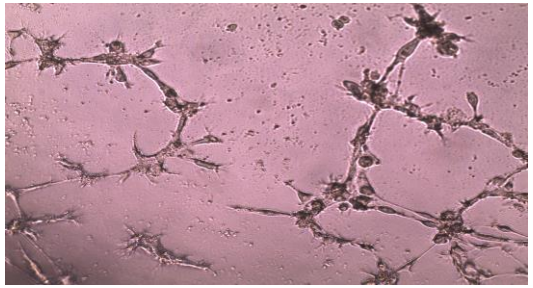
Vessels n.° = 26 ± 6

bortezomib 7.5 nM



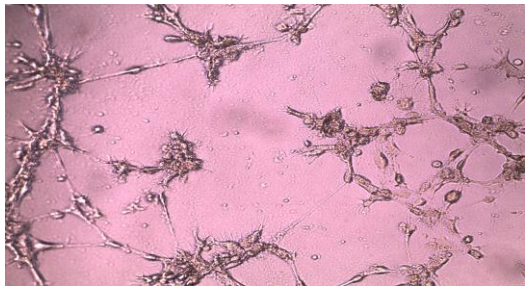
n.° = 16 ± 4

lenalidomide 1.75 µM



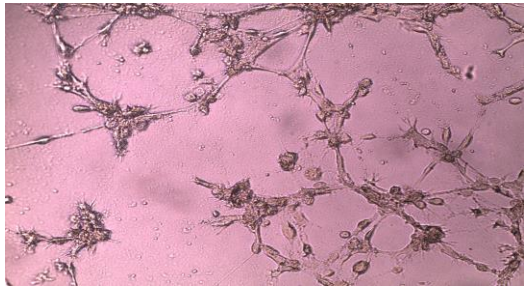
n.° = 19± 5

axitinib 100 nM



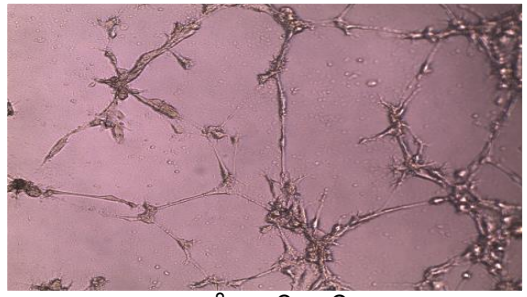
n.° = 11 ± 3

SU11274 10 µM



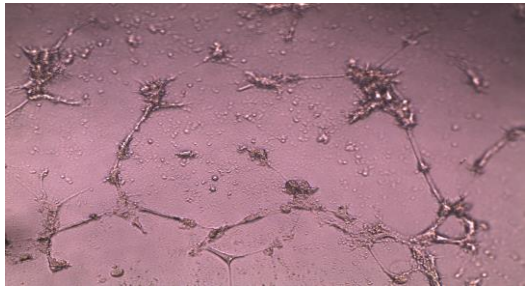
n.° = 12 ± 4

SU11274 10 µM
+ bortezomib 7.5 nM



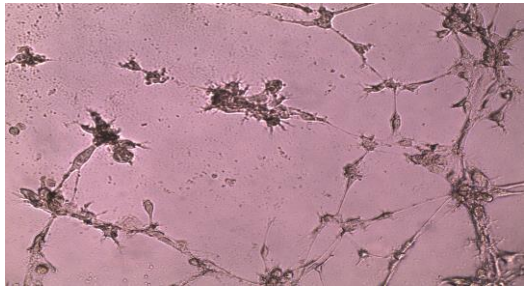
n.° = 8 ± 2

SU11274 10 µM
+ lenalidomide 1.75 µM



n.° = 9± 2

SU11274 10 µM
+ axitinib 100 nM

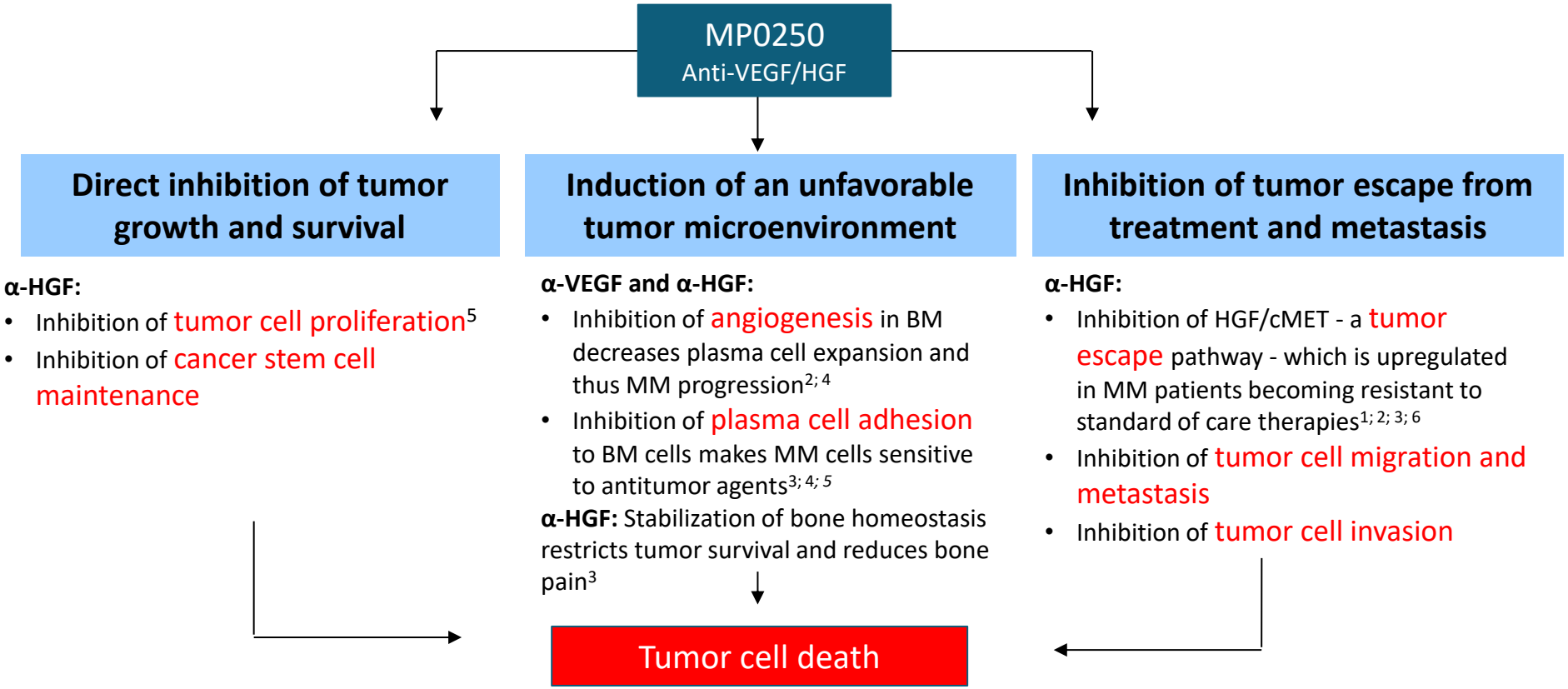


n.° = 4 ± 1

Roccaro A. M. (2006) Cancer Research, 66(1), 184–191; Moschetta M. (2010) Eur J Cancer. 46(2):420-9; De Luisi A. (2011) Clin Cancer Res, 17(7):1935-4.

MP0250: MECHANISM OF ACTION IN MULTIPLE MYELOMA

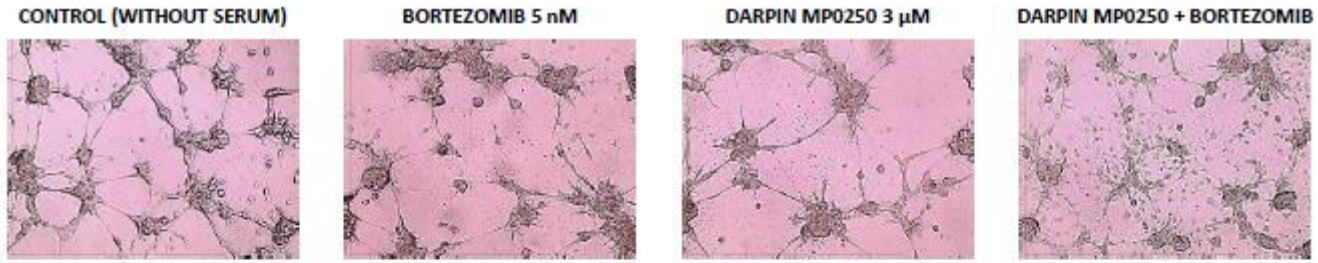
MP0250 induces tumor cell death by multiple direct and indirect mechanisms of action via blocking VEGF and HGF and downstream VEGFR-2 and c-MET

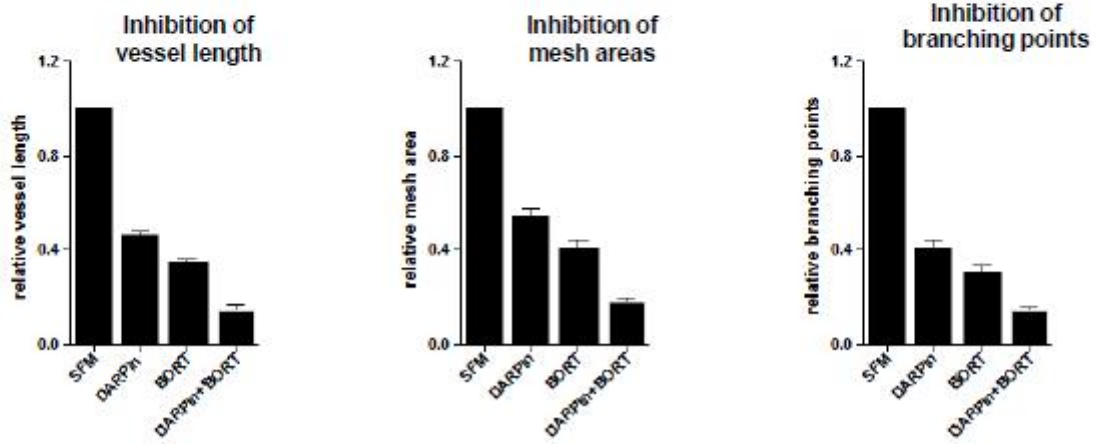


[1]Rampa et al. 2014; [2]Ferrucci et al. 2014; [3]Gambella et al. 2015; [4]Ria et al. 2011; [5]Derksen et al. 2003; [6]Moschetta et al. 2013

HALTING VEGF/ VEGFR-2 AND HGF/ c-MET PATHWAYS AS AN ATTRACTIVE THERAPY IN MYELOMA

Combination of VEGFR-2 and c-MET targeting induces the strongest antiangiogenic effect in myeloma and may impair several activities including: chemotaxis, motility, adhesion, spreading, and whole angiogenesis in bone marrow endothelial cells significantly when combined with proteasome inhibitors or immunomodulating drugs

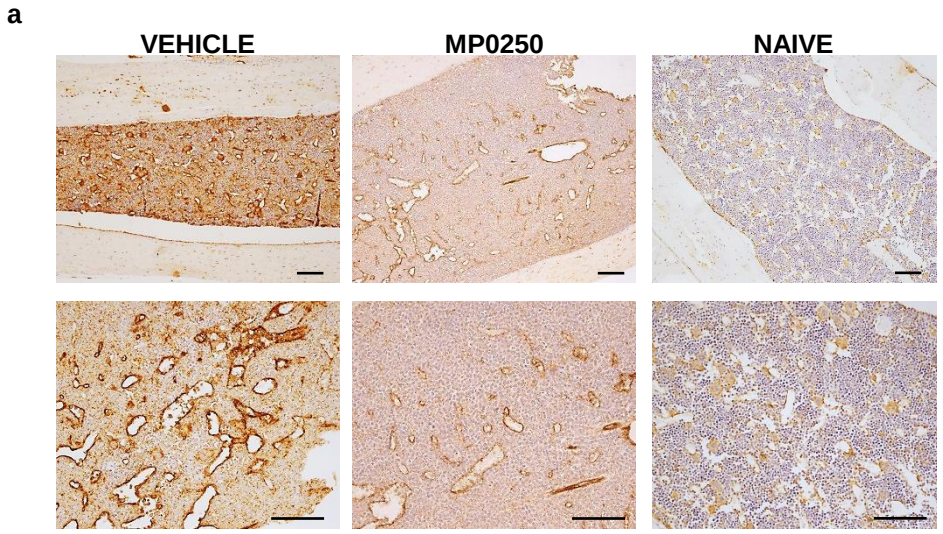


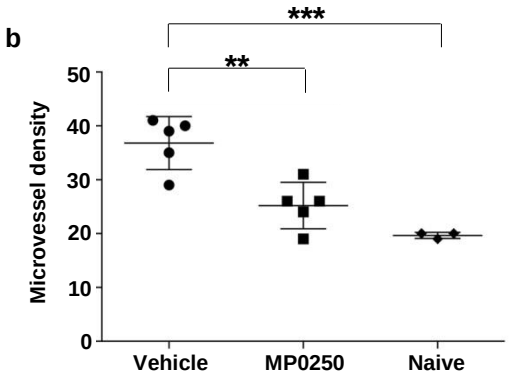


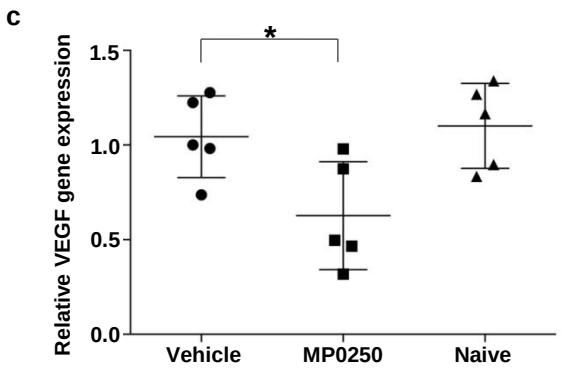
Rao – Vacca. Submitted

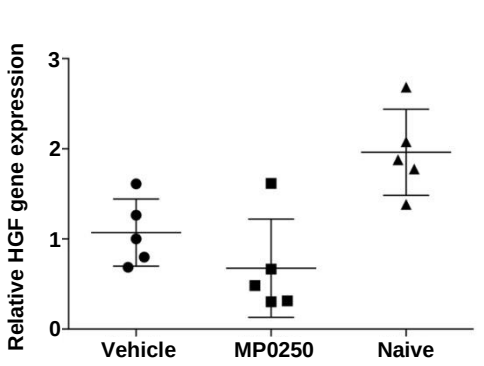
MP0250 IS A STRONG ANTIANGIOGENIC DRUG IN SYNGENEIC MOUSE MYELOMA MODEL

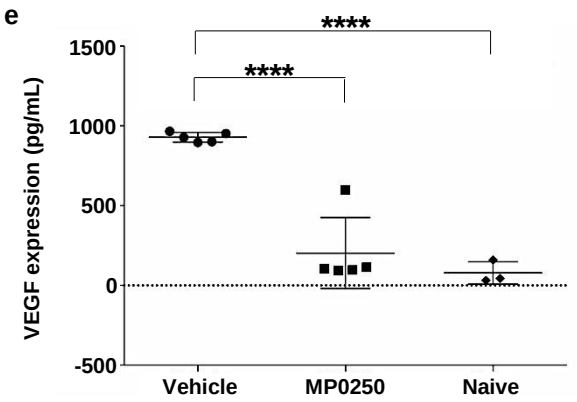
Mice were inoculated with 5T33MM cells (day 0). Starting from day 1, mice were treated with MP0250, 4 mg/kg, every third day, i.p. Evaluation at day 21.

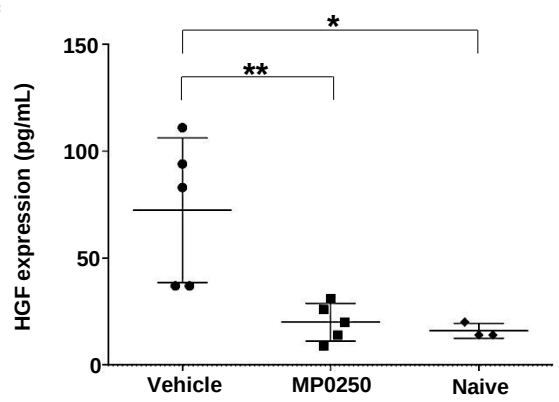








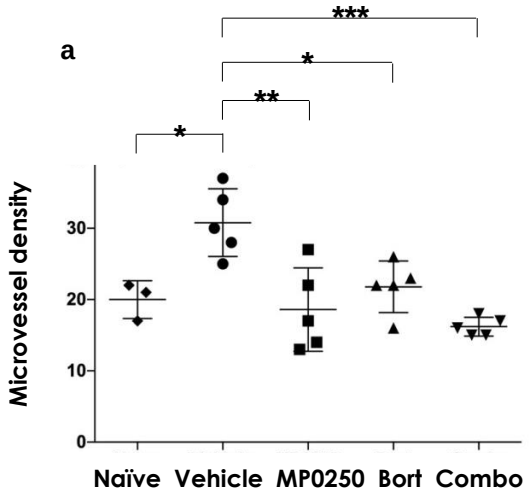


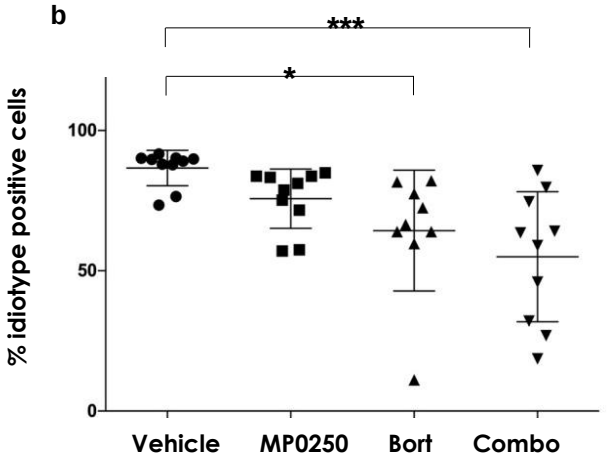


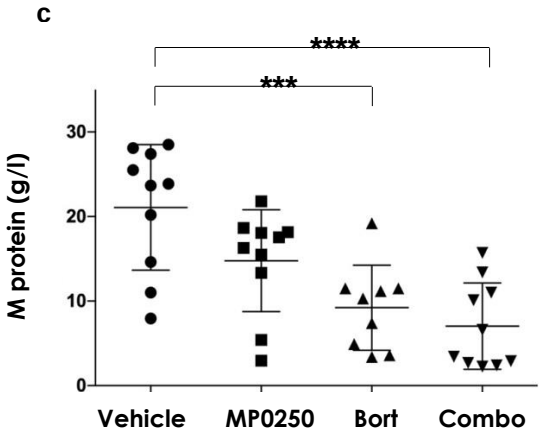
Rao - Vacca. Submitted

MP0250 AND BORTEZOMIB AFFECT BOTH VESSELS AND TUMOR IN SYNGENEIC MOUSE MYELOMA MODEL

Mice were inoculated with 5T33MM cells (day 0). Starting from day 1, mice were treated with MP0250, 4 mg/kg, every third day, i.p., bortezomib 0.6 mg/kg, twice a week, s.c., or with the combination (Combo). Evaluation at day 21.







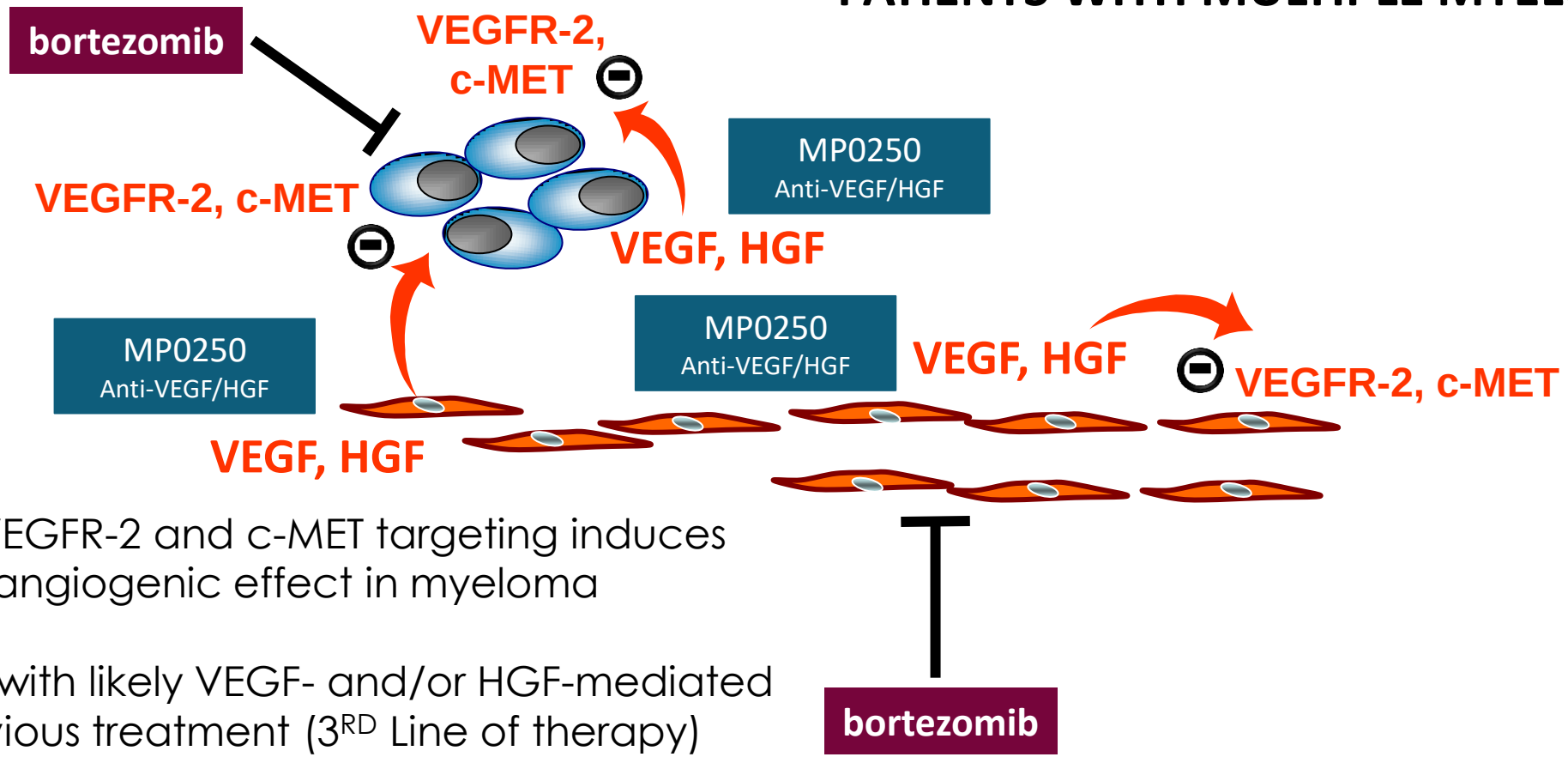
d

Vehicle vs	MP0250	Bort	Combo
MVD	0.0016	0.0187	0.0002
Idiotype+ cells	ns	0.0323	0.0010
M protein	ns	0.0007	<0.0001

Rao - Vacca. Submitted

CONCLUSION

MP0250 PLUS BORTEZOMIB - A TWO FOLD DUAL TARGETING IN PATIENTS WITH MULTIPLE MYELOMA



- Combination of VEGFR-2 and c-MET targeting induces the strongest antiangiogenic effect in myeloma
- Ideal for patients with likely VEGF- and/or HGF-mediated escape from previous treatment (3RD Line of therapy)
- In Relapsed/ Resistant MM, MP0250 can be combined with standard therapy

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DEGLI STUDI DI BARI
ALDO MORO

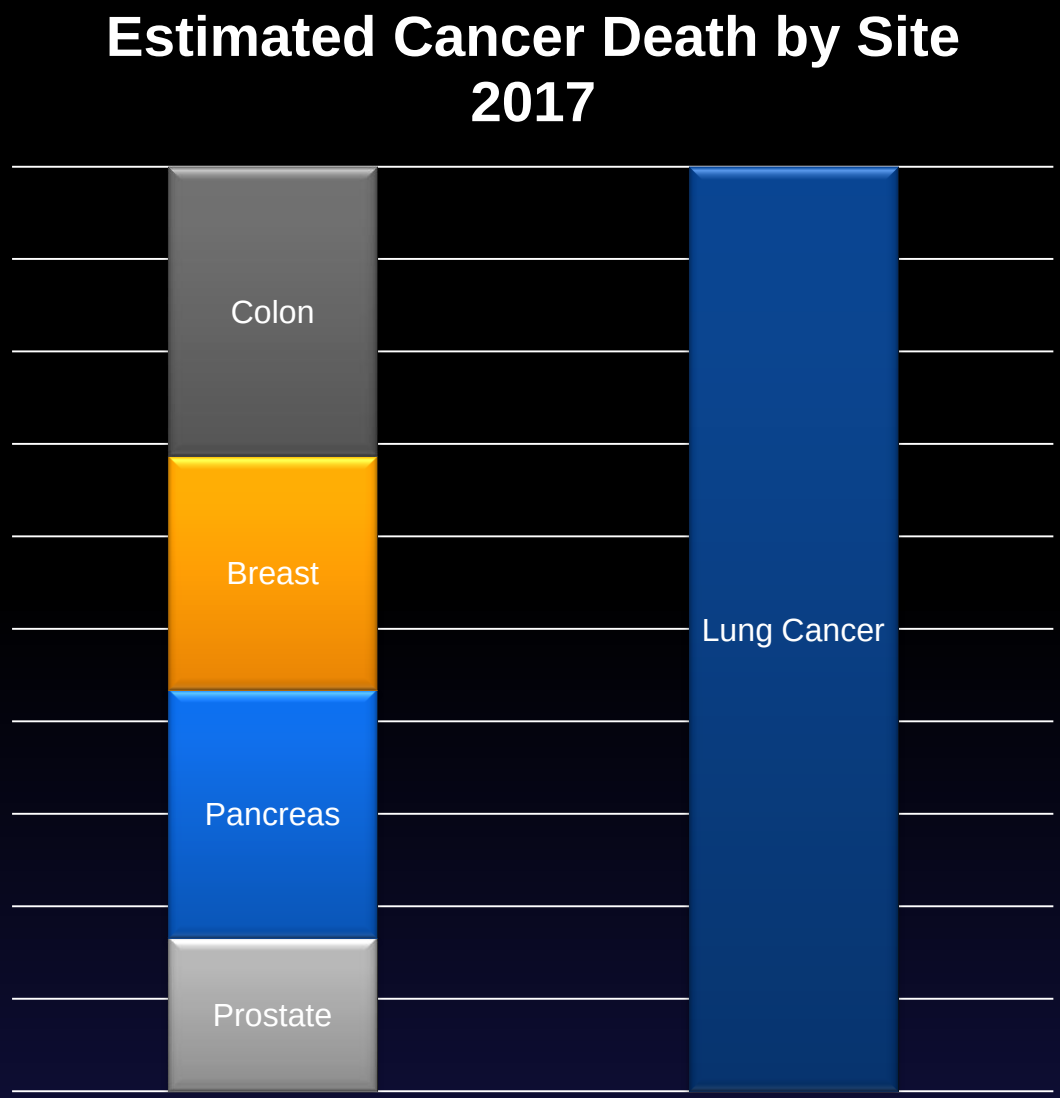
Thank you

MP0250 in Non-Small Cell Lung Cancer

Kate Gold, MD
University of California, San Diego

Introduction to Lung Cancer: Incidence and Mortality

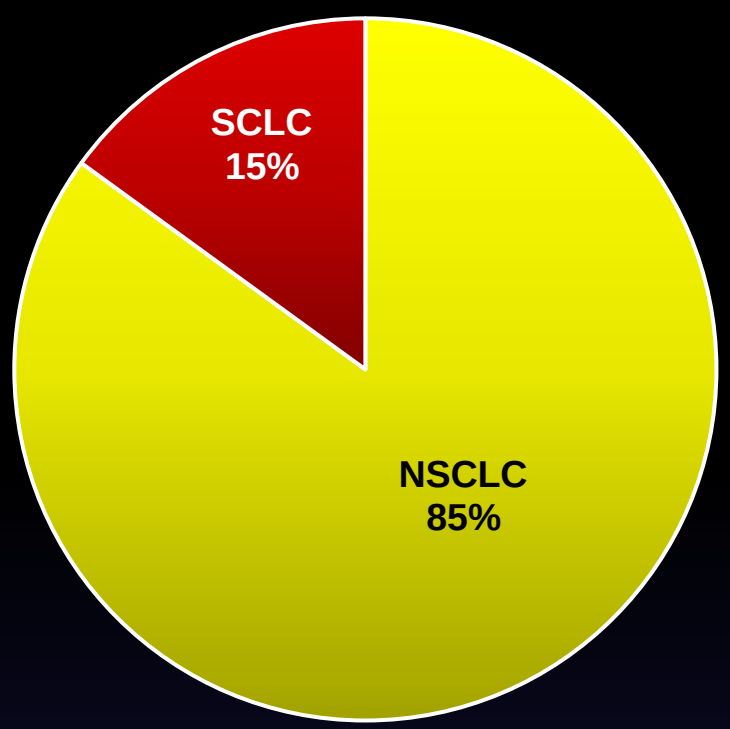
- New cases in 2017:
  222,500
  - 60% with stage IV disease
    at presentation
- ~ 155,870 deaths in 2017, comparable to prostate, breast, colon and rectum cancer and pancreas *combined*
- 5-year relative survival rate:
  4.5% for patients with distant-stage disease



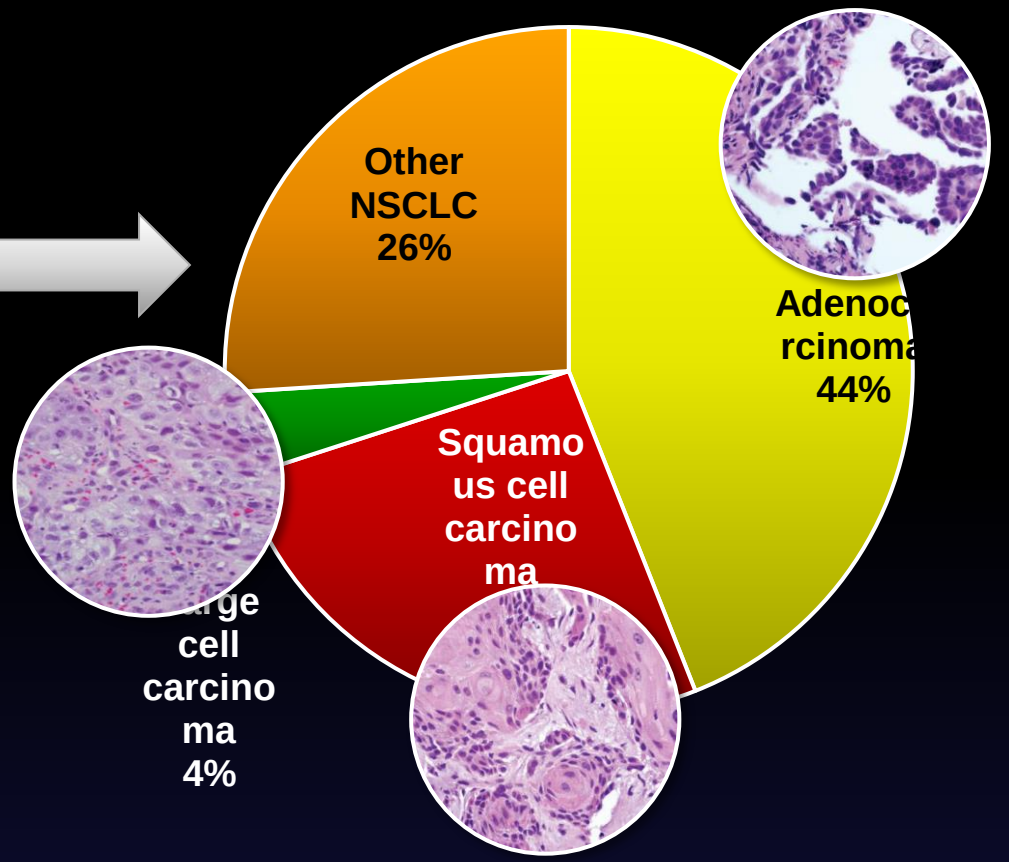
NCI. Non small-cell lung cancer treatment (PDQ®). SEER Cancer Statistics Review https://seer.cancer.gov/statfacts/html/lungb.html)

Introduction to NSCLC

NSCLC accounts for 85% of all lung cancers[1]

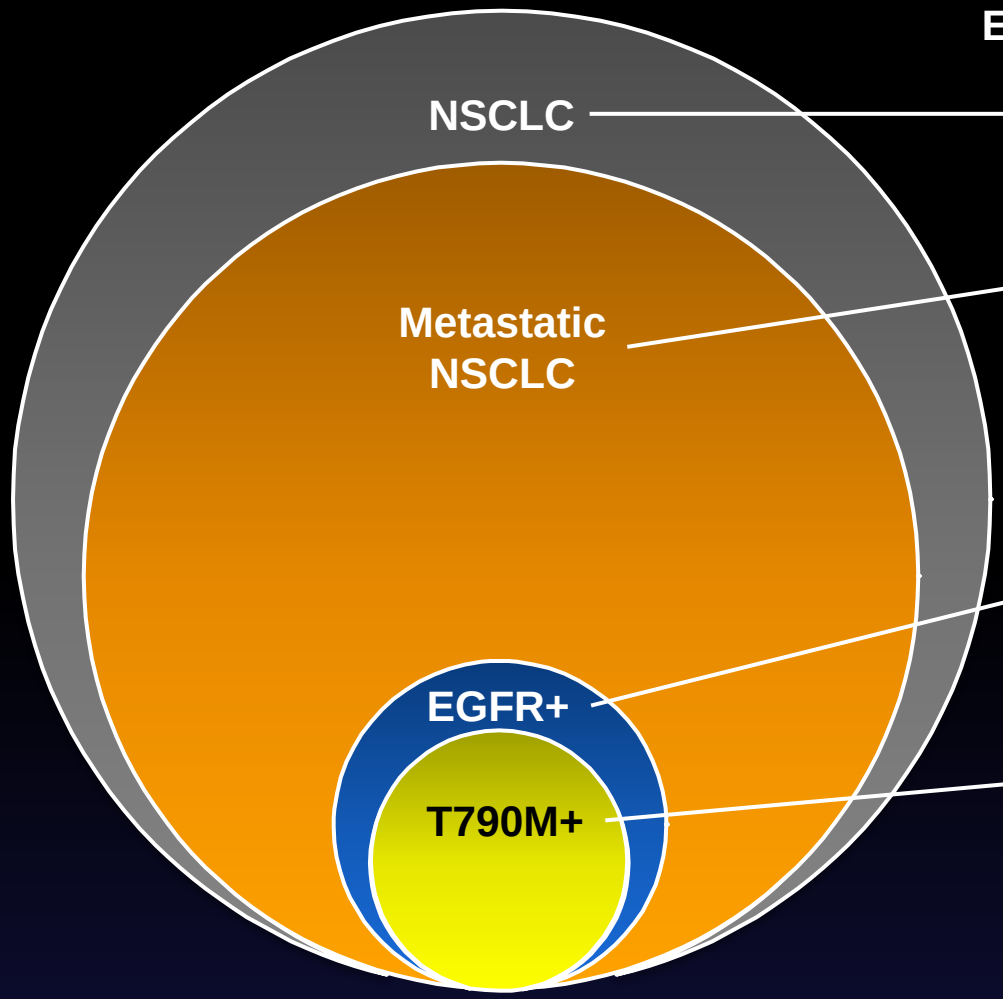


Histologic classification of NSCLC[1,2]



1. Houston KA et al. *Lung Cancer.* 2014;86:22–28; 2. Travis WD et al. *Arch Pathol Lab Med.* 2013;137:668–684.

EGFR Mutation is an Important Cause of Lung Cancer



Estimated incidence in USA

NSCLC → ~220,000 cases[1]

>80% of all NSCLC[2]

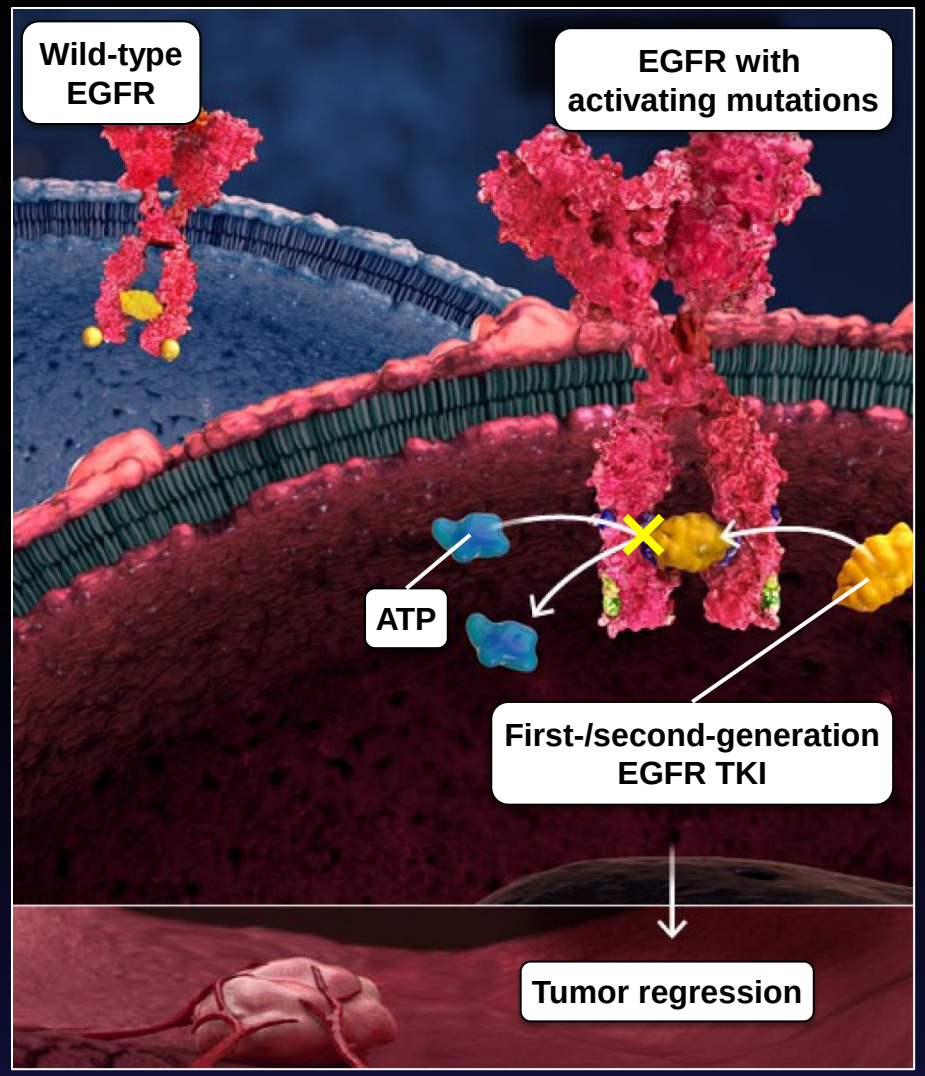
15-30% of all metastatic cases[3]

T790M+ cases 60% of all EGFR+[4,5]

Estimated incidence in EU5, Japan, USA
~513,700 cases[7]

1. Siegel et al, Ca Cancer J Clin 2017;66:7–30 ; 2. Jemal A, Bray F, Center MM et al. Global cancer statistics. CA Cancer J Clin 2011: 61: 69–90;
3. Gerber DE et al. *Am Soc Clin Oncol Educ Book.* 2014:e353–e365; 4. Yu HA et al. *Clin Cancer Res.* 2013;19:2240–2247;
5. Cortot AB, Jänne PA. *Eur Respir Rev.* 2014;23:356–366; 6. Yu HA et al Ann Oncol. 2014;25(2):423-428.; 7 Datamonitor Health Data 2015 accessed 23rd October 2017

EGFR TKIs are the Standard Front-line Treatment in EGFR Mutant NSCLC

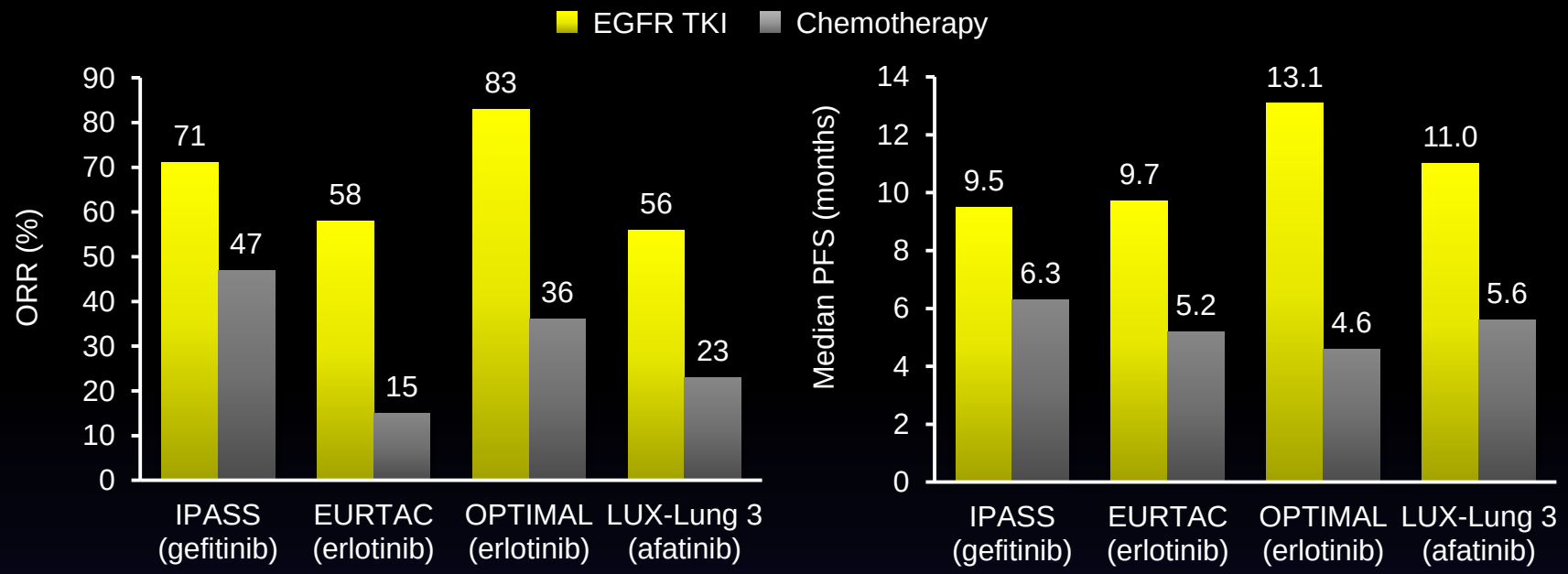


- EGFR TKIs are competitive ATP inhibitors that bind to the kinase domain of EGFR
  - First-generation: reversible binding (gefitinib, erlotinib)
  - Second-generation: irreversible binding (afatinib)
- Both first- and second-generation EGFR TKIs inhibit both wild-type and mutant EGFR

Peters S et al. *Cancer Treat Rev.* 2014;40:917–926.

EGFR TKIs are Standard Front-line Treatment in EGFR Mutant NSCLC

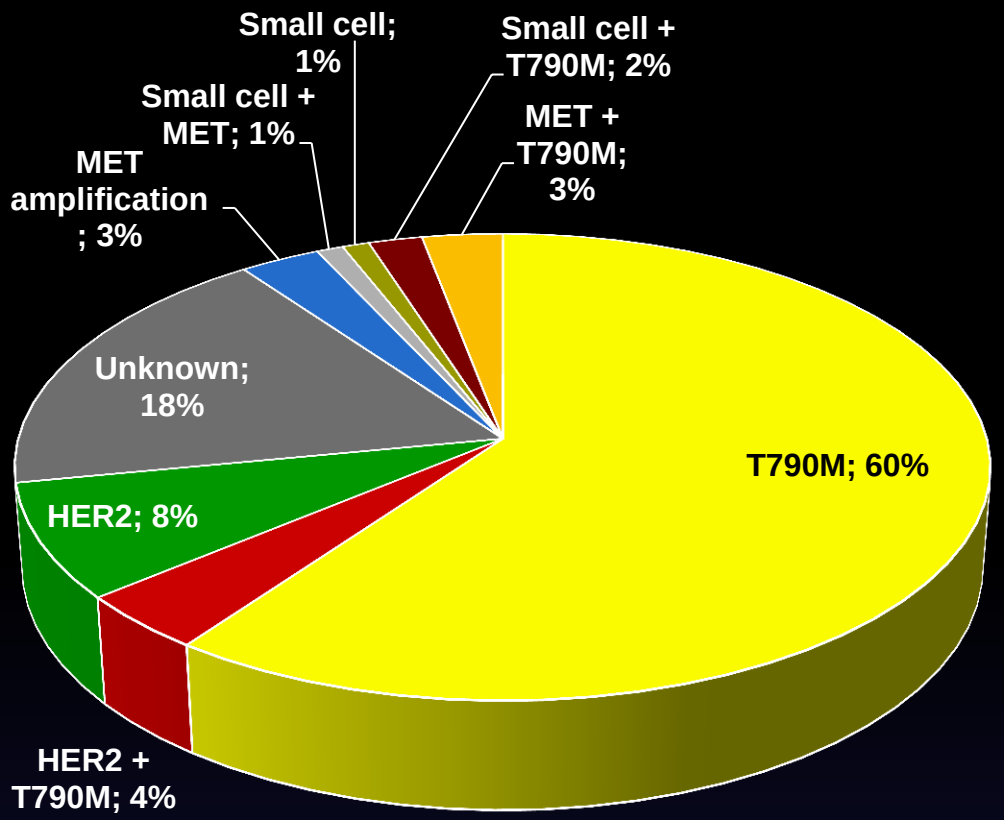
Phase 3 studies of first- and second-generation EGFR TKIs vs chemotherapy as first-line treatment in EGFR mutant patients[1,2]



Long-term efficacy of first- and second-generation EGFR TKIs is limited by emergence of resistance

1. Wang Y et al. *Ther Adv Med Oncol.* 2012;4:19–29; 2. Engle JA, Kolesar JM. *Am J Health Syst Pharm.* 2014;71:1933–1938.

T790M is the Dominant Cause of Acquired Resistance to First- and Second Generation EGFR TKIs



- The vast majority of patients with activating EGFR mutations initially respond to EGFR TKI treatment
- However, acquired resistance to therapy inevitably develops
- Approximately two-thirds of those patients develop resistance due to the emergence of the T790M secondary EGFR mutation

Blakely CM, Bivona TG. *Cancer Discov* 2012;2:872–875. Yu HA, *et al. Clin Cancer Res* 2013;19:2240–2247.

Osimertinib – a Third Gen. TKI

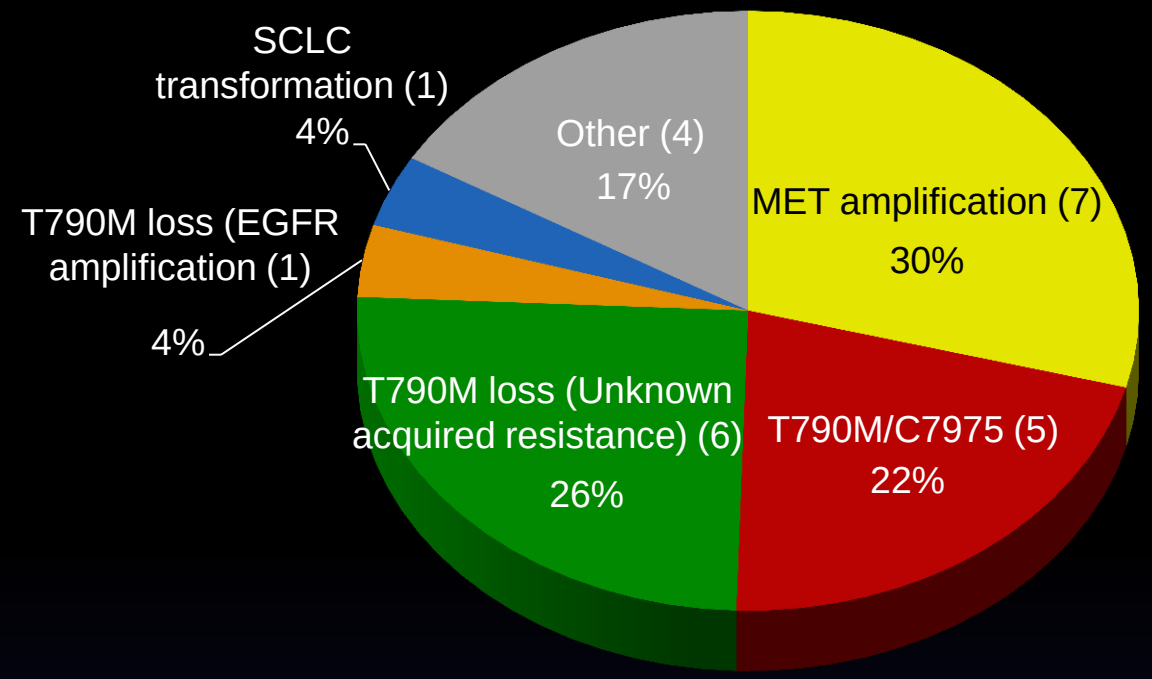
- Osimertinib is a third-generation, CNS-active EGFR-TKI that potently and selectively inhibits both EGFRm and T790m resistance mutations[1-4]
- Osimertinib is currently approved for the treatment of patients with advanced NSCLC and T790-mediated acquired resistance[5]
- Preclinical and recent clinical data suggest osimertinib to be an effective first-line therapy for EGFRm advanced NSCLC[3,6-8]

1. Yang et al. J Clin Oncol 2017;35:1288–96. 2. Ballard et al. ClinCancer Res 2016;22:5130–40; 3. Cross et al. Cancer Discov2014;4:1046–61; 4. Mok et al. J ClinOncol2017;35(suppl); abstr9005; 5. FDA https://www.accessdata.fda.gov/drugsatfda_docs/label/2015/208065s000lbl.pdf (accessed october 9th 2017). 6. Ramalingam et al. J ClinOncol 2017:JCO.2017.74.7576; 7. Eberleinet al. Cancer Res 2015;75:2489–500; 8. Ramalingam et al. ESMO 2017 (abstract LBA2)

HGF/c-Met - Key Escape Pathway of EGFR TKI Treatment in NSCLC

HGF/c-Met key pathway of resistance in NSCLC

- Treatment with EGFR TKIs lead to resistance
  - T790M mutation accounts for ~60% resistance[1]
  - HGF/c-Met amplification accounts for 5 -10% of resistance[1]
- Treatment with T790M EGFR TKIs further increases HGF/c-Met resistance pathway
  - HGF/c-Met amplification is the most frequent observed resistance mechanism to T790M EGFR TKI[2]



Main mechanisms of acquired resistance to EGFR TKIs in EGFR-mutant NSCLC[3]
Met amplification share expected to further increase after T790M EGFR TKI treatment.[4]

1 Cross et al., 2014, Ohashi et al., 2013; 2 Chabon et al, 2016; 3 Alexis B. Cortot, and Pasi A. Jänne Eur Respir Rev 2014;23:356-366;; 4 Piotrowska Z et al., ASCO 2017, abstract 9020

IASLC
INTERNATIONAL ASSOCIATION FOR THE STUDY OF LUNG CANCER
IASLC 18TH WORLD CONFERENCE ON LUNG CANCER
October 15–18, 2017 | Yokohama, Japan
WWW.IASLC.ORG

TATTON Phase Ib expansion cohort: osimertinib plus savolitinib for patients with *EGFR*-mutant *MET*-amplified NSCLC after progression on prior EGFR-TKI

Myung-Ju Ahn[1], Ji-Youn Han[2], Lecia V. Sequist[3], Byoung Chul Cho[4], Jong Seok Lee[5], Sang-We Kim[6], Wu-Chou Su[7], Chun-Ming Tsai[8], James Chih-Hsin Yang[9], Helena Yu[10], Leora Horn[11], Kang-Yun Lee[12], Vincent Haddad[13], Melanie M. Frigault[14], Ghada F. Ahmed[13], Liu Yang[15], Dana Ghiorghiu[13], Geoffrey R. Oxnard[16]

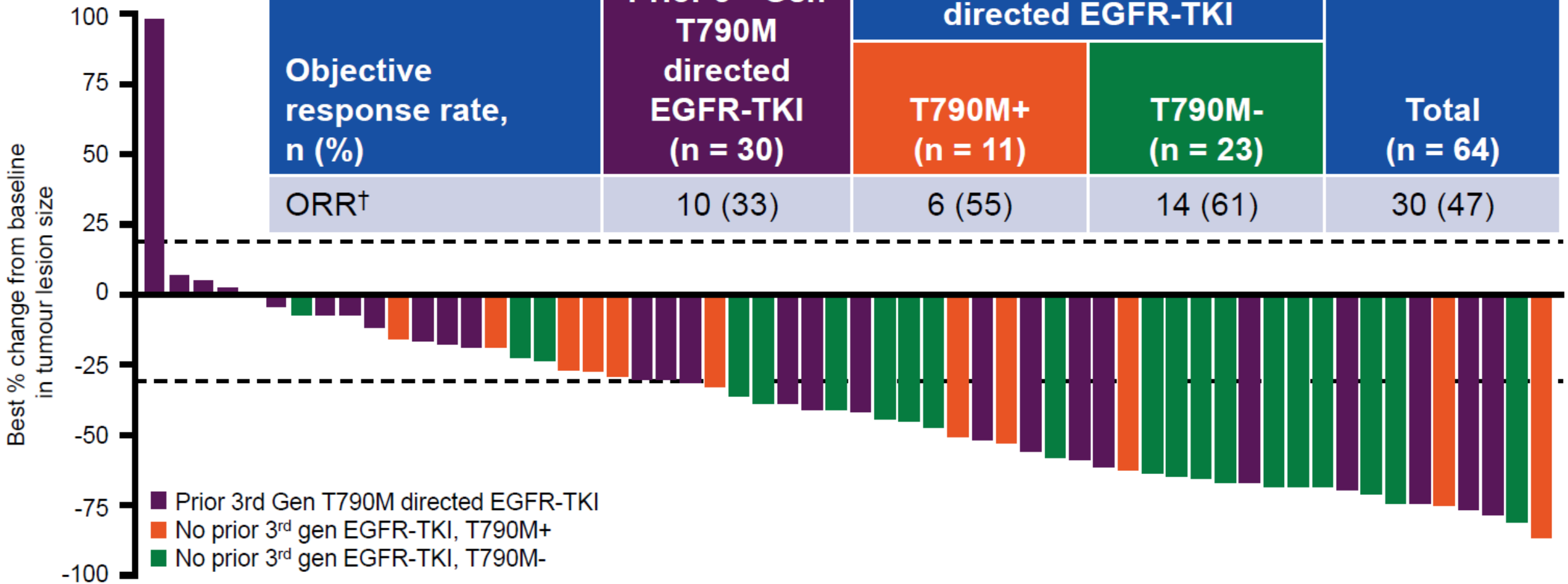
[1]Samsung Medical Center, Seoul, Republic of Korea; [2]National Cancer Center, Goyang, Republic of Korea; [3]Massachusetts General Hospital, Boston, MA, US; [4]Yonsei University Severance Hospital, Seoul, Republic of Korea; [5]Seoul National University Bundang Hospital, Seoul, Republic of Korea; [6]Asan Medical Center, Seoul, Republic of Korea; [7]National Cheng Kung University Hospital, Tainan City, Taiwan; [8]Taipei Veterans General Hospital, Taipei City, Taiwan; [9]National Taiwan University Hospital, Taipei City, Taiwan; [10]Memorial Sloan Kettering Cancer Center, New York, NY, US; [11]Vanderbilt University Medical Center, Nashville, TN, US; [12]Shuang-Ho Hospital, Taipei City, Taiwan; [13]AstraZeneca, Cambridge, UK; [14]AstraZeneca, Waltham, MA, US; [15]AstraZeneca, Shanghai, China; [16]Dana Farber Cancer Institute, Boston, MA, US

Presented by Professor Myung-Ju Ahn

IASLC 2017

Preliminary anti-tumour activity in all MET-positive patients*, n = 64

Objective response rate, n (%)	Prior 3rd Gen T790M directed EGFR-TKI (n = 30)	No prior 3rd Gen T790M directed EGFR-TKI		Total (n = 64)
		T790M+ (n = 11)	T790M- (n = 23)	
ORR†	10 (33)	6 (55)	14 (61)	30 (47)



Waterfall plot based on evaluable patients (n = 64): all patients dosed and with on-treatment assessment or discontinuation prior to first tumour assessment
Data cut-off 31 Aug 2017

*17 patients did not have central FISH confirmation of MET-positive status (n = 6 MET-negative; n = 11 unknown by central lab); †Confirmed by a later scan performed at least 4 weeks after initial response observed

TATTON Part B
NCT02143466

Conclusions

- Despite recent advances, NSCLC remains an area of high unmet medical need
  - PD-1 inhibitors only effective for a sub-set of patients
  - PD-1 inhibitors not active in patients with EGFR mutations
- Novel treatment options and combinations are needed for patients with relapsed EGFR-mutated NSCLC
- Recent data underscore the strong biological rationale for combination treatments of 3rd gen. EGFR TKI and HGF-inhibitors
- Blocking Anti-VEGF and Anti-HGF axis together with an EGFR-TKI may provide additional patient benefit and warrants clinical research

Dr. Richard Baird

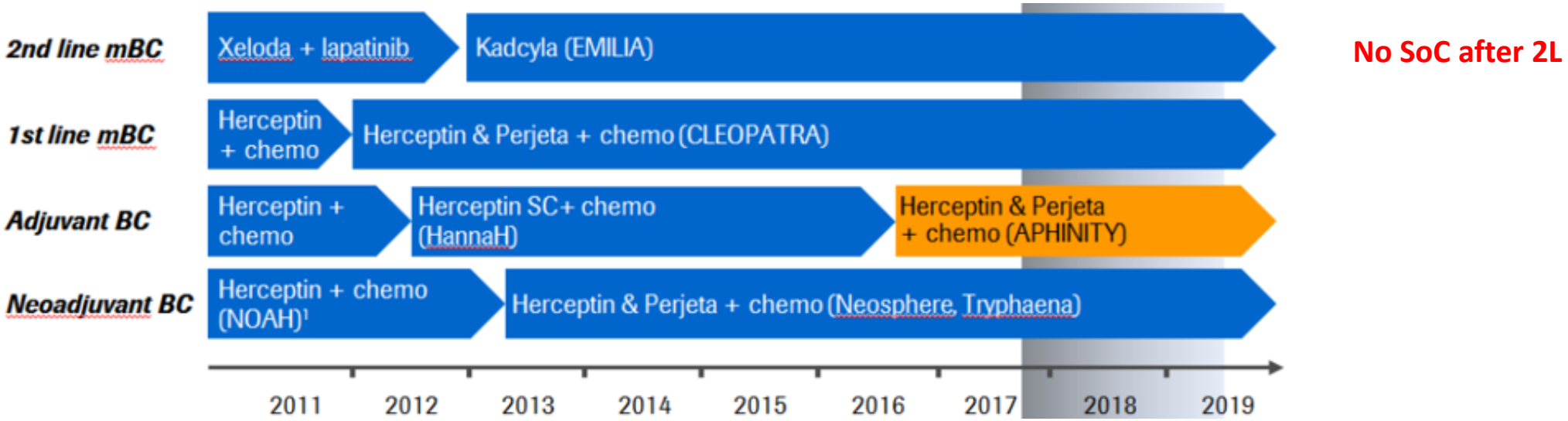
MA MBBS PhD FRCP

Academic Consultant in Experimental Cancer Therapeutics, University of Cambridge

Honorary Consultant in Medical Oncology, Cambridge University Hospitals, U.K.

HER2-positive Metastatic Breast Cancer (MBC) – currently no cure

- The outcome for patients with HER2-positive MBC has improved in recent years, since the introduction of effective anti-HER2 therapies, including trastuzumab, pertuzumab and T-DM1:



No SoC after 2L

- 15%–20% of patients with HER2-positive local/regional breast cancer still develop incurable metastatic disease despite currently available neo/adjuvant therapies[2]
- HER2-positive metastatic breast cancer essentially remains an incurable disease, with treatment failing eventually in all patients despite the best available treatments:
  - Median-progression free survival in later lines is only approximately 6 months[3]

1. Swain 2015.; 2. Martin 2016; 3. Baselga 2010

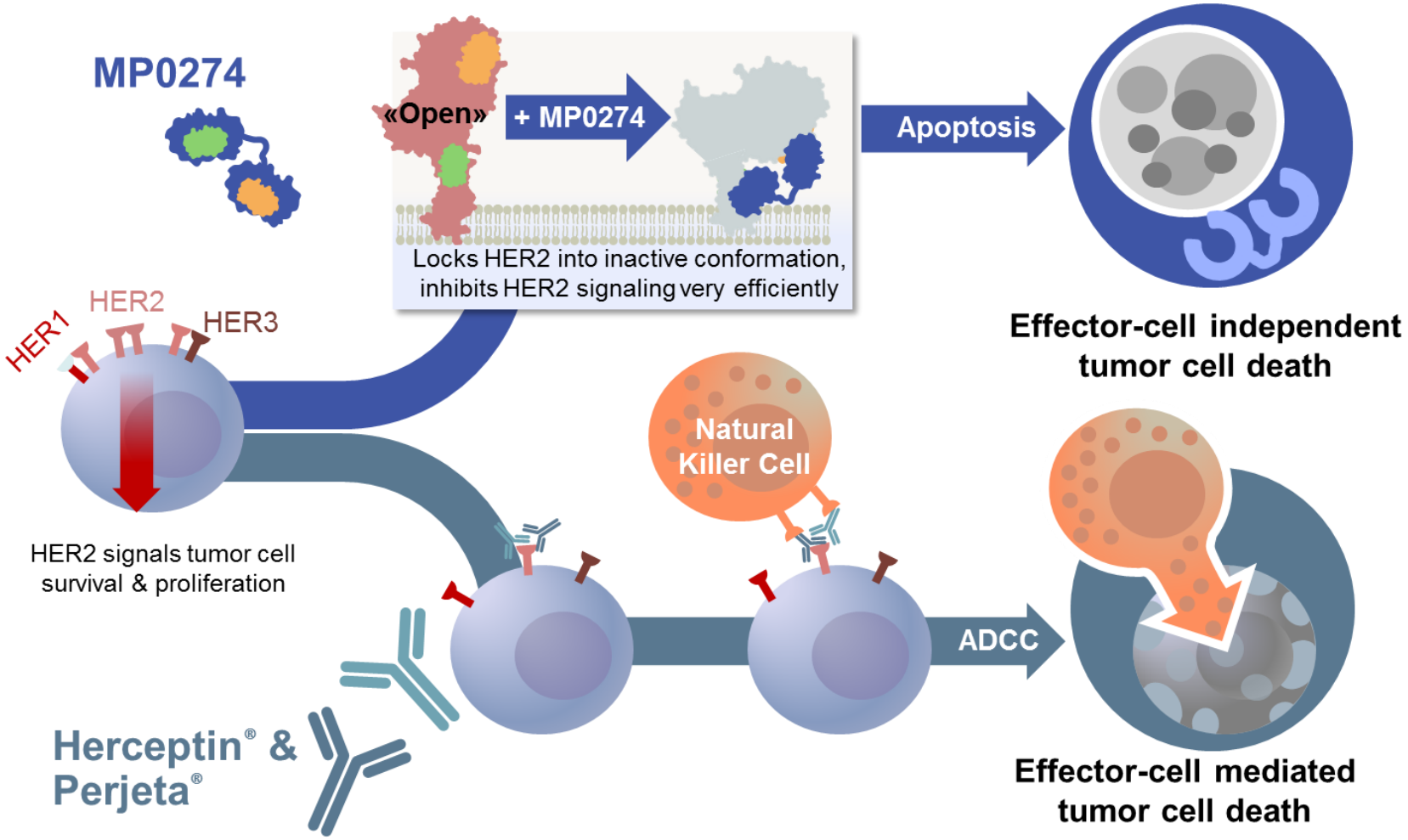
HER2 is a oncogenic driver across many tumor types – currently treatments registered for HER2+ Breast and Gastric Cancers only

- **Gastric Cancer (22% HER2-positive)**
  - Trastuzumab currently registered in combination with cisplatin + capecitabine/5-FU for first line treatment of metastatic disease, based on TOGA trial[1]
    - Median overall survival 11.1 months for chemo alone, 13.8 months for chemo + trastuzumab (p=0·0046)
- **Non-Small Cell Lung Cancer (2% HER2-positive)**
  - PFS 6.1 months with H, OS 12.2. with H[2]
- **Bladder cancer (52% HER2-positive)**
  - HER2 overexpression associated with features of biological and clinical aggressiveness[3,4]
- **Biliary tract malignancies (26.5% HER2-positive)** [5]
- **Ovarian Cancer (37.5% HER2-positive)** [6]

1 Bang 2010; 2. Gatzemeier 2004; 3. Hussain 2007; 4. Soria 2016; 5. Galdy 2017; 6. Jafri 2017

Potentially active against all HER2-dependent tumors independent of Antibody-Dependent Cellular Cytotoxicity (ADCC)

- MP0274 has an unique Mode-of-Action distinct from HER2 targeting antibodies:
  - it induces apoptosis
- MP0274 has the potential to be active against all HER2-dependent tumors including those resistant to trastuzumab and pertuzumab due to incomplete signalling inhibition or inadequate ADCC function

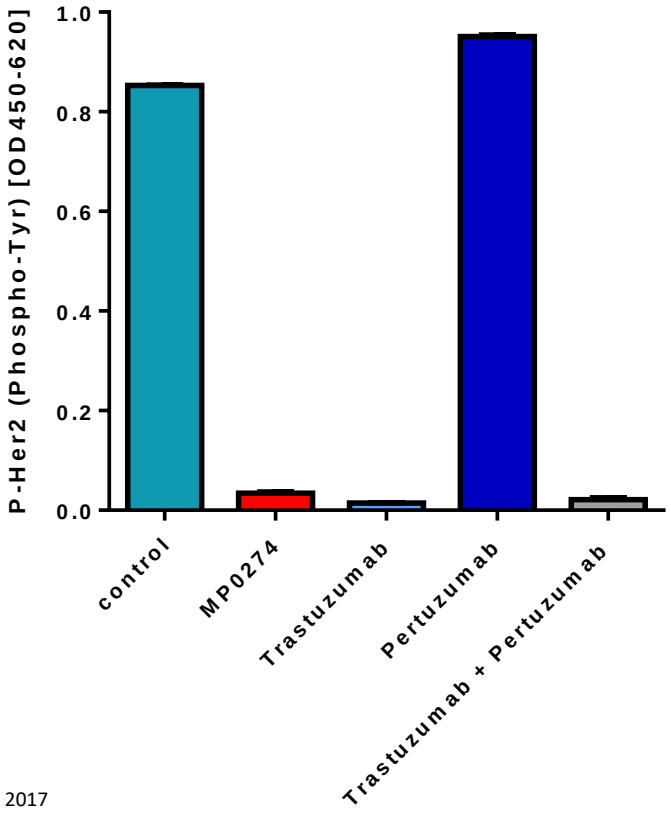


1 Fiedler AACR 2017

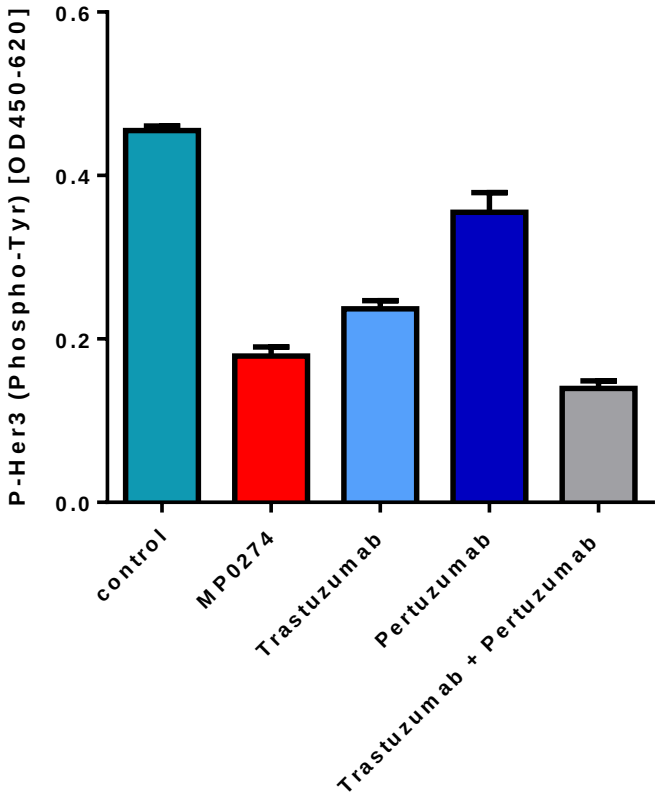
MP0274 inhibits HER2 signaling

- MP0274 inhibits HER2 and HER3 phosphorylation and downstream signaling via AKT

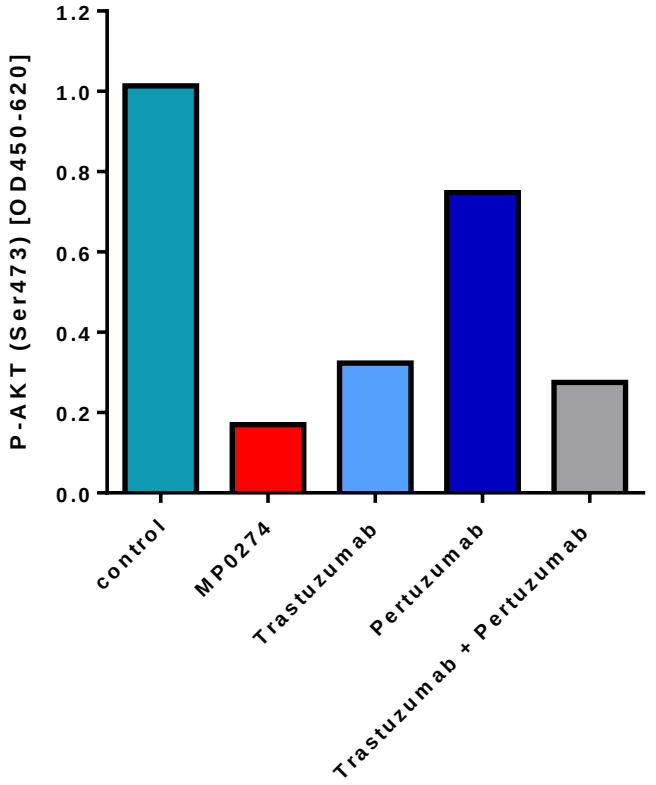
HER2-phosphorylation



HER3-phosphorylation



AKT-phosphorylation

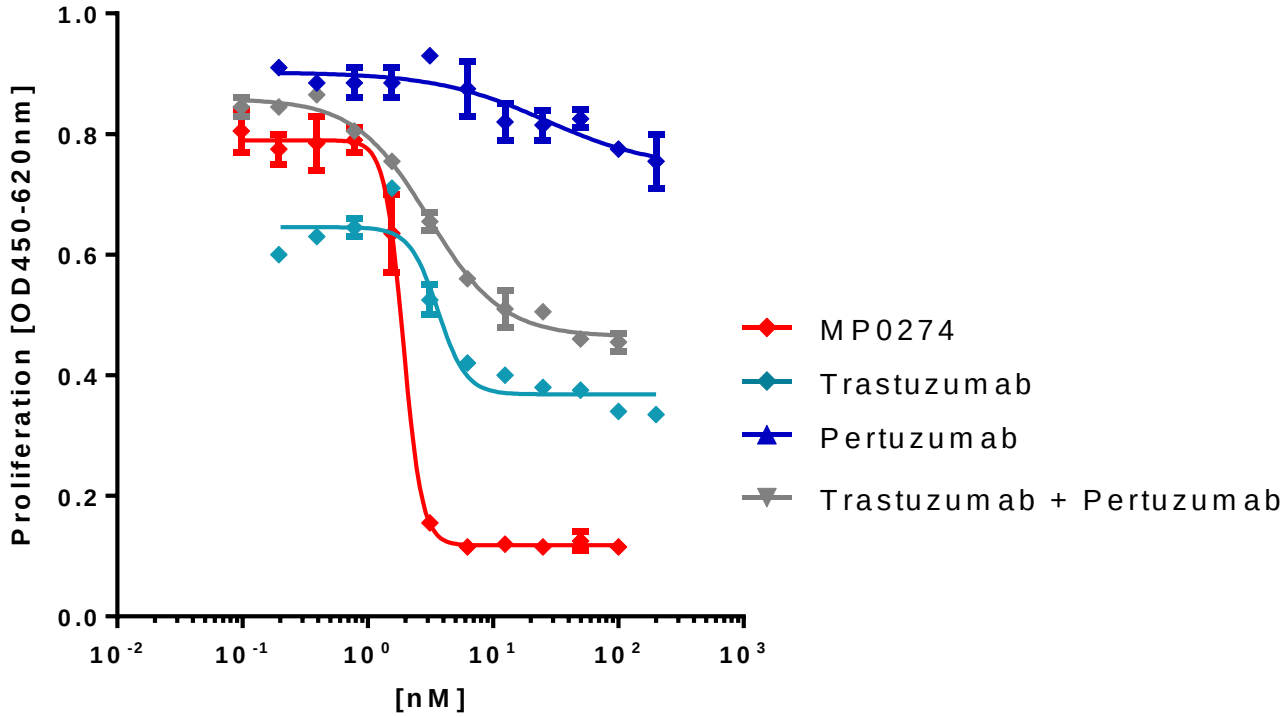


1 Fiedler AACR 2017

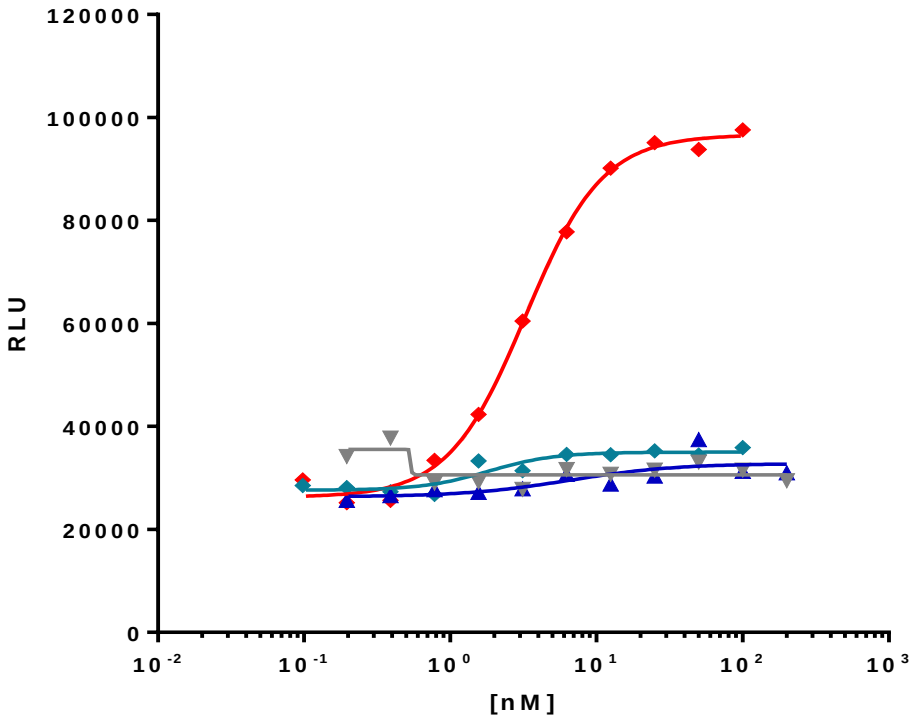
MP0274 induces apoptosis in HER2 addicted cells different mode of action than trastuzumab and pertuzumab

- MP0274 efficiently inhibits HER2-driven cell proliferation
- MP0274 drives HER2 addicted cancer cells into apoptosis

Inhibition of Proliferation



Induction of apoptosis



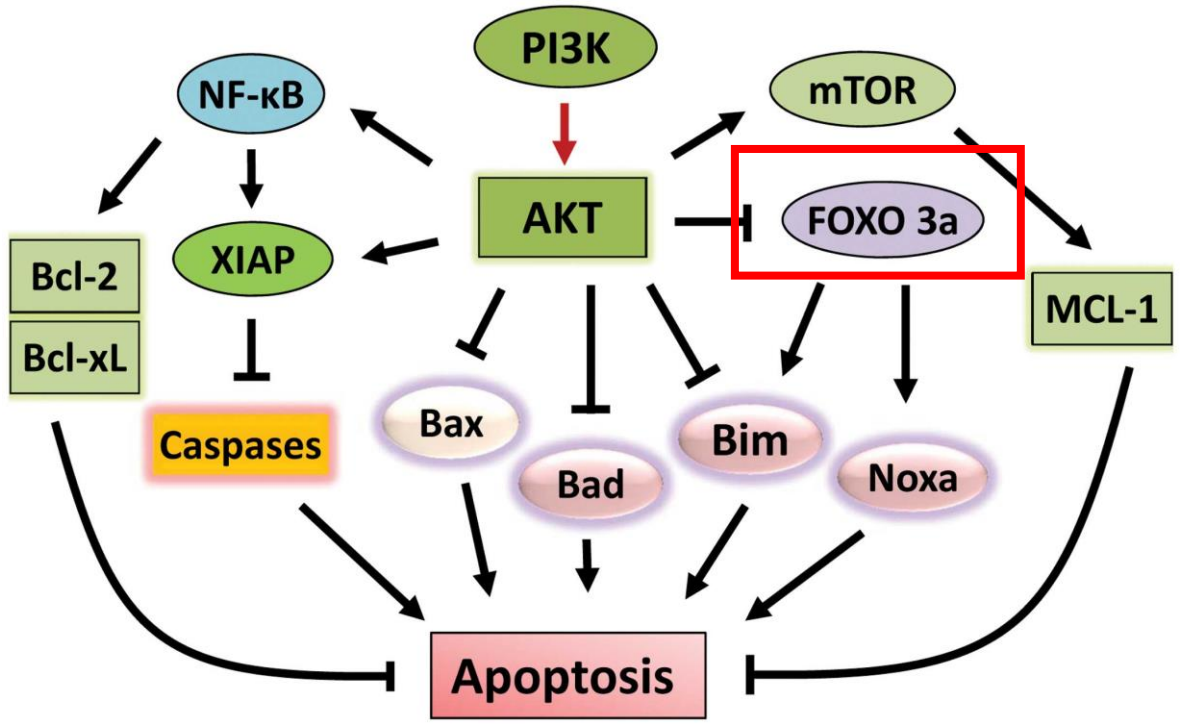
1 Kiemle-Kallee SABCS 2016

MP0274 leads to increased Foxo3

FOXO3 is a key regulator of cell survival and cell death

- MP0274 changes the phosphorylation of FOXO3a resulting in increased FOXO3a protein expression in both cell lines
- MP0274 upregulates FOXO3:
  - AKT phosphorylates FOXO3a which results in its degradation -> promotion of cell survival
  - Inhibition of FOXO3a phosphorylation leads to its stabilization, translocation into the nucleus

MP0274 has a different mode of action which leads to induction of apoptosis

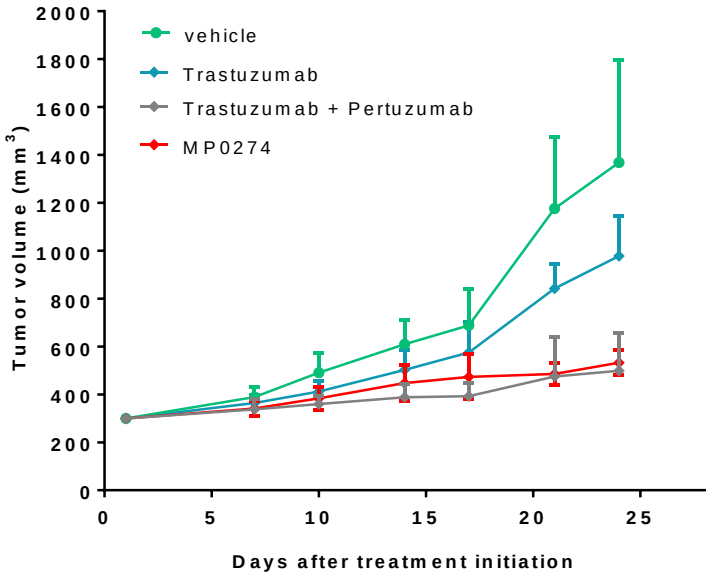


Hein et al, 2014 IJO

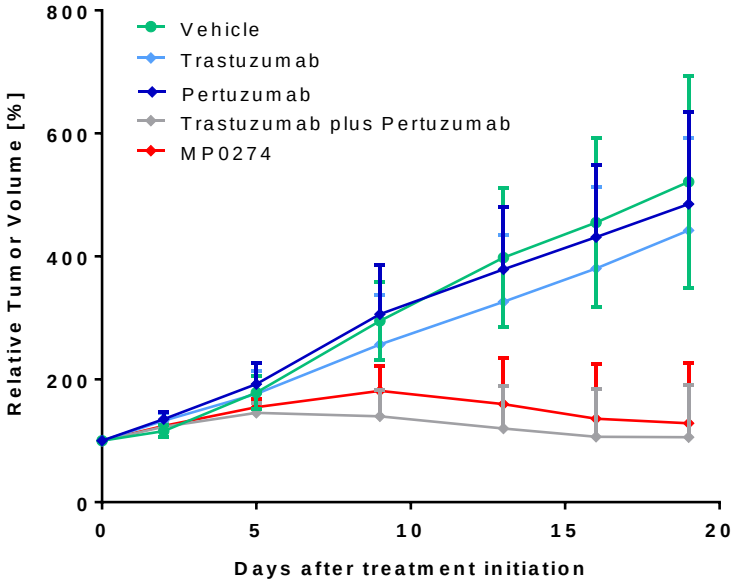
MP0274 inhibits tumor growth in a variety of HER2-positive patient-derived cancer models

- MP0274 induces tumor regression and durable anti-tumor responses in Patient-Derived Xenograft (PDX) models ranging from IHC3+ to 1+ and FISH amplified and non-amplified
- MP0274 strongly inhibits tumor growth in HER2-positive patient-derived cancer models without ADCC involvement

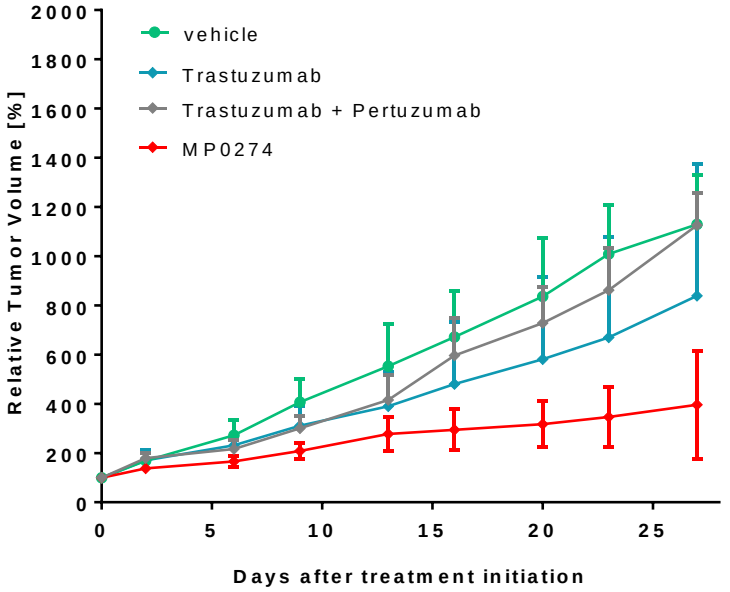
Breast cancer PDX model
Maca4898 (Her2 IHC2+)



Gastric cancer PDX model
GXA3039 (Her2 IHC3/2+)



Colorectal cancer PDX model
CXF1991 (Her2 IHC3+)



Conclusions

- HER2 overexpression is found across a range of tumour types and is often associated with poorer prognosis
- Anti-HER2 therapies are currently approved only for HER2-positive breast and gastric cancers, however in the metastatic setting nearly all patients ultimately grow through these
- Therefore, there is still a high unmet medical need to develop new treatment approaches to overcome limitations of available anti-HER2 therapies and expand to HER2 positive cancers beyond breast and gastric cancer.

- MP0274 induces apoptosis in HER2 addicted cells in a way which is different to trastuzumab and pertuzumab (upregulation of FOXO3, inhibition of survival signaling)
- MP0274 inhibits tumor growth in a variety of HER2-positive patient-derived cancer models
- MP0274, through its unique mode of action, may offer benefits to HER2-positive cancer patients compared to currently approved therapies

Turning the DARPin® Differentiation into Patient Outcome – Our Target Profiles

Pre-Clinical	Phase 1	Phase 2	Phase 3
MP0310	**MP0274**	**MP0250**	**Abicipar**
		NSCLC	Wet AMD
		MM	
Tumor-restricted activity (switch) to avoid dose-limiting side effects	Molecular handcuff inducing cell death in HER2+ cancer cells	Blocking 2 key escape pathways in parallel	Long-acting VEGF inhibitor in the eye
Opening a new therapeutic window for combinations	Activity in patients no longer benefiting from approved antibodies	Restore activity of drugs to which cancer has become resistant in MM and NSCLC	Non-inferiority to competition with less frequent ocular injections

DARPin® Technology & DARPin® Candidates in I/O

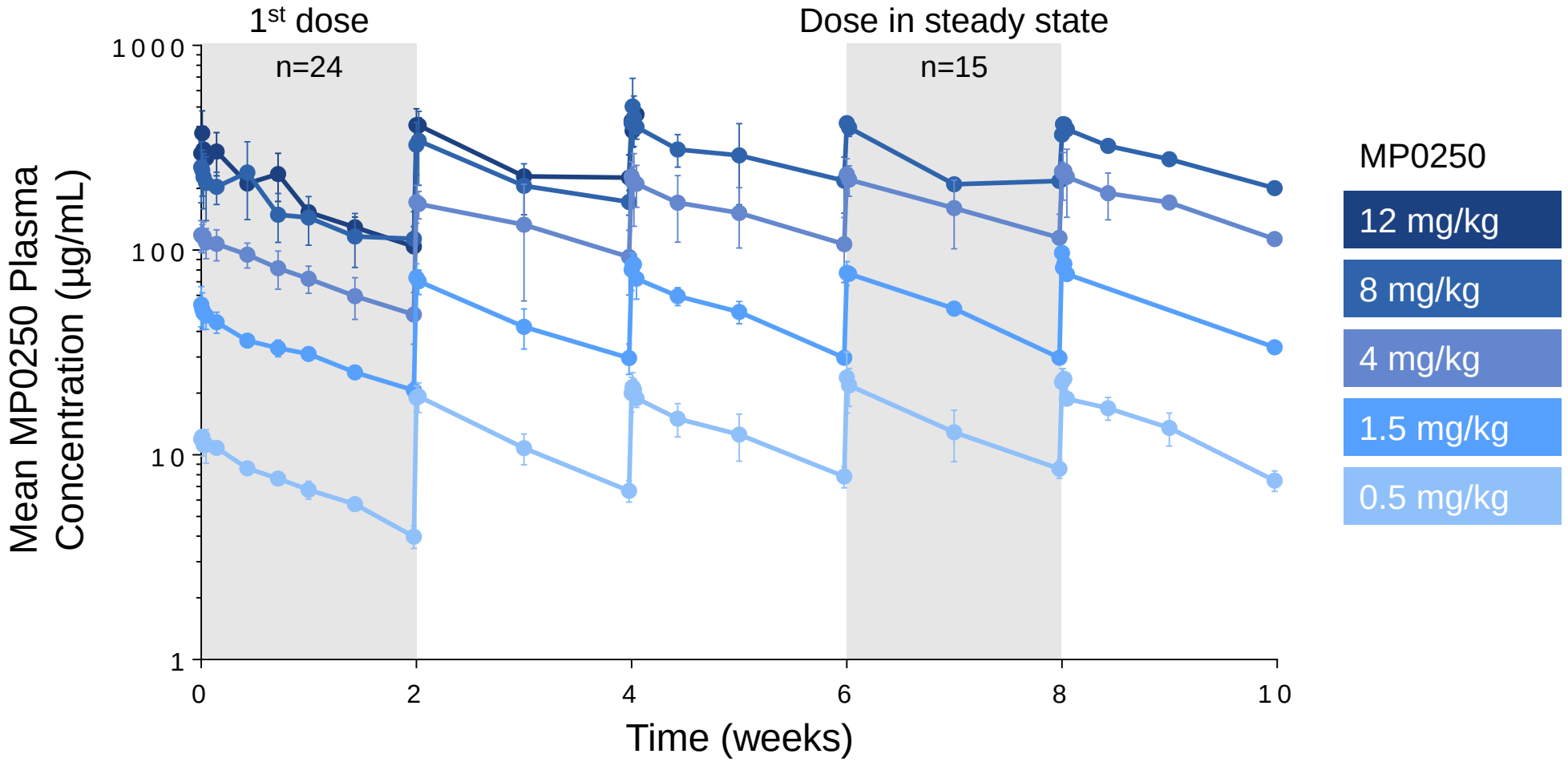
Michael Stumpp, CSO

MOLECULAR partners

How We Started in 2004: A Big Dream Begins…

DARPin® MP0250 has PK Comparable to mAbs: ~2 Weeks

MP0250

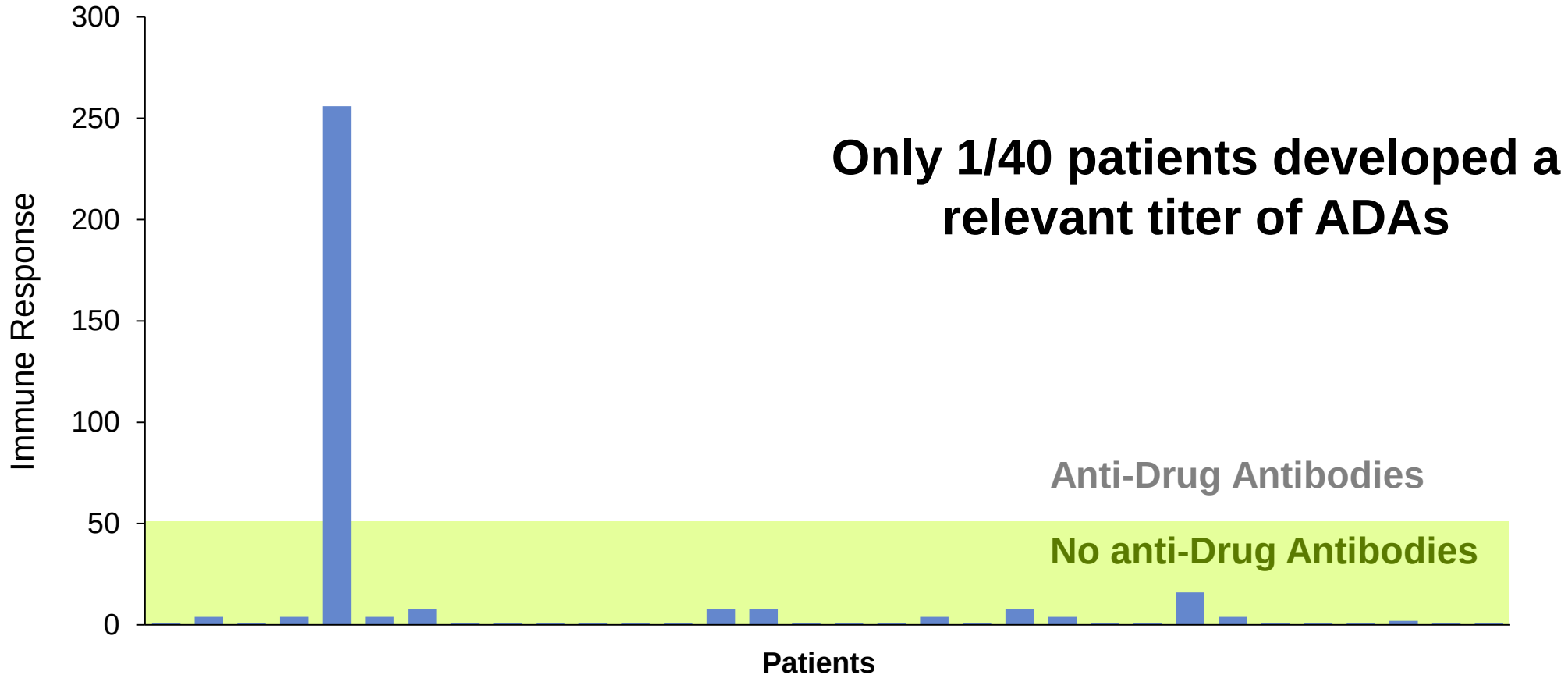


MP0250 plasma concentration over time traces following 5 repeated biweekly doses. Shown are mean ± SD concentrations of patients receiving the same dose level. Number of patients vary per cohort and dosing interval due to different number of patients per cohort and drop out of patients during the course of the trial.

MOLECULAR partners

MP0250 Shows Very Low Immune Response in Humans

MP0250



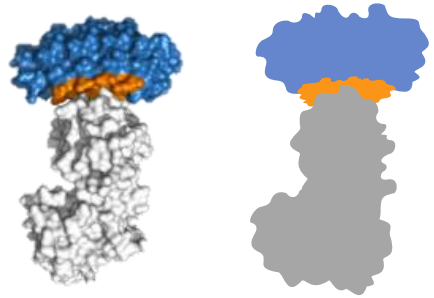
Immune response: Maximum fold titer increase relative to baseline

DARPin® Proteins: A Different Class of Therapeutics

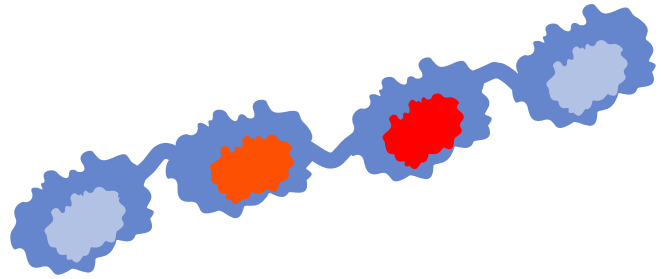
- Flexible architecture: can target single OR multiple pathways
  - Mono-DARPin® candidate:
    - Selected to bind to a target with high affinity and specificity (large libraries)
  - Multi-DARPin® candidate:
    - Linked mono-DARPin® domains (so far ≤6) and directly used for functional screening
- Ideal properties: small size, high potency, high stability, high affinity (strong binding)
- Proof of platform: low immunogenicity* and long $t_{1/2}$ in bloodstream and eye†
- Fast and cost effective drug discovery engine: unlock novel mode of actions

Derived from ankyrin repeat proteins which are naturally-occurring binding proteins in multifunctional contexts

Abicipar: Mono-DARPin®



MP0250: Multi-DARPin® (4x)



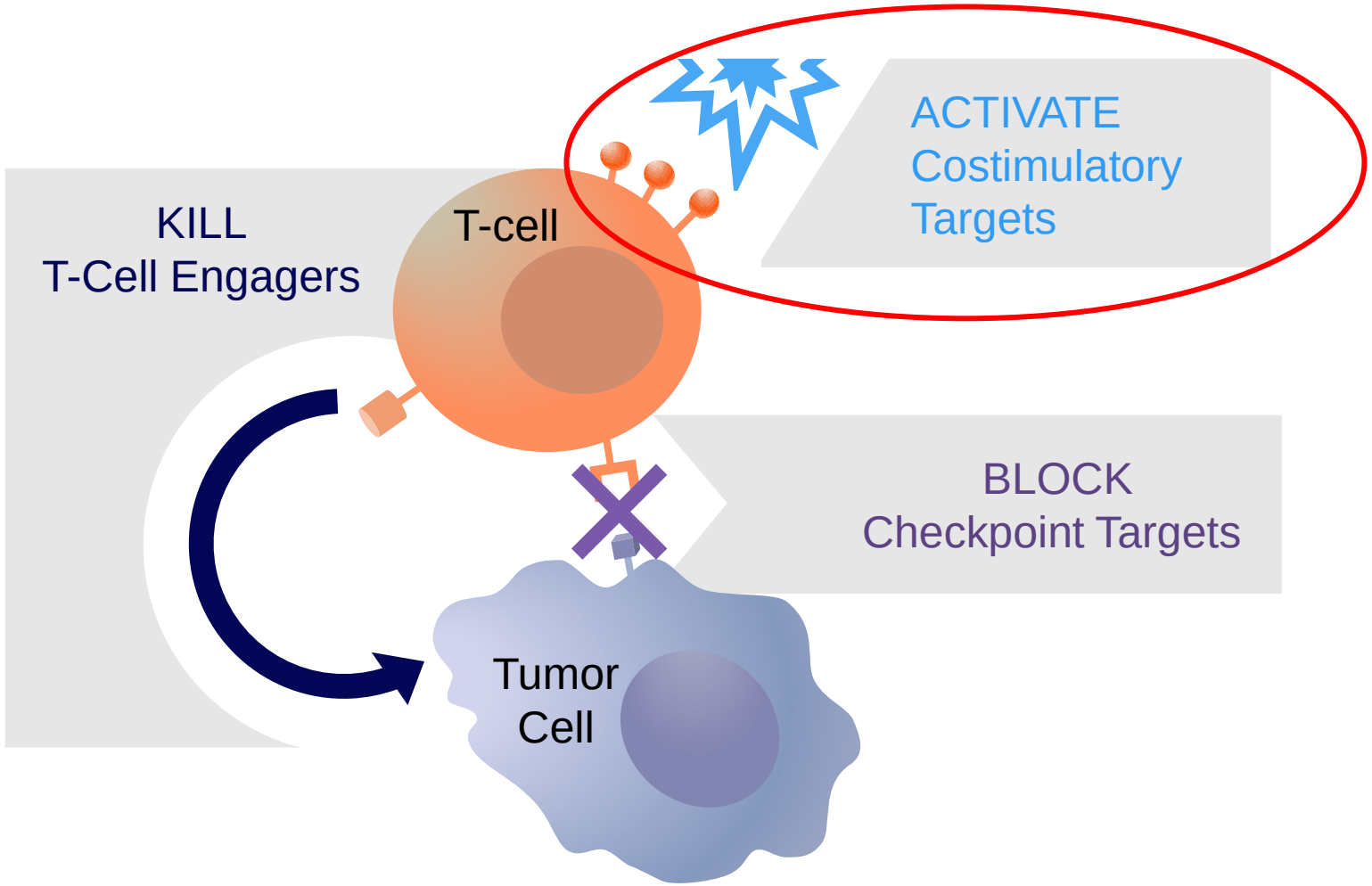
† Systemic $t_{1/2}$ ~12 d (MP0250 Phase 1), ~14 d in the eye (abicipar);
*MP0250 Phase 1 study results show sustained exposure indicating absence of clearing antibodies.

DARPin® Technology: Conclusions

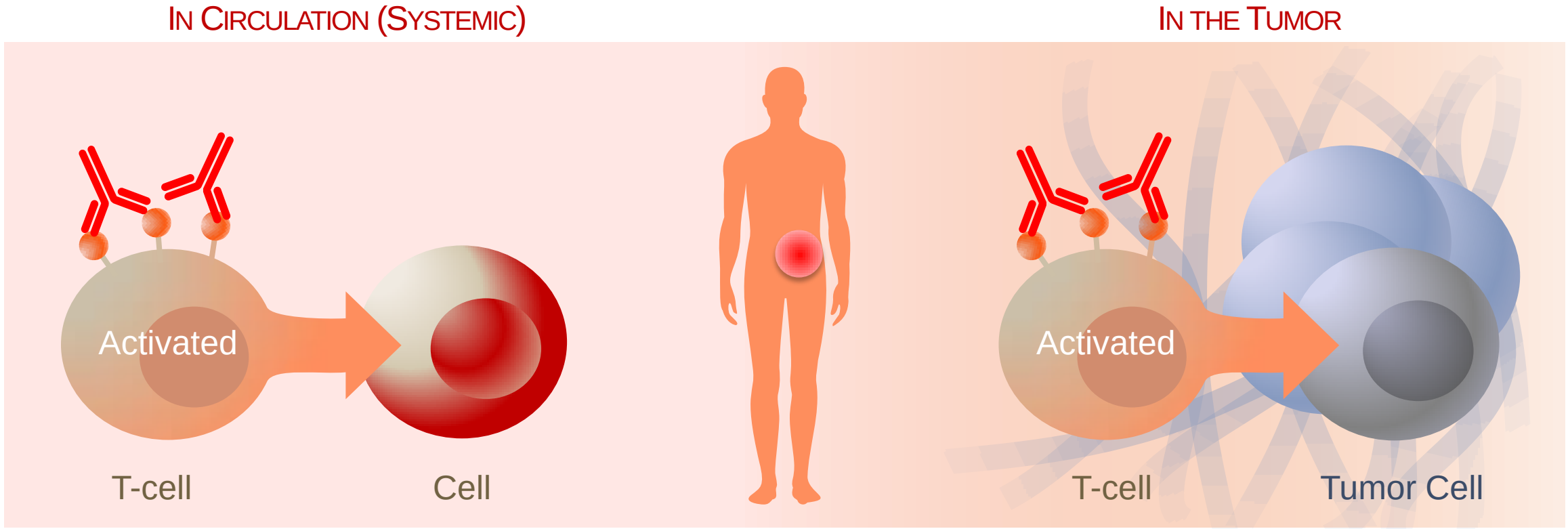
- DARPin® Difference: truly delivering on the promise of multi-specificity
  - Novel architecture beyond antibody-based approaches
  - Novel modes of action filling our DARPin® toolbox
  - Highest quality and developability characteristics
  - De-risked on immunogenicity and systemic PK
  - Full “freedom to operate” from an IP point of view
- Now: Full attention to applying the DARPin® Difference to deliver patient benefit
- Strong internal capabilities to move compounds forward

Ready to deliver more

Our Approach to I/O: Activate Immune Cells with a Switch

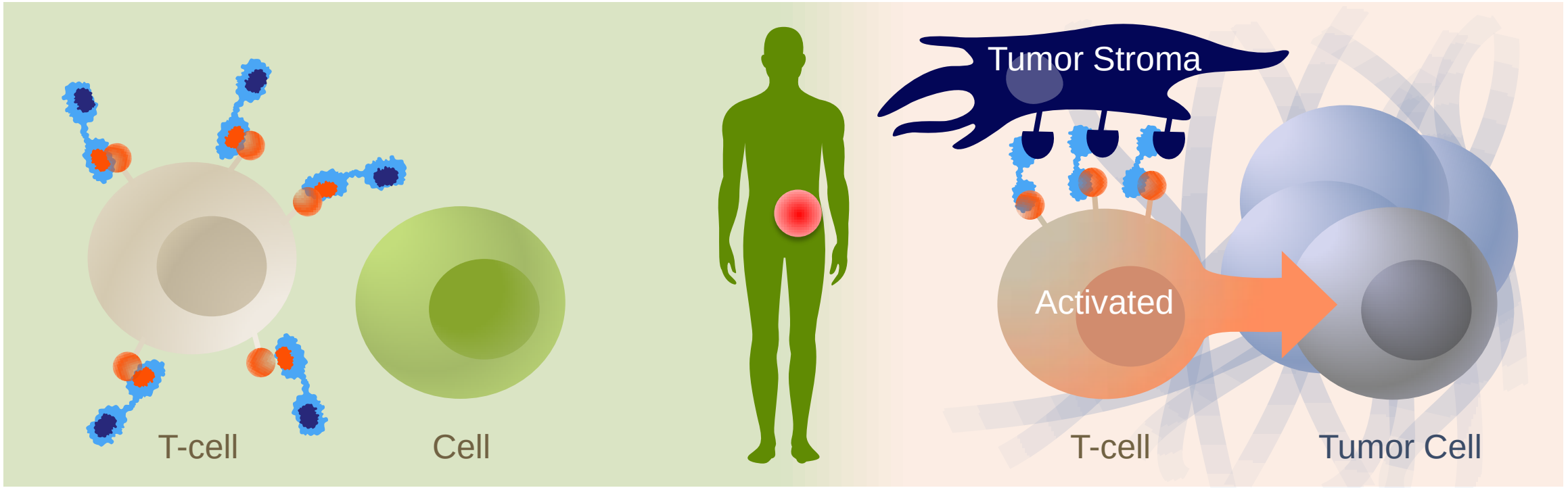


Toxicity Limits Full Potential of Antibody Agonists

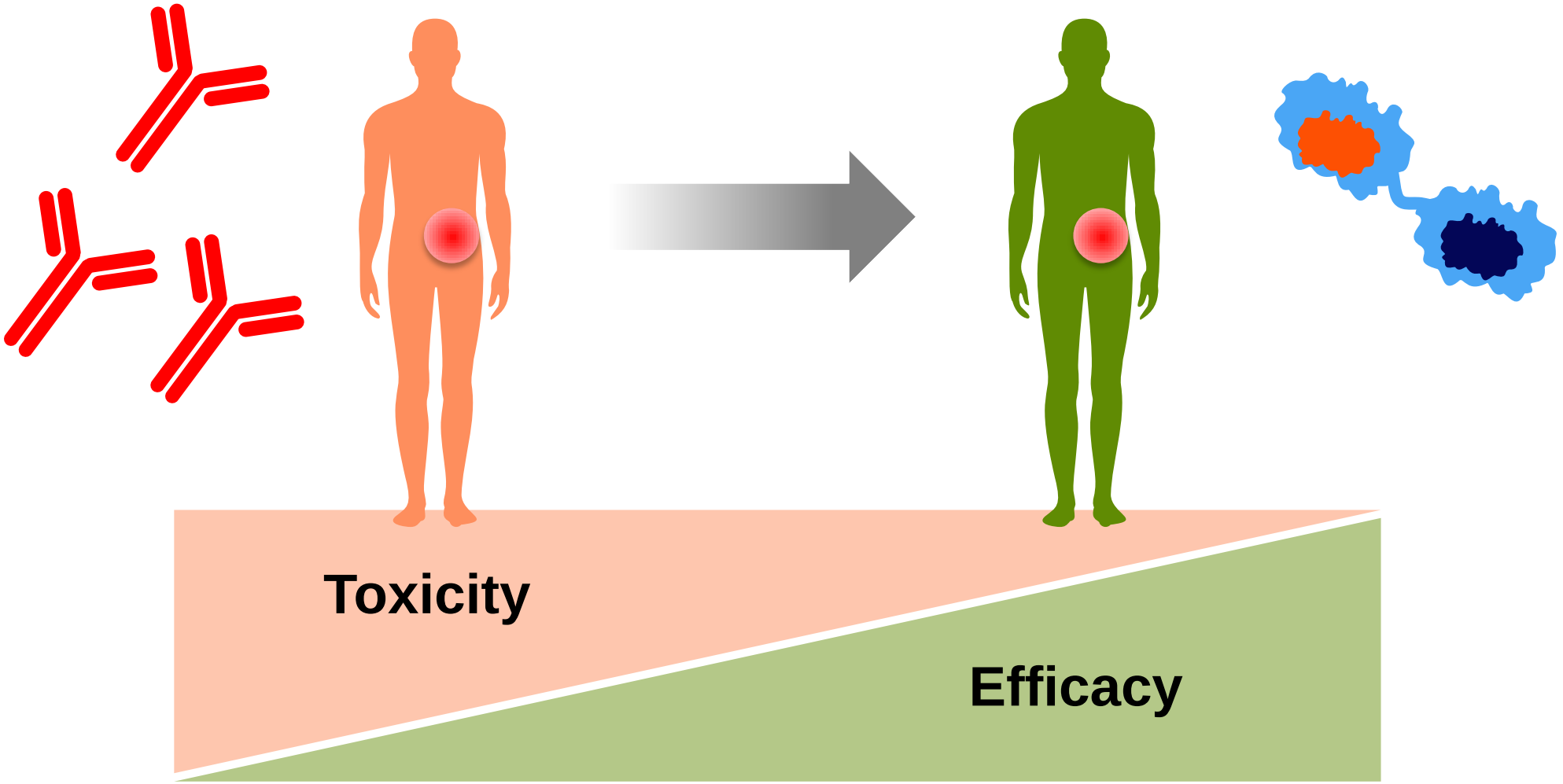


Clustering = activation of T-cells everywhere in the body

Our Approach Allows to “Switch on” DARPin® Agonist only in the Tumor



Our Vision: Expand the Therapeutic Window, Enabling Combinations to Become Successful



MP0310: Activating T-cells (only) in the Tumor

MP0310

- Tumor-restricted T-cell co-stimulator

- Local activation of immune cells
- No systemic side effects expected
- Ideal combination partner for other T-cell based therapies

- Many FAP positive cancers that could benefit from T-cell activation

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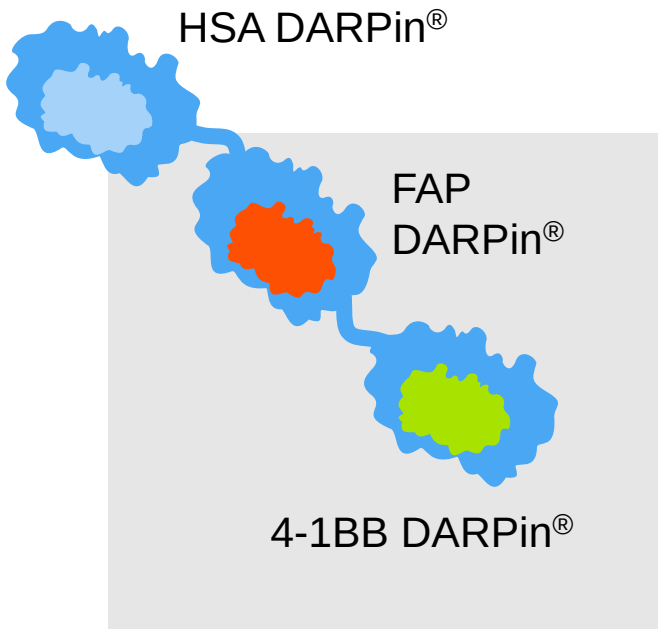
- Fully owned by Molecular Partners – IP protection at least until 2038

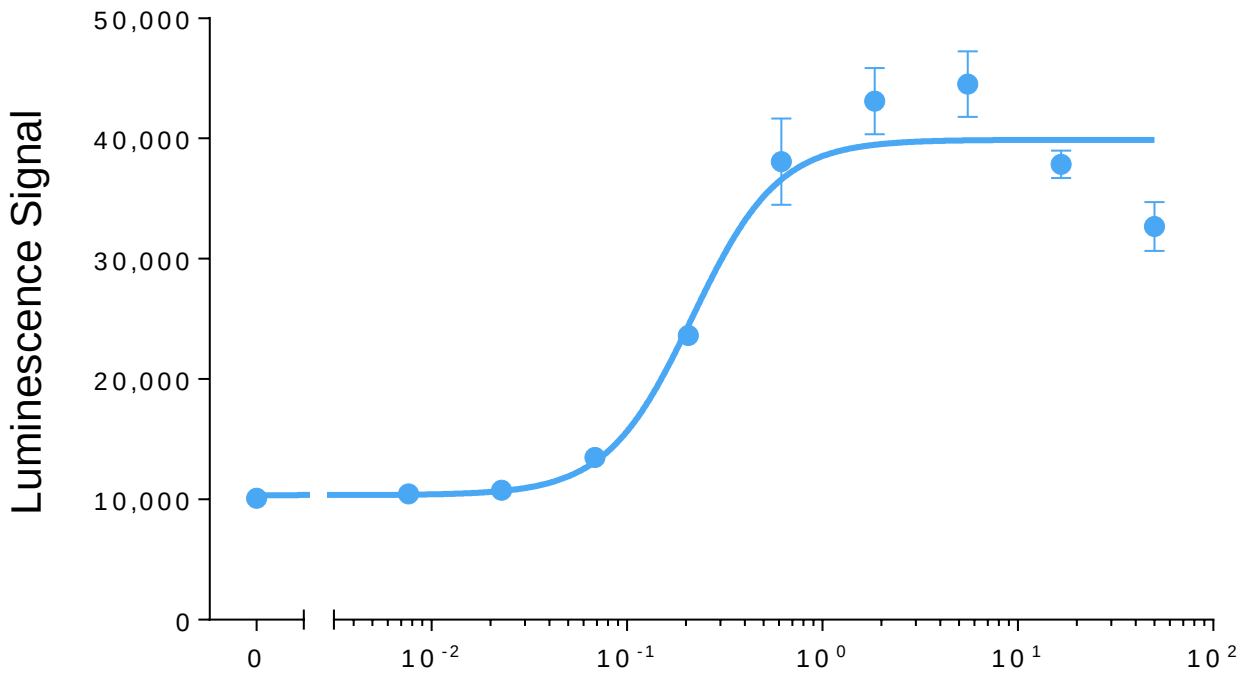


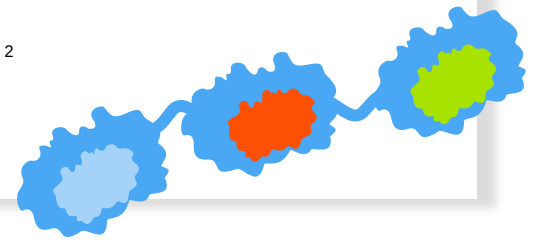
*FAP = Fibroblast activation protein

Overview of MP0310 Data

MP0310

Tumor-site switch	Tumor localized	No systemic toxicity	Highly efficacious	Ideal for combinations



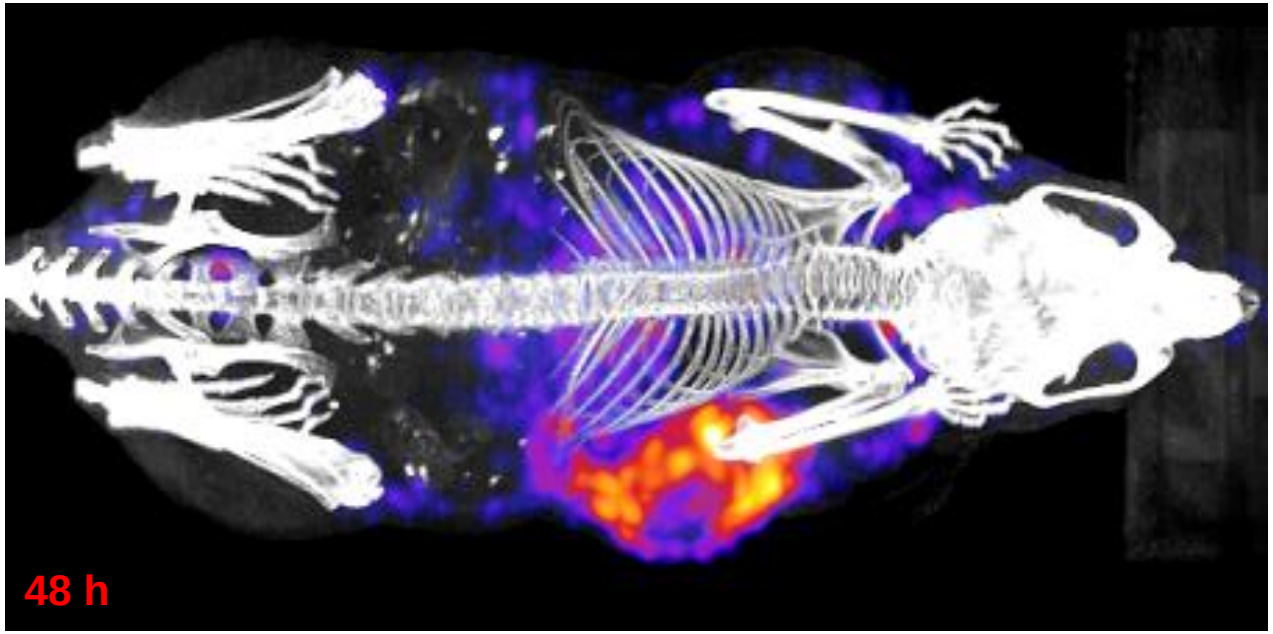


MOLECULAR partners

Overview of MP0310 Data

MP0310

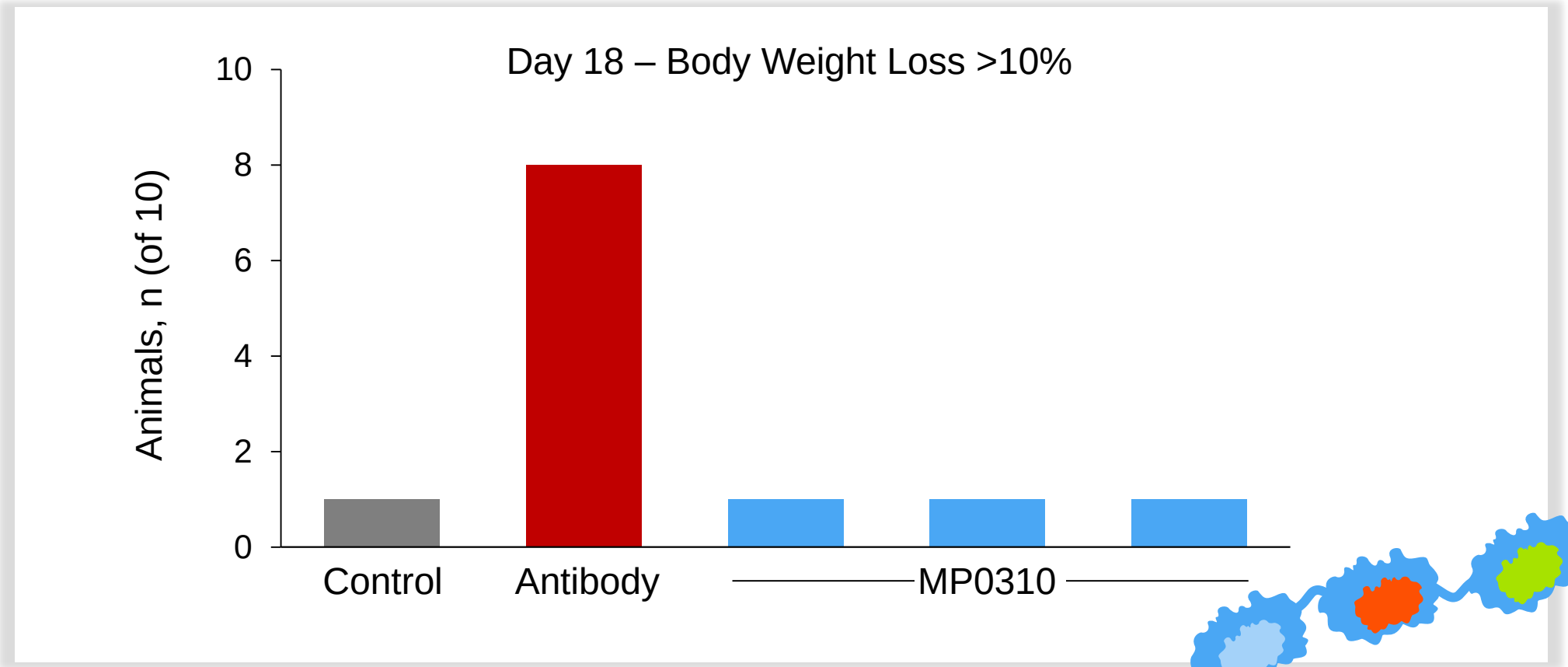
Tumor-site switch	**Tumor localized**	No systemic toxicity	Highly efficacious	Ideal for combinations



Overview of MP0310 Data

MP0310

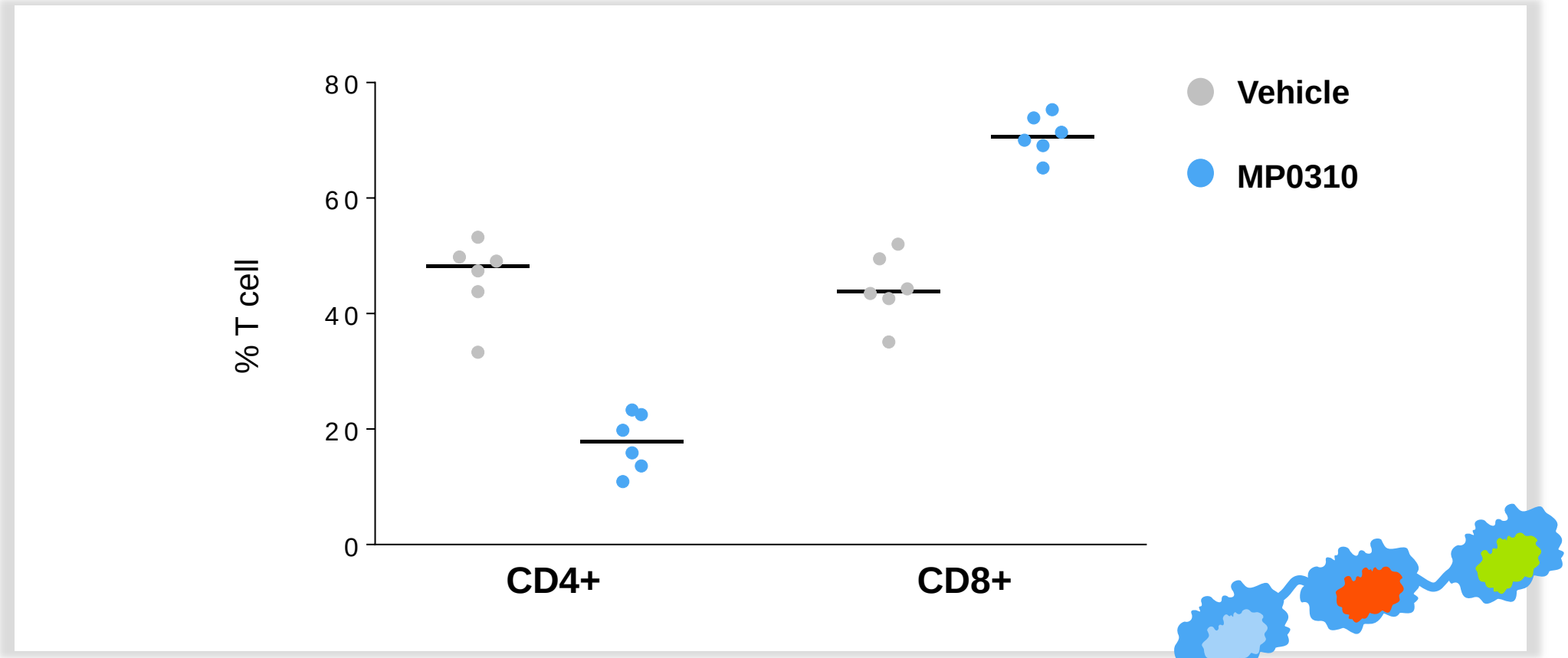
Tumor-site switch	Tumor localized	**No systemic toxicity**	Highly efficacious	Ideal for combinations



Overview of MP0310 Data

MP0310

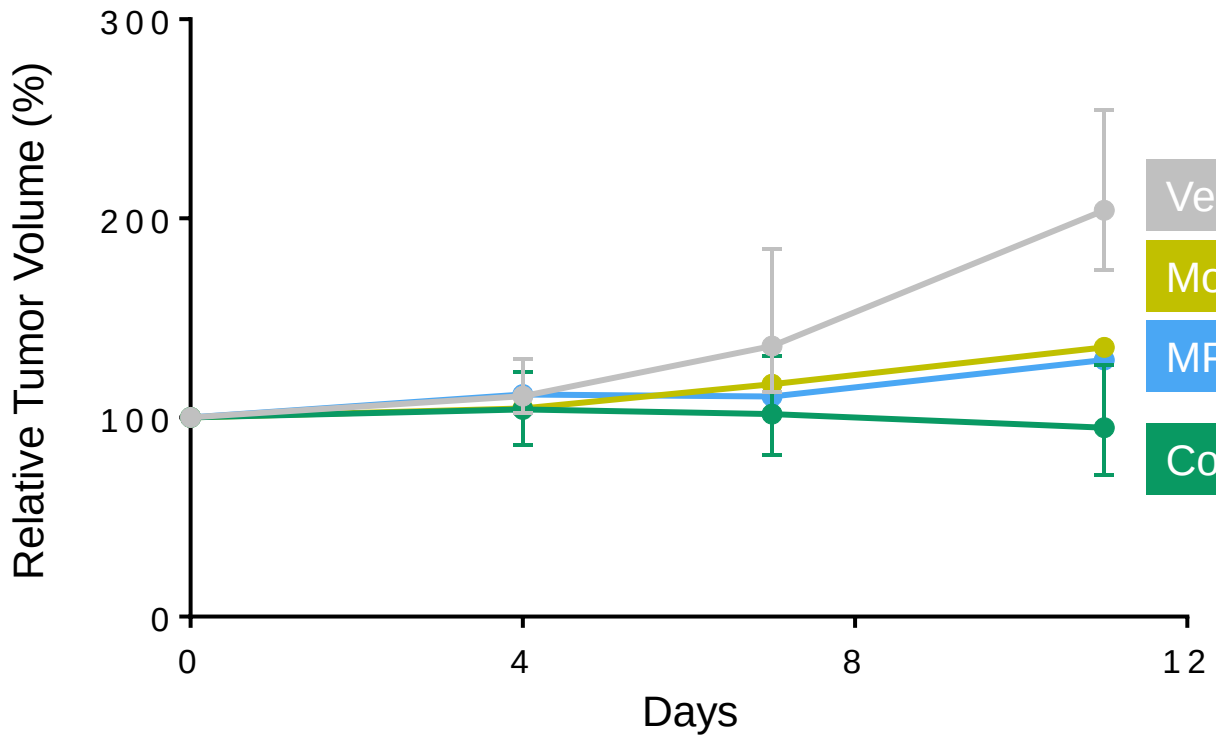
Tumor-site switch	Tumor localized	No systemic toxicity	**Highly efficacious**	Ideal for combinations

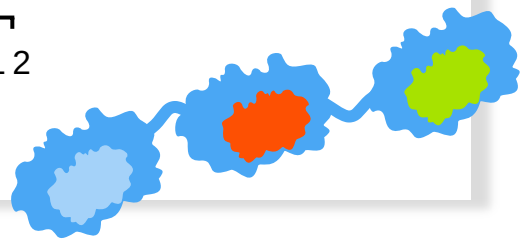


Overview of MP0310 Data

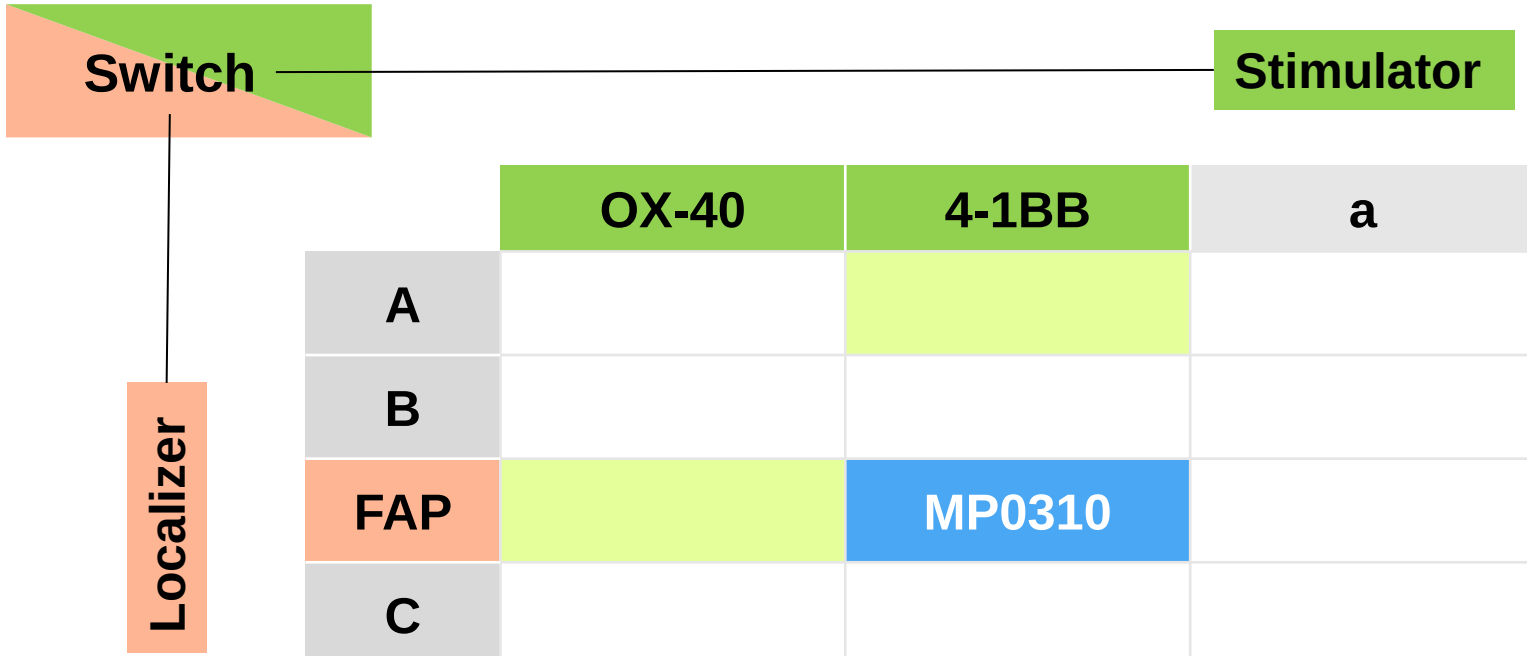
MP0310

Tumor-site switch	Tumor localized	No systemic toxicity	Highly efficacious	**Ideal for combinations**





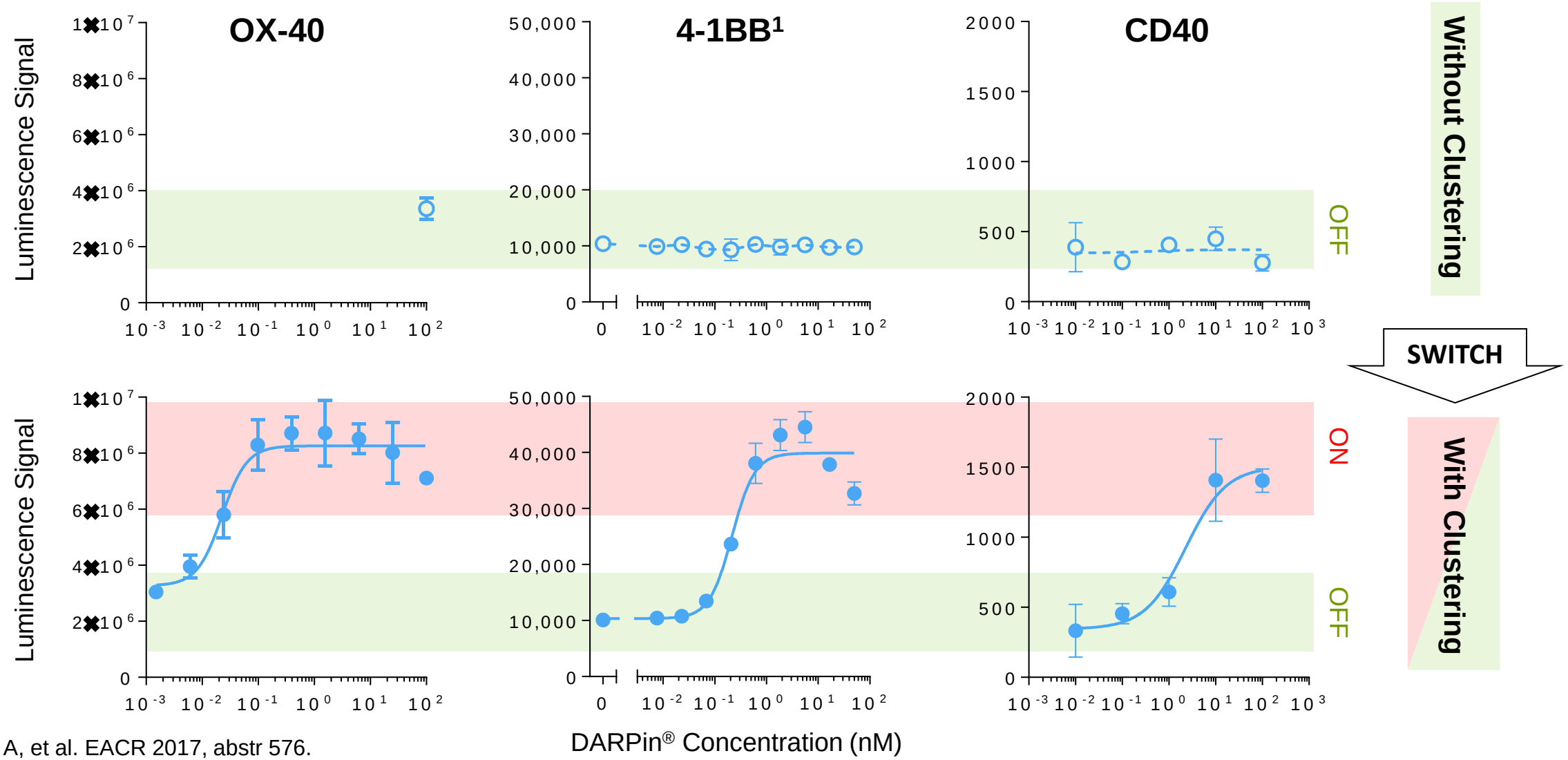
DARPin® Toolbox with Unlimited Combinations



	OX-40	4-1BB	a	CD40	c
A					
B					
FAP		MP0310			
C					
D					

Many DARPin® candidates under investigation for both solid and liquid tumors (including combinations)

All DARPin® Stimulators Successful to Date



1. Link A, et al. EACR 2017, abstr 576.

Conclusions from Our I/O Pipeline

Pre-Clinical	Phase 1	Phase 2	Phase 3
MP0310	**MP0274**	**MP0250**	**Abicipar**
		NSCLC MM	Wet AMD
Tumor-restricted activity (switch) to avoid dose-limiting side effects	Molecular handcuff inducing cell death in HER2+ cancer cells	Blocking 2 key escape pathways in parallel	Long-acting VEGF inhibitor in the eye
Opening a new therapeutic window for combinations	Activity in patients no longer benefiting from approved antibodies	Restore activity of drugs to which cancer has become resistant in MM and NSCLC	Non-inferiority to competition with less frequent ocular injections

Ophthalmology: Abicipar

Baldo Scassellati Sforzolini, SVP
Clinical Development, Allergan

MOLECULAR partners

ABICIPAR

BALDO SCASSELLATI SFORZOLINI, MD, PHD, MBA
SENIOR VICE PRESIDENT,
CLINICAL DEVELOPMENT,
ALLERGAN

Allergan

ALLERGAN CAUTIONARY STATEMENTS

Forward Looking Statements

This communication includes statements that refer to estimated or anticipated future events and are forward-looking statements. We have based our forward-looking statements on management's beliefs and assumptions based on information available to our management at the time these statements are made. Such forward-looking statements reflect our current perspective of our business, future performance, existing trends and information as of the date of this filing. These include, but are not limited to, our beliefs about future revenue and expense levels and growth rates, prospects related to our strategic initiatives and business strategies, including the integration of, and synergies associated with, strategic acquisitions, express or implied assumptions about government regulatory action or inaction, anticipated product approvals and launches, business initiatives and product development activities, assessments related to clinical trial results, product performance and competitive environment, and anticipated financial performance. Without limiting the generality of the foregoing, words such as "may," "will," "expect," "believe," "anticipate," "plan," "intend," "could," "would," "should," "estimate," "continue," or "pursue," or the negative or other variations thereof or comparable terminology, are intended to identify forward-looking statements. The statements are not guarantees of future performance and involve certain risks, uncertainties and assumptions that are difficult to predict. We caution the reader that these statements are based on certain assumptions, risks and uncertainties, many of which are beyond our control. In addition, certain important factors may affect our actual operating results and could cause such results to differ materially from those expressed or implied by forward-looking statements. These factors include, among others the inherent uncertainty associated with financial projections; the anticipated size of the markets and continued demand for Allergan's existing products; Allergan's ability to successfully develop and commercialize new products; Allergan's ability to conform to regulatory standards and receive requisite regulatory approvals; availability of raw materials and other key ingredients; uncertainty and costs of legal actions and government investigations; fluctuations in Allergan's operating results and financial condition, particularly given our manufacturing and sales of branded products; the impact of uncertainty around of timing of generic entry related to key products, including Restasis®, on our financial results; risks associated with acquisitions, mergers and joint ventures, such as difficulties integrating businesses, uncertainty associated with financial projections, projected cost reductions, projected synergies, restructurings, increased costs, and adverse tax consequences; expectations regarding contingent payments, including regarding litigation and related liabilities, purchase price adjustment or transaction consideration payments; the results of the ongoing business following the completion of the divestiture of Allergan's generics business to Teva; the adverse impact of substantial debt and other financial obligations on the ability to fulfill and/or refinance debt obligations; risks associated with relationships with employees, vendors or key customers as a result of acquisitions of businesses, technologies or products; our compliance with federal and state healthcare laws, including laws related to fraud, abuse, privacy security and others; generic product competition with our branded products; uncertainty associated with the development of commercially successful branded pharmaceutical products; costs and efforts to defend or enforce technology rights, patents or other intellectual property; expiration of patents on our branded products and the potential for increased competition from generic manufacturers; competition between branded and generic products; Allergan's ability to obtain and afford third-party licenses and proprietary technology we need; Allergan's potential infringement of others' proprietary rights; our dependency on third-party service providers and third-party manufacturers and suppliers that in some cases may be the only source of finished products or raw materials that we need; Allergan's competition with certain of our significant customers; the impact of our returns, allowance and chargeback policies on our future revenue; successful compliance with governmental regulations applicable to Allergan's and Allergan's respective third party providers' facilities, products and/or businesses; the difficulty of predicting the timing or outcome of product development efforts and regulatory agency approvals or actions, if any; Allergan's vulnerability to and ability to defend against product liability claims and obtain sufficient or any product liability insurance; Allergan's ability to retain qualified employees and key personnel; the effect of intangible assets and resulting impairment testing and impairment charges on our financial condition; Allergan's ability to obtain additional debt or raise additional equity on terms that are favorable to Allergan; difficulties or delays in manufacturing; our ability to manage environmental liabilities; global economic conditions; Allergan's ability to continue foreign operations in countries that have deteriorating political or diplomatic relationships with the United States; Allergan's ability to continue to maintain global operations and the exposure to the risks and challenges associated with conducting business internationally; risks associated with tax liabilities, or changes in U.S. federal or international tax laws to which we are subject, including the risk that the Internal Revenue Service disagrees that Allergan is a foreign corporation for U.S. federal tax purposes; risks of fluctuations in foreign currency exchange rates; risks associated with cyber-security and vulnerability of our information and employee, customer and business information that Allergan stores digitally; Allergan's ability to maintain internal control over financial reporting; changes in the laws and regulations, affecting among other things, availability, pricing and reimbursement of pharmaceutical products; the highly competitive nature of the pharmaceutical industry; Allergan's ability to successfully navigate consolidation of our distribution network and concentration of our customer base; the difficulty of predicting the timing or outcome of pending or future litigation or government investigations; developments regarding products once they have reached the market; risks related to Allergan's incorporation in Ireland, such as changes in Irish law and such other risks and other uncertainties detailed in Allergan's periodic public filings with the Securities and Exchange Commission, including but not limited to Allergan's Annual Report on Form 10-K for the year ended December 31, 2016 and Quarterly Report on Form 10-Q for the quarter ended June 30, 2017; and from time to time in Allergan's other investor communications. Except as expressly required by law, Allergan disclaims any intent or obligation to update or revise these forward-looking statements.

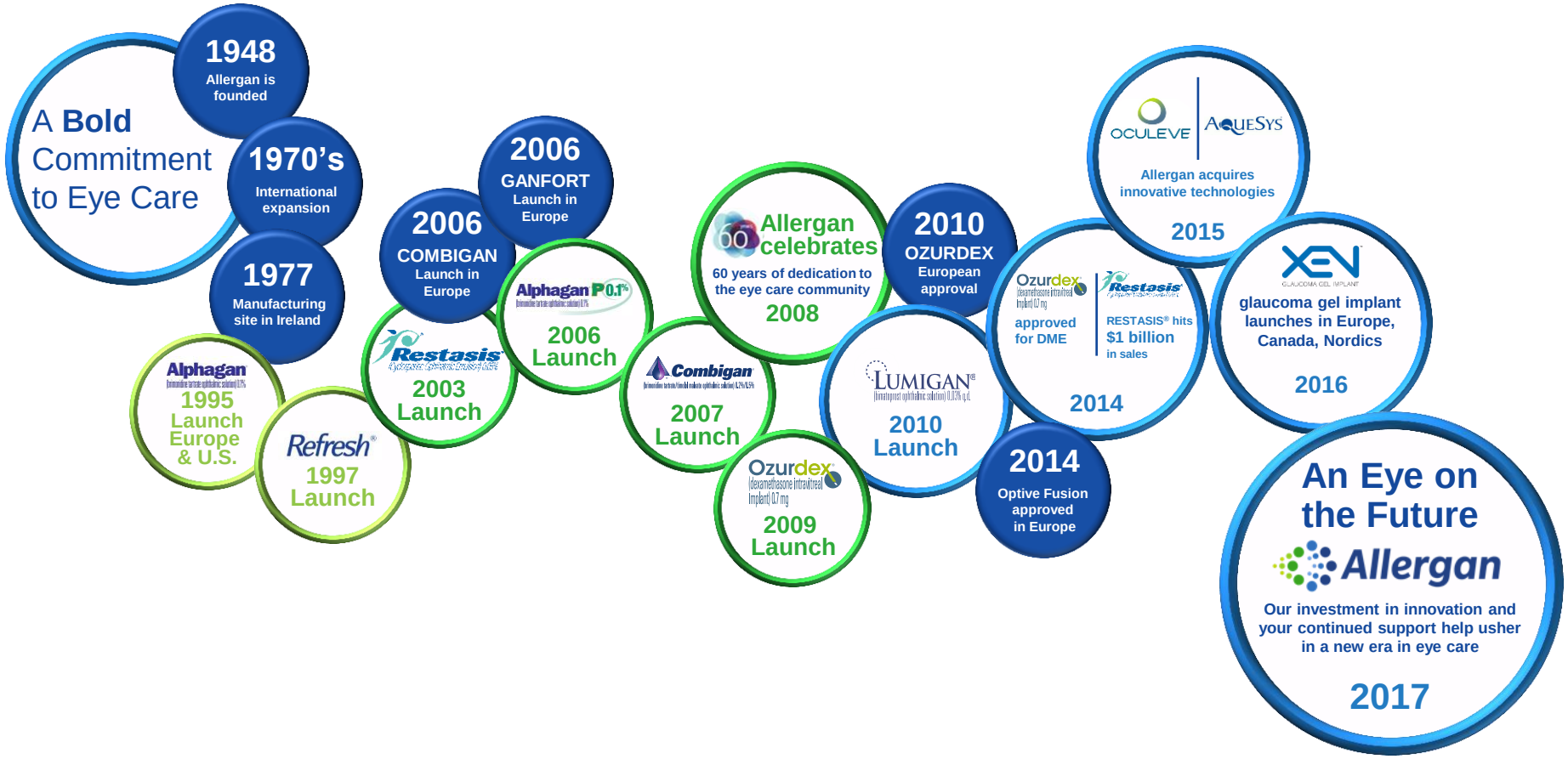
Non-GAAP Financial Measures

This document contains non-GAAP financial measures. The Appendix hereto presents reconciliations of certain non-GAAP financial measures to the most directly comparable GAAP measures. The non-GAAP measures include non-GAAP performance net income, non-GAAP performance net income per share, adjusted EBITDA, non-GAAP operating income and other non-GAAP financial statement line items.

The Company believes that its non-GAAP measures provide useful information to investors because these are the financial measures used by our management team to evaluate our operating performance, make day to day operating decisions, prepare internal forecasts, communicate external forward looking guidance to investors, compensate management and allocate the Company's resources. We believe this presentation also increases comparability of period to period results.

The Company's determination of significant charges or credits may not be comparable to similar measures used by other companies and may vary from period to period. The Company uses both GAAP financial measures and the disclosed non-GAAP adjusted financial measures internally. These non-GAAP adjusted financial measures are in addition to, not a substitute for, or superior to, measures of financial performance prepared in accordance with GAAP.

ALLERGAN EYE CARE: HISTORICAL LEADERSHIP



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ALLERGAN EYE CARE LEADERSHIP GLOBALLY

AGN Ranked in Top 5

> AGN Presence in ~ 73 Countries
> AGN Ranked #1 or #2 in 26 Countries
> Strong Double-Digit Growth in 30+ Countries

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GAPS IN WET AMD TREATMENT TODAY DRIVING TOMORROW'S INNOVATIONS

Monthly/PRN dosing schedule for majority of anti-VEGFs

Treatment and monitoring required in a patient population including working adults and patients with bilateral disease

Do not get 3-line gains after 1 year of use in trials (9-12 injections)

Control with fewer injections to stop or reverse severe vision loss

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ABICIPAR IS A NOVEL BEST-IN-CLASS ANTI-VEGF THERAPY THAT ALLOWS A GREATER PERCENTAGE OF nAMD PATIENTS TO IMPROVE AND MAINTAIN VISION

- **nAMD: Large US Market of ~$3.7B growing at 4.7% CAGR 2016 to 2021[1]**
  - Anti-VEGFs are the Standard of Care in nAMD with 97% Unit Share
  - Focused and Efficient Customer Base: ~ 2,400 Retina Specialists[2]

- **Transformational product will change nAMD treatment with practical dosing**
  - New DARPin technology enables higher affinity and longer duration
  - May reduce treatment and ease patient, caregiver and practice burden

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Source: 1. Mixture of IMS, actual sales data and estimates, 2. Allergan Internal, Wolters Kluwer claims data, IMS

ABICIPAR VALUE PROPOSITION DIRECTLY ADDRESSES MARKET NEED

> **Abicipar is an advanced Anti-VEGF therapy with potential to allow the greatest number of nAMD patients to improve and maintain vision with 12 week dosing**

- Equivalent 3-line vision gains as Lucentis
- Longer dosing interval than Lucentis
- Clinically acceptable safety profile

> **Rationale**

- Patients do not maintain the vision gains seen in clinical trials due to the required current anti-VEGF treatment schedules
- The higher affinity of Abicipar for VEGF-A and longer vitreous half-life extends injection intervals which means sustained vision with less frequent injections
- Phase 2 REACH trial demonstrated potential for dosing every 12 weeks

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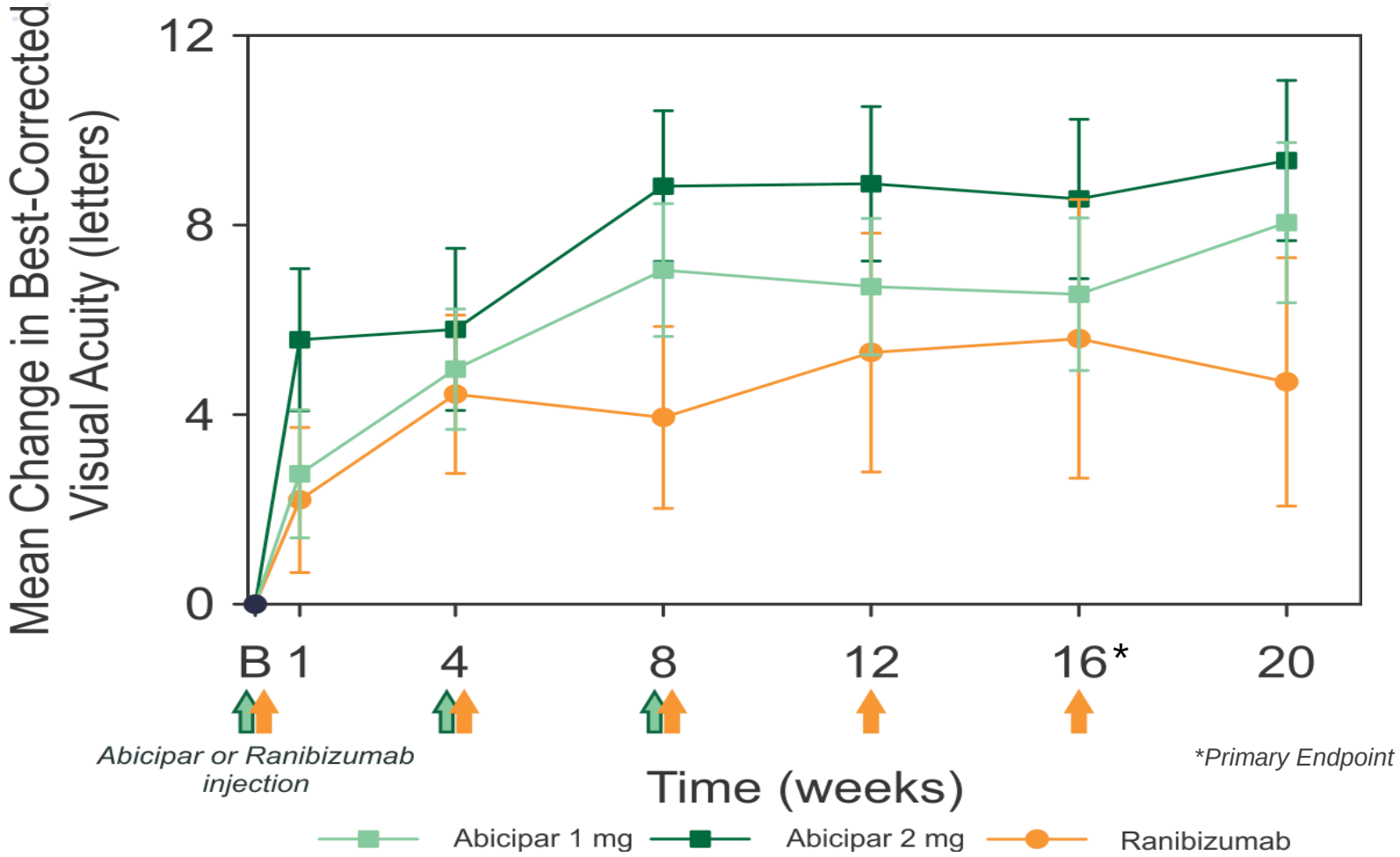
REACH STAGE 3
MEAN CHANGE IN BCVA FROM BASELINE



mITT (treated patients with more than one measurement) with LOCF; SE = standard error of the mean

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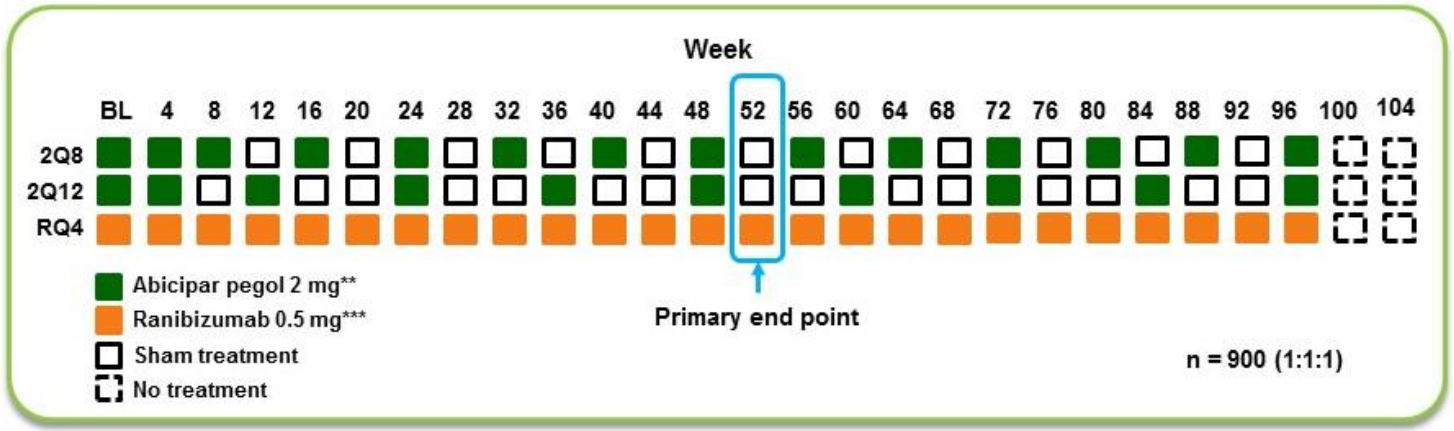
Presented at AAO Chicago in 2014 by Raj Maturi, MD

ClinicalTrials.gov Identifier: NCT01397409; REACH: United States, Australia, Austria, Switzerland, France, Germany, Israel, Italy

ABICIPAR PHASE 3 STUDY DESIGN ALLOWS SIGNIFICANTLY FEWER DOSES



CEDAR
SEQUOIA

Powered to show efficacy at q8 and q12 weeks

Primary endpoint

- Proportion of patients with stable vision (loss of ≤ 15 ETDRS letters compared to baseline)

Secondary endpoints

- Mean change from baseline in ETDRS BCVA
- Mean change from baseline in CRT
- Proportion of patients with ≥ 15-letter ETDRS gain
- Mean change from baseline in NEI-VFQ-25 composite score

Abicipar-treated eyes are receiving fewer doses over 2 years than eyes treated with monthly ranibizumab

- 6-8 vs 12 doses in the first year
- 4-6 vs 12 doses in the second year

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ClinicalTrials.gov Identifier: NCT02462928, Cedar.
ClinicalTrials.gov Identifier: NCT02462486; Sequoia.

*Loss of < 15 ETDRS letters compared to baseline
**2Q8: 8 doses 1 year, 14 total. 2Q12: 6 doses 1 year, 10 doses total
***RQ4: 14 doses 1 year, 25 doses total

ABICIPAR POTENTIAL

> Abicipar for the Treatment of nAMD

- Abicipar will be the first and only DARPin® therapeutic specifically designed to deliver maximum potency and inhibit VEGF in the eye for an extended period of time
- The DARPin® therapeutic platform enables the anti-VEGF efficacy of abicipar, effectively easing the treatment burden with a dosing schedule that will complement real-world clinical practice patterns

> Performance and Benefits

- OCT results in phase 2 clinical trials show that abicipar quickly neutralizes VEGF with a rapid onset of action
- Abicipar provides a high level of functional vision with stable, consistent results

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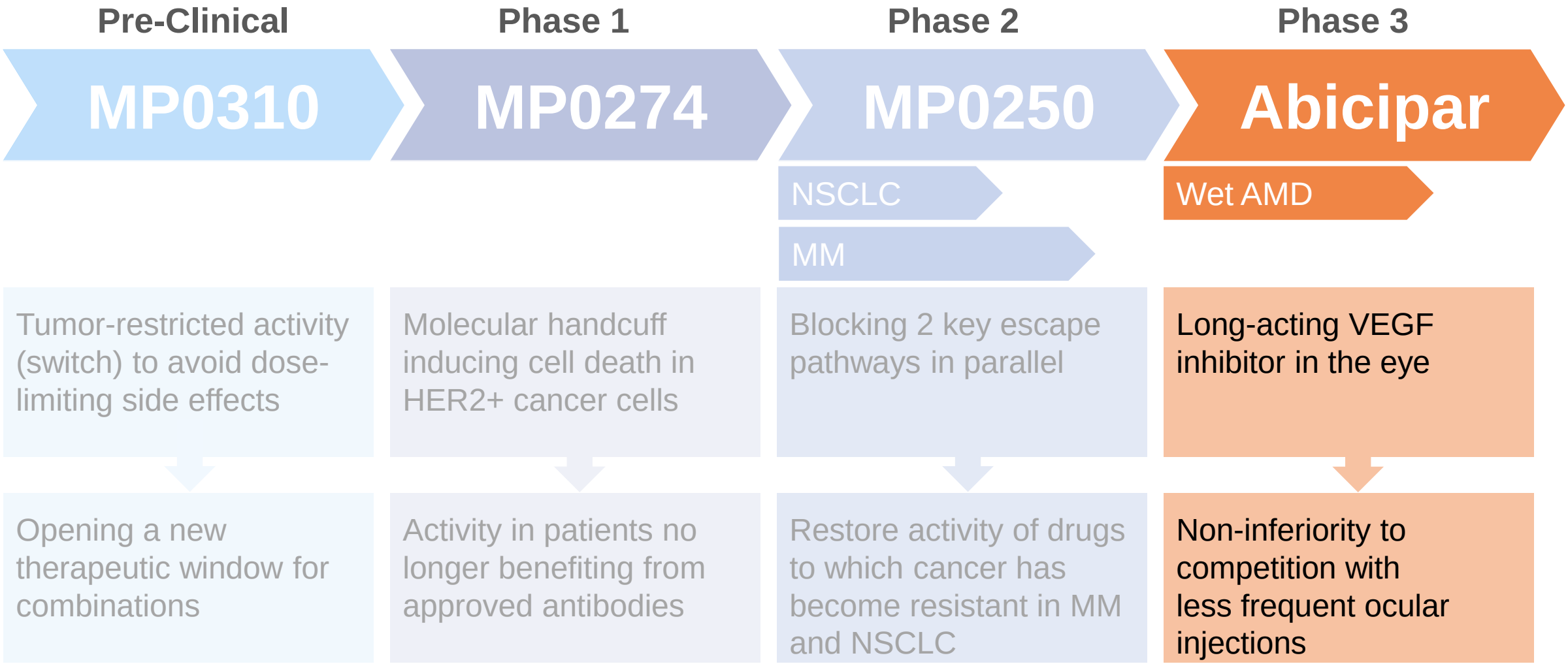
KEY HIGHLIGHTS OF THE ABICIPAR PROGRAM

- **Abicipar development program is on track**
- **Phase 3 topline planned for 2nd Half of 2018**
- **Launch of abicipar targeting 2020**

Conclusions from Our Ophthalmology Pipeline



Conclusions

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Key messages for today

- Successful transition from DARPin® platform into clinical product company
- Key value inflection points ahead:
  - MP0250 (2x Phase 2) and MP0274 (Phase 1) in oncology
  - Abicipar (Phase 3 data) in ophthalmology
- MP0310 selected as 1st development candidate from our I/O DARPin® toolbox
- Financed beyond 2019, capturing key value inflection points
- Keep on forward integrating towards late-stage development and the market

Q&A

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End

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