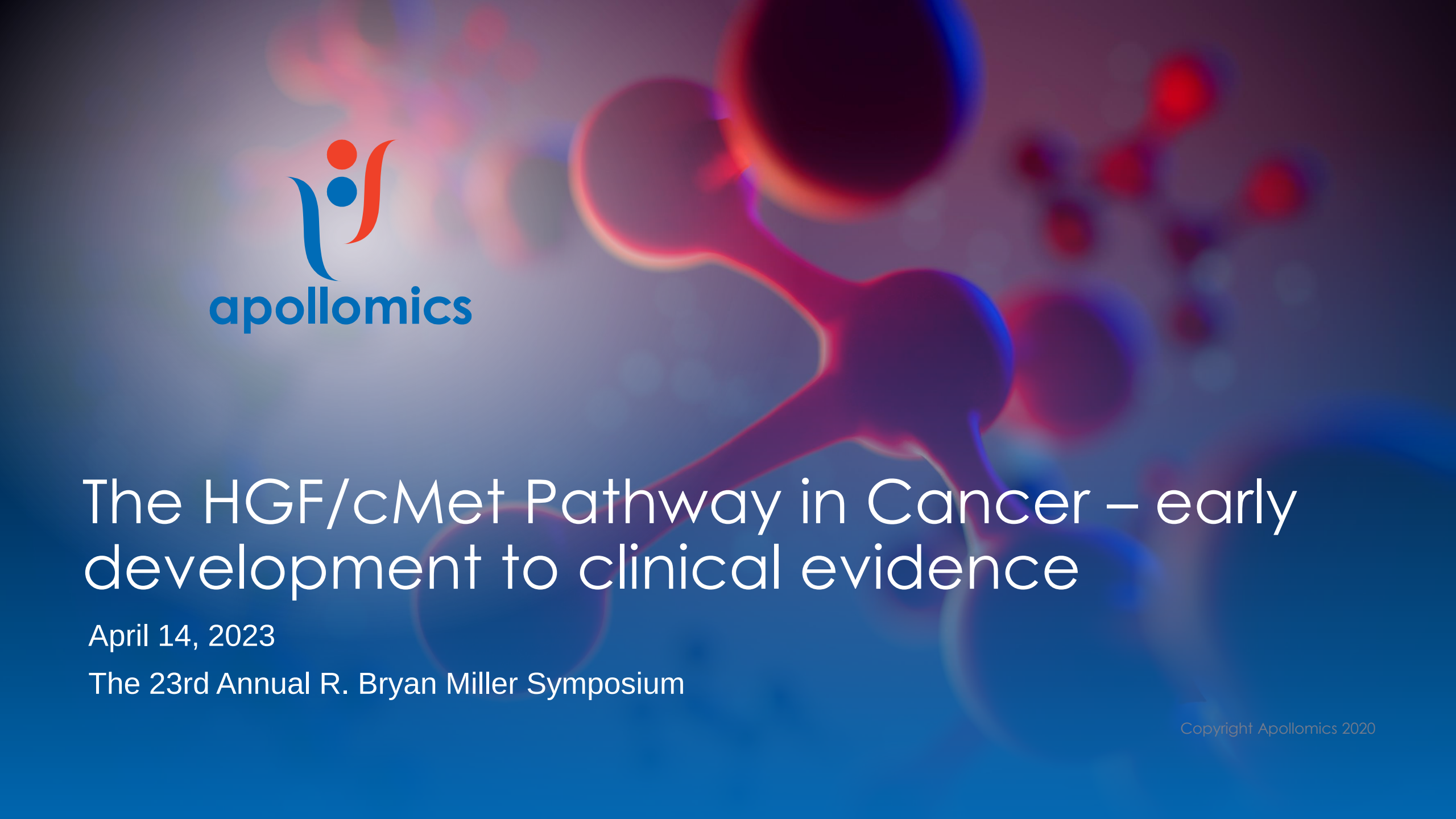


The HGF/cMet Pathway in Cancer – early development to clinical evidence

April 14, 2023

The 23rd Annual R. Bryan Miller Symposium

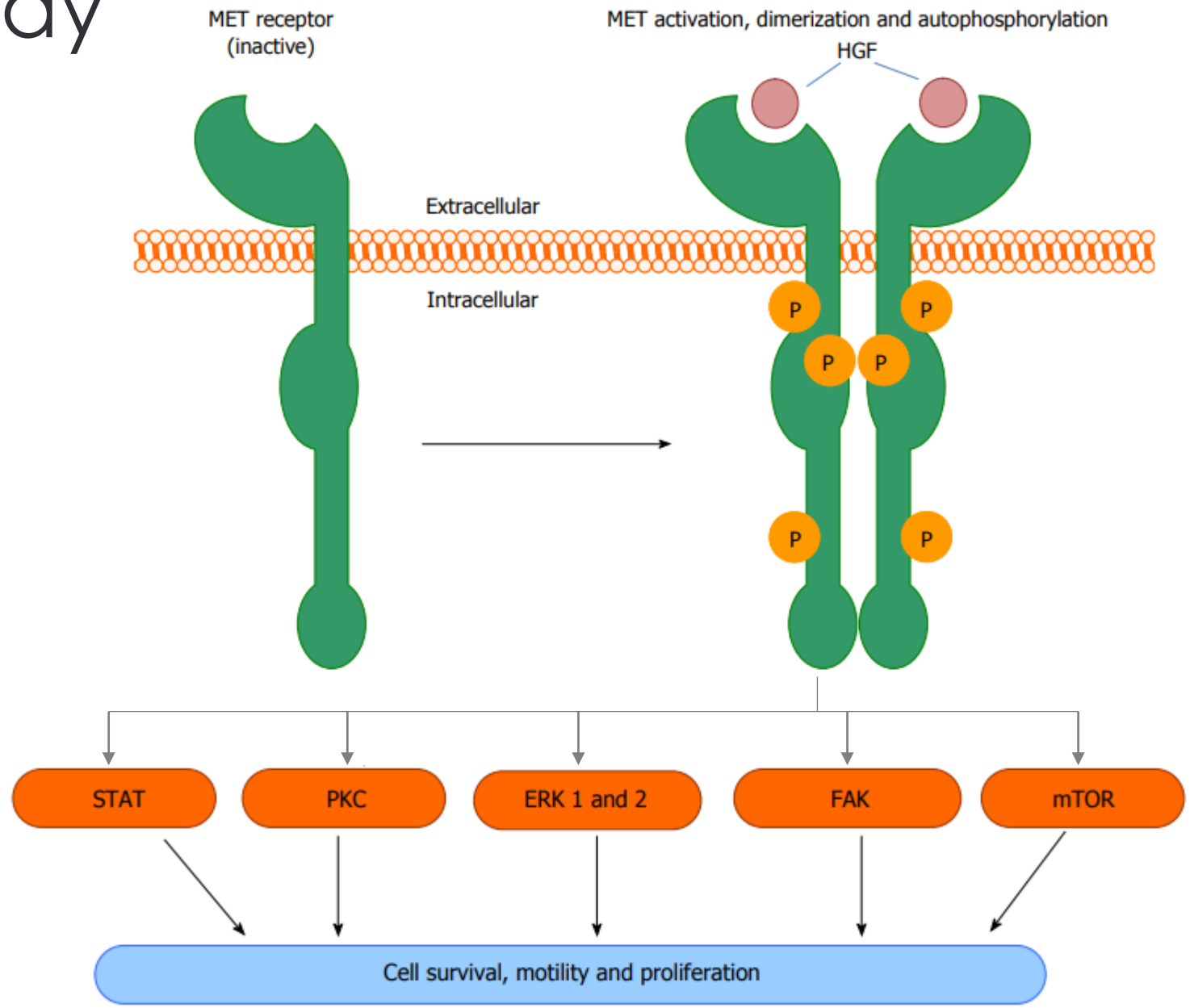
Forward-Looking Statements

This presentation includes statements that constitute “forward-looking statements” within the meaning of the federal securities laws, including Section 27A of the Securities Act of 1933, as amended (the “Securities Act”), and Section 21E of the Securities Exchange Act of 1934, as amended (the “Exchange Act”). All statements, other than statements of present or historical fact included in this press release, regarding the Company’s future financial performance, as well as the Company’s strategy, future operations, revenue guidance, projected costs, prospects, plans and objectives of management are forward-looking statements. When used in this press release, the words “could,” “should,” “will,” “may,” “believe,” “anticipate,” “intend,” “estimate,” “expect,” “project,” the negative of such terms and other similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain such identifying words. These forward-looking statements are based on management’s current expectations and assumptions about future events and are based on currently available information as to the outcome and timing of future events. Apollomics cautions you that these forward-looking statements are subject to numerous risks and uncertainties, most of which are difficult to predict and many of which are beyond the control of Apollomics. In addition, Apollomics cautions you that the forward-looking statements contained in this press release are subject to unknown risks, uncertainties and other factors, including: (i) the impact of any current or new government regulations in the United States and China affecting Apollomics’ operations and the continued listing of Apollomics’ securities; (ii) the inability to achieve successful clinical results or to obtain licensing of third-party intellectual property rights for future discovery and development of Apollomics’ oncology projects; (iii) the failure to commercialize product candidates and achieve market acceptance of such product candidates; (iv) the failure to protect Apollomics’ intellectual property; (v) breaches in data security; (vi) risks related to the ongoing COVID-19 pandemic and response; (vii) the risk that Apollomics may not be able to develop and maintain effective internal controls; (viii) unfavorable changes to the regulatory environment; and those risks and uncertainties discussed in the Form F-4 (as amended) filed by Apollomics, Inc. with the U.S. Securities and Exchange Commission (“SEC”) under the heading “Risk Factors” and the other documents filed, or to be filed, by the Company with the SEC. Other unknown or unpredictable factors also could have material adverse effects on the Company’s future results and/or could cause our actual results and financial condition to differ materially from those indicated in the forward-looking statements. Should one or more of the risks or uncertainties described in this press release materialize or should underlying assumptions prove incorrect, actual results and plans could differ materially from those expressed in any forward-looking statements. New risk factors that may affect actual results or outcomes emerge from time to time and it is not possible to predict all such risk factors, nor can Apollomics assess the impact of all such risk factors on its business, or the extent to which any factor or combination of factors may cause actual results to differ materially from those contained in any forward-looking statements. Forward-looking statements are not guarantees of performance. You should not put undue reliance on these statements, which speak only as of the date hereof. Additional information concerning these and other factors that may impact the operations and projections discussed herein can be found in the reports that Apollomics has filed and will file from time to time with the SEC. These SEC filings are available publicly on the SEC’s website at www.sec.gov. Apollomics undertakes no obligation to update publicly any of these forward-looking statements to reflect actual results, new information or future events, changes in assumptions or changes in other factors affecting forward-looking statements, except to the extent required by applicable laws. If Apollomics updates one or more forward-looking statements, no inference should be drawn that Apollomics will make additional updates with respect to those or other forward-looking statements.

This presentation contains discussions of investigational products that are under preclinical or clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration (FDA). Investigational products are currently limited by Federal law to investigational use, and no representations are made as to their safety or effectiveness for the purposes for which they are being investigated.

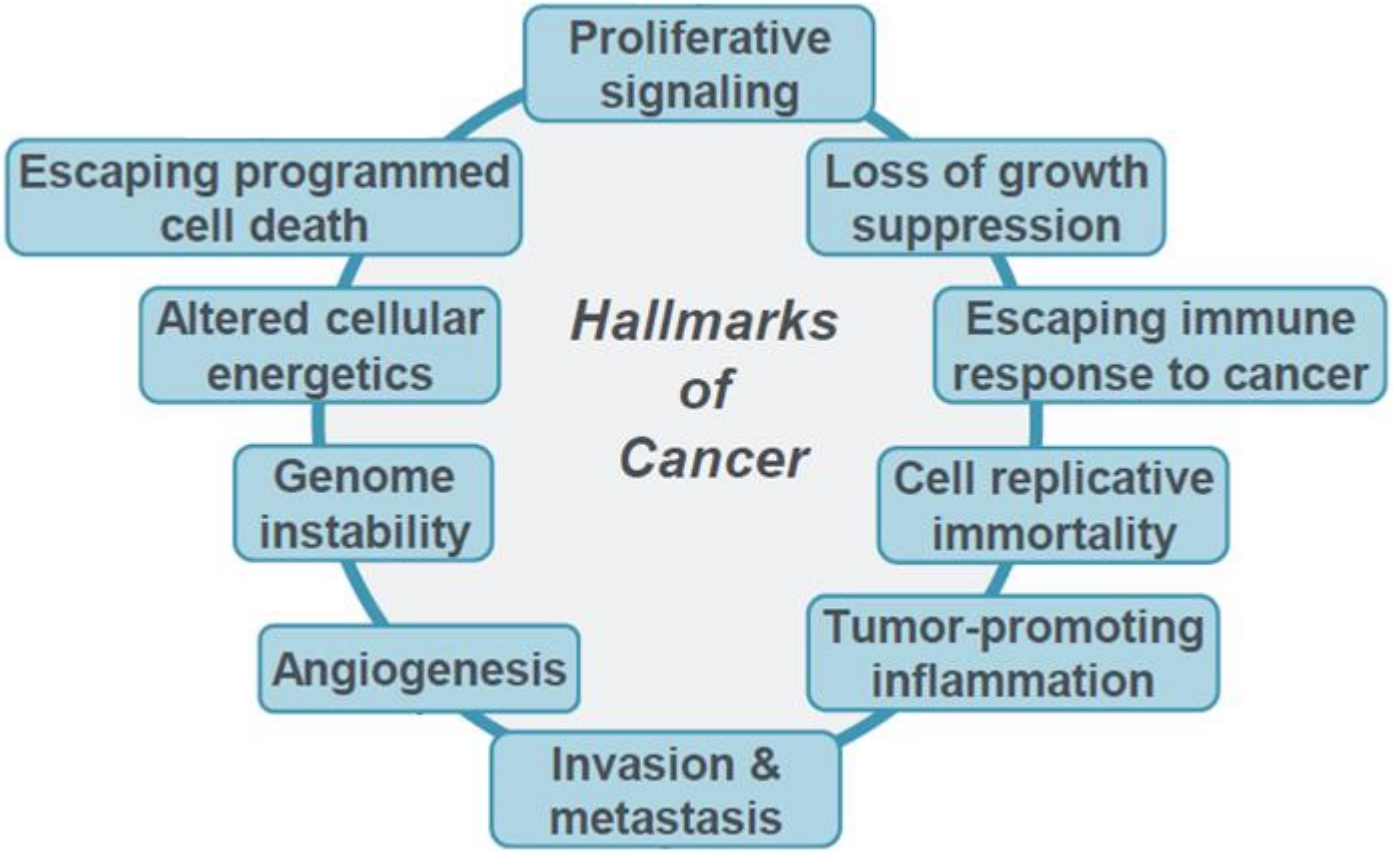
HGF-cMET Pathway

- The Mesenchymal-Epithelial Transition (MET) factor receptor is a transmembrane tyrosine kinase receptor expressed on epithelial cells
- Binding to its stromal ligand, the Hepatocyte Growth Factor (HGF), leads to activation of the HGF-Met pathway
- Normal Met involved in tissue homeostasis – embryonic development, organ regeneration & wound healing.
- Pathway activation in cancers leads to a host of intracellular signaling inducing proliferation, motility, migration and invasion



Delitto D et. al. 2014

Hallmarks of Cancer



Hanahan and Weinberg, Cell 2011

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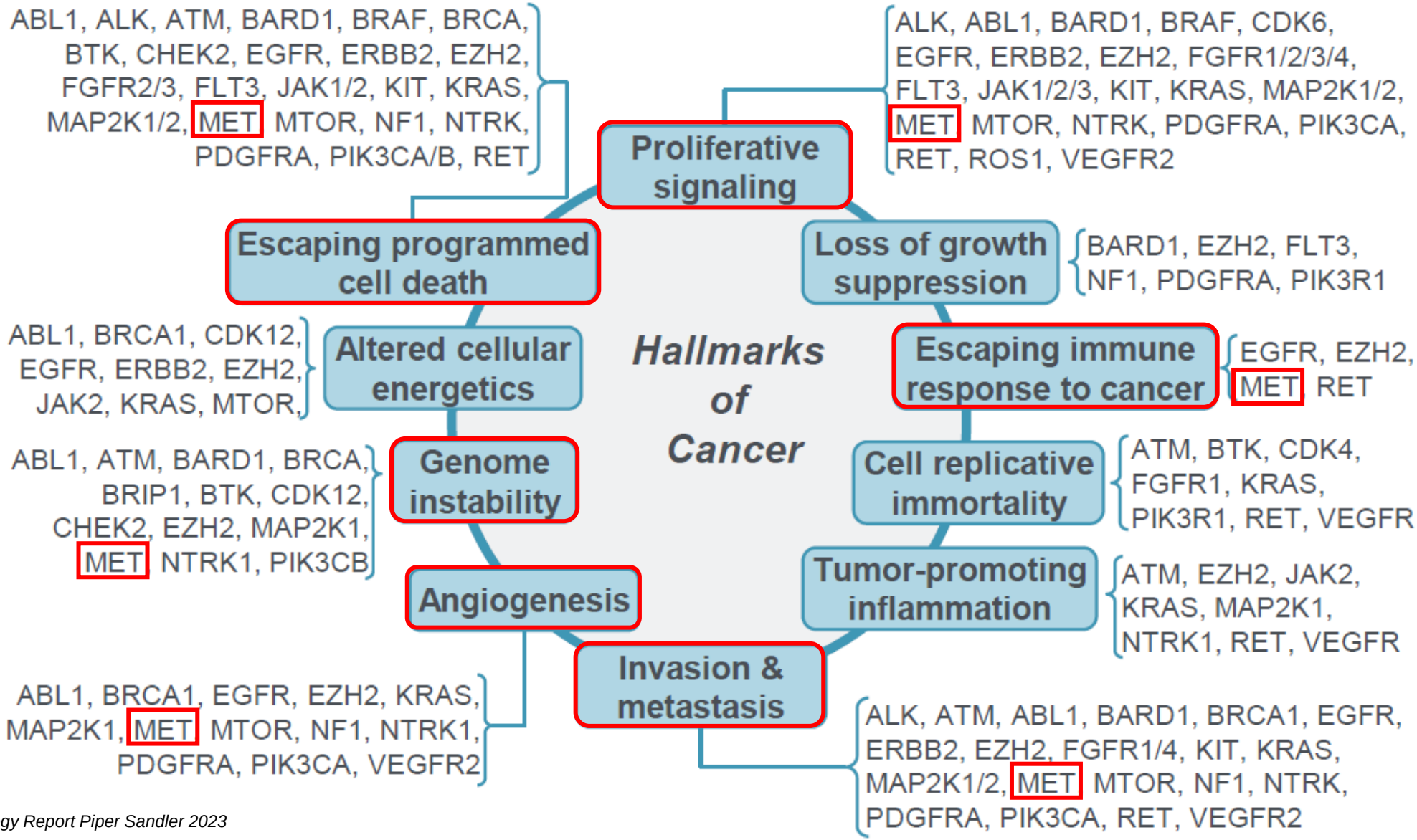
Hallmarks of Cancer – Gene Alterations



Precision Oncology Report Piper Sandler 2023

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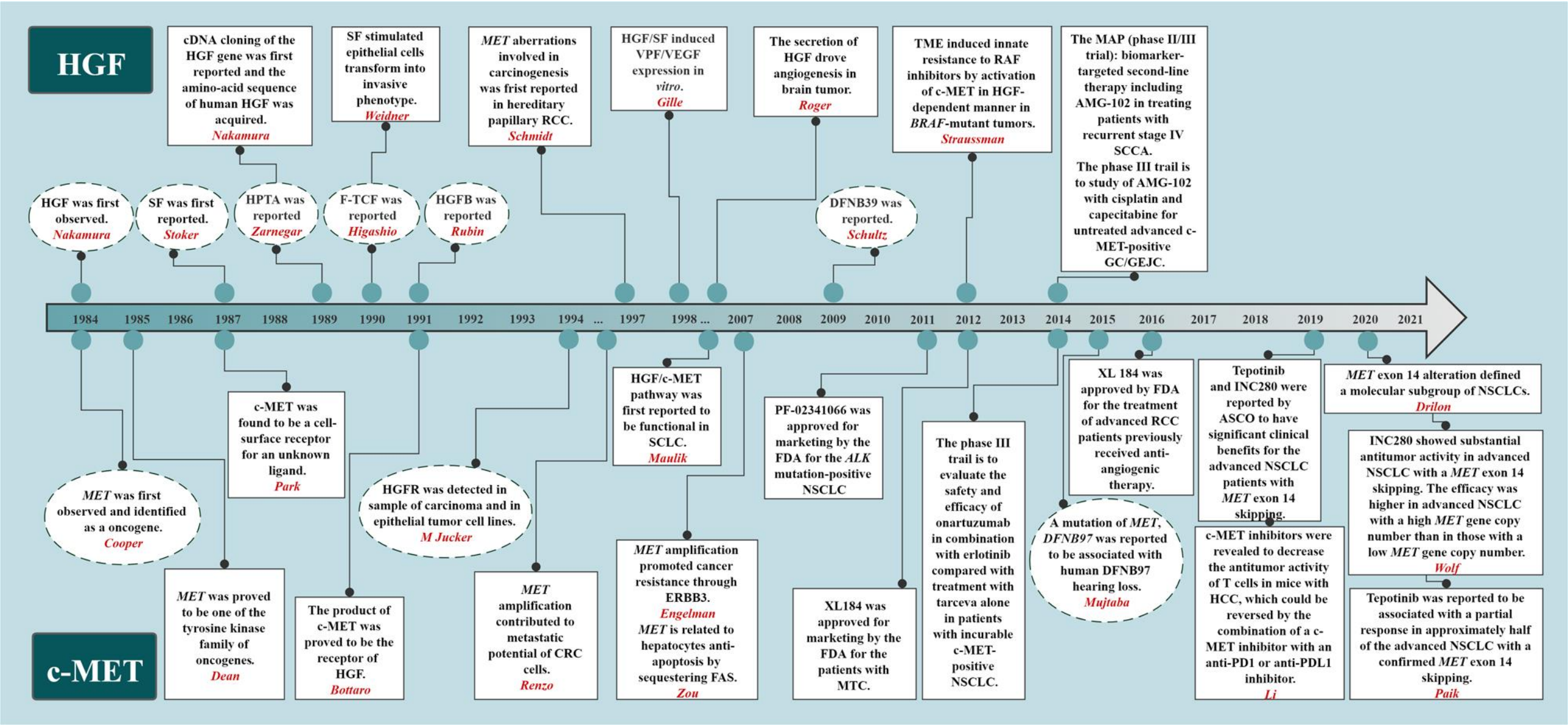
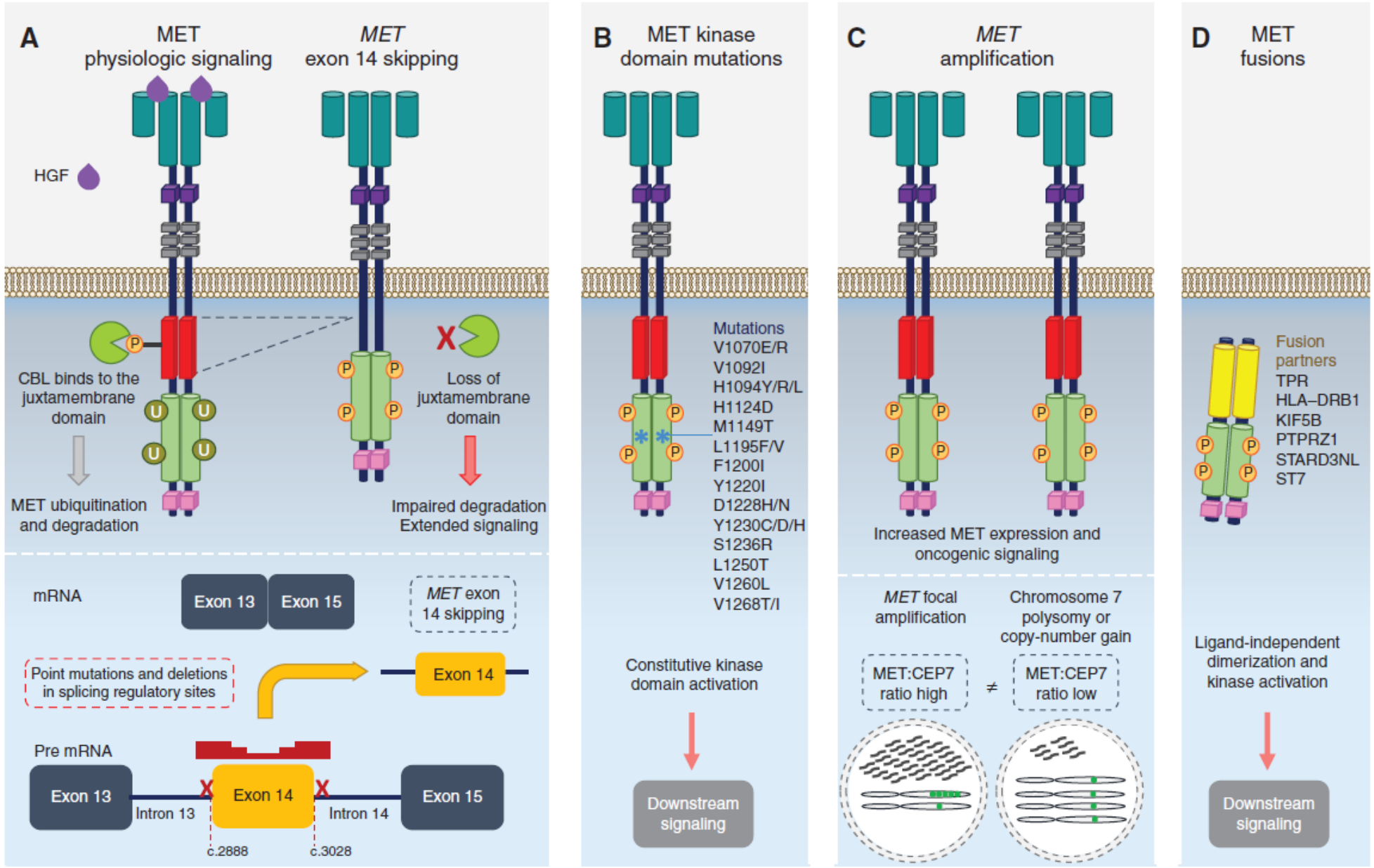
Timeline from ’84 to 1st approval in 2020

Jianjiang Fu et. al. 2021



HGF/Met Pathway is activated in multiple dysregulations



G. Recondo et. al. 2020

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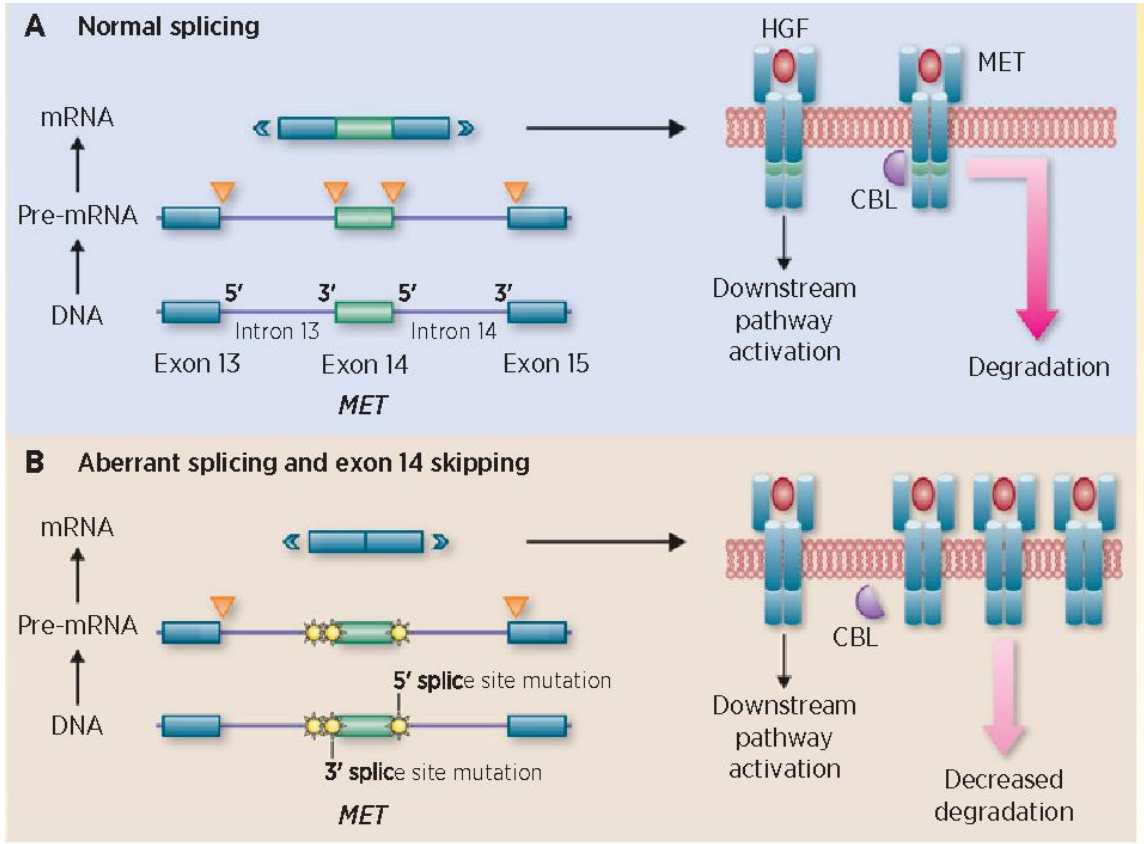
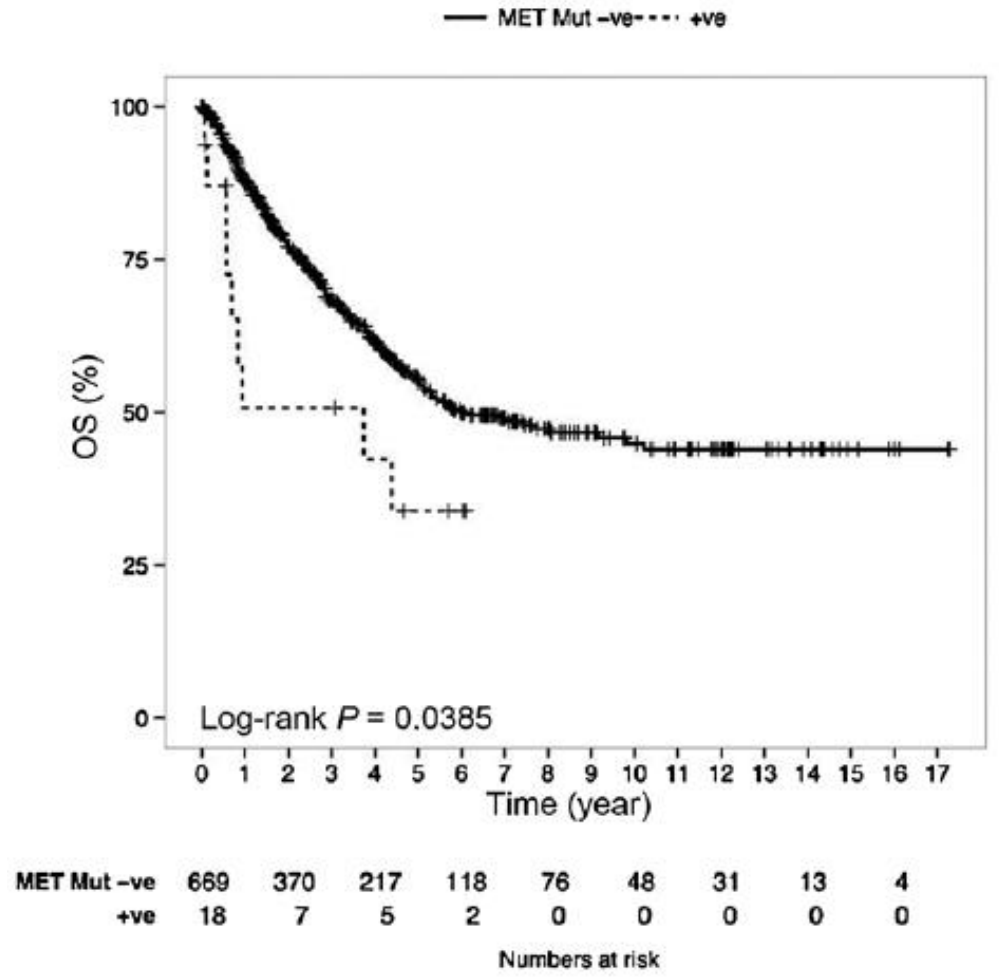
MET Exon 14 skipping mutations

- MET exon 14 skipping mutations ↑ MET protein levels
  - loss of Cbl binding and protein ubiquitination
- 3% incidence in non-squamous NSCLC[1]
- 22.2% in pulmonary sarcomatoid carcinoma[2]





1. *Awad et. al. 2016*
2. *Liu et al. 2016*
3. *Tong et al 2016*

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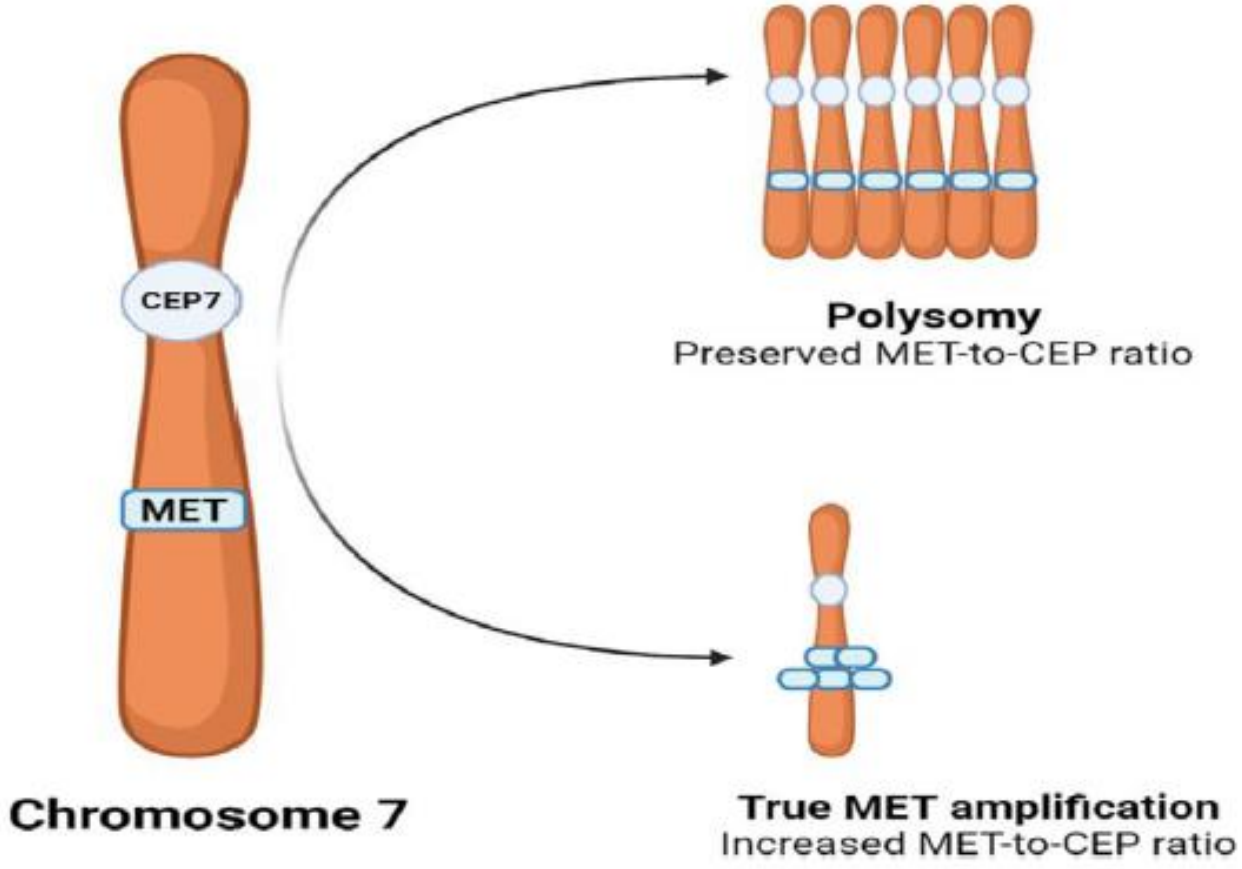
MET Amplification

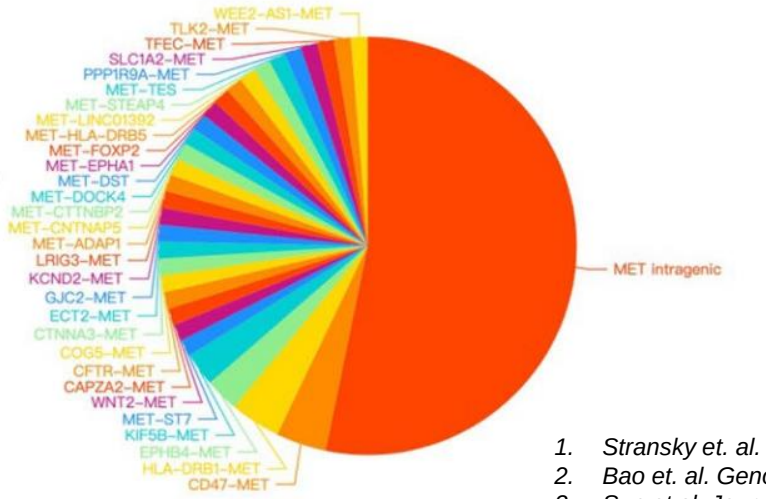
- MET copy number gains consist of polysomy or amplification.
- De novo amplification is 1%–5% of untreated NSCLC tumors and with a strong smoking association
- 15-30% as resistance to TKIs in EGFR+ NSCLC; one of the resistance mechanisms in other mutated NSCLCs.
- Can be detected by FISH (MET/CEP7 > 2) or by GCN using Next Gen Sequencing (NGS) > 5 or 6



Remon at al. 2022

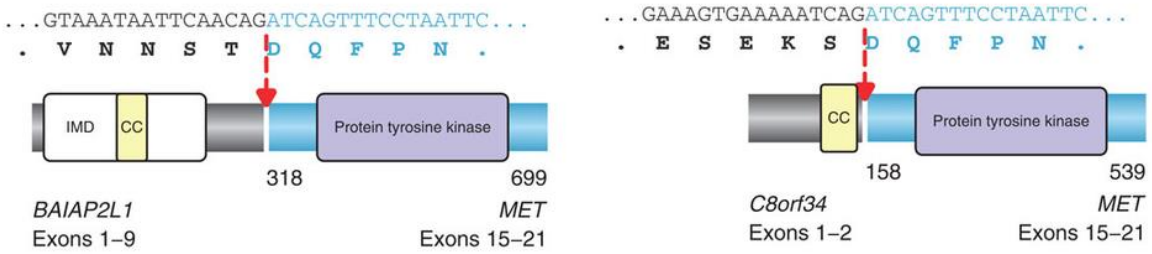
MET Fusions

- First identified in patient samples in 2014[1]
  - BAIAP2L1–MET and C8orf34–MET in RCC
  - PTPRZ1-MET in low grade gliomas
  - OXR1-MET in HCC
  - KIF5B-MET in lung adenocarcinomas
  - TFG-MET in thyroid carcinomas
- PRPRZ1-MET fusions seen in 15% of secondary GBM[2]
- MET fusions in lung cancer[3]

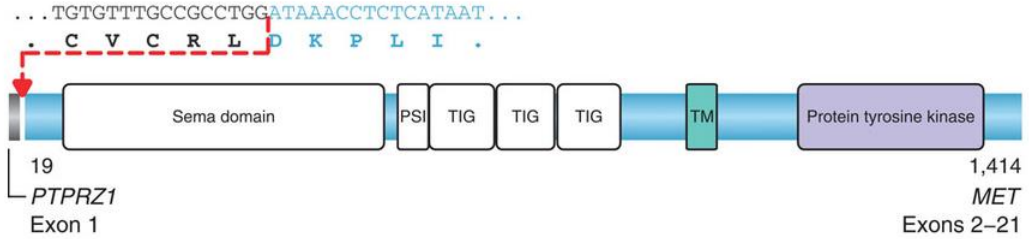


1. *Stransky et. al. 2014 Nature Comm.*
2. *Bao et. al. Genome Res. 2016*
3. *Sun et al. Jour. Trans. Med 2023*

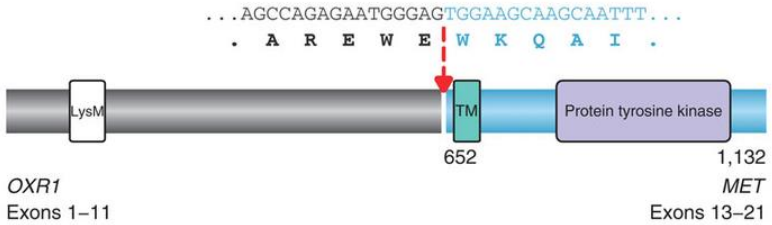
Papillary Renal Cell Carcinoma



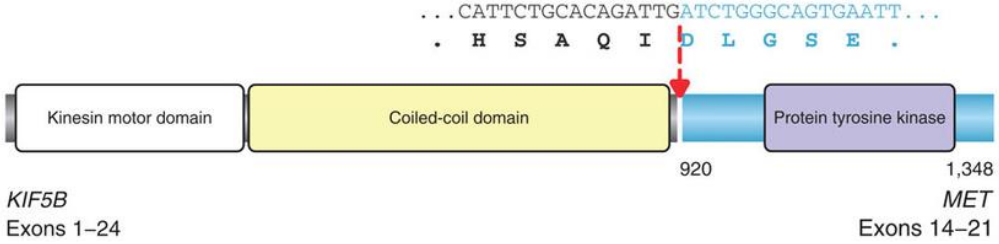
Low grade Gliomas



Hepatocellular Carcinoma



Lung adenocarcinoma



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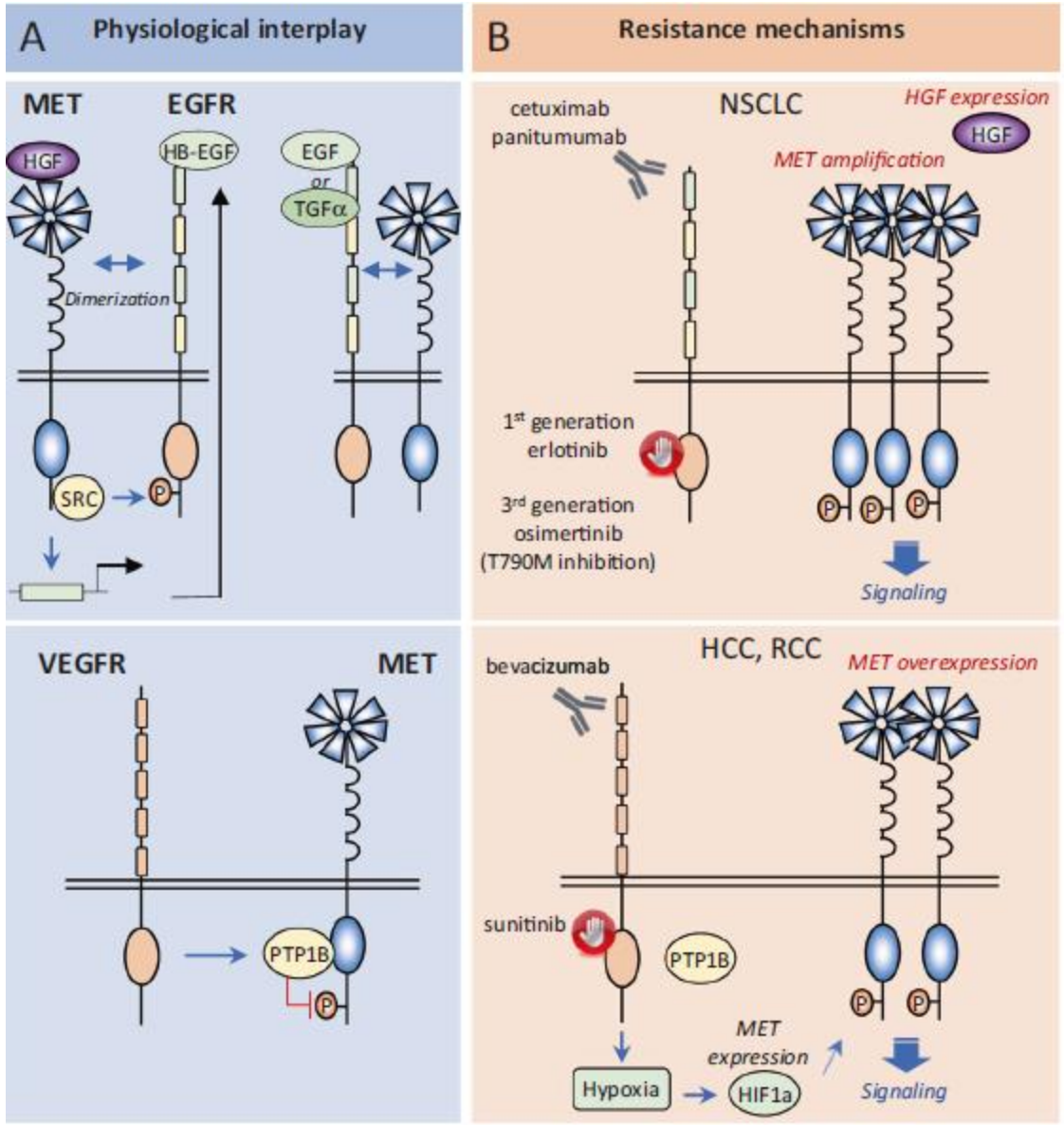
MET driven Resistance Mechanisms



- MET activation through MET gene amplification acts as a bypass pathway leading to resistance to EGFR TKIs in NSCLC
- MET amplification promotes an aggressive phenotype in EGFR mutated cells: increased cell proliferation, anchorage independent growth, and migration, leading to an increased capacity to metastasize

- In HCC and RCC, inhibition by the anti-VEGF bevacizumab restores MET phosphorylation – makes MET one of the main suspects in resistance to VEGF/VEGFR inhibitors
- Associated with an increase in both HIF1a and MET expression

Fernandes et al 2021

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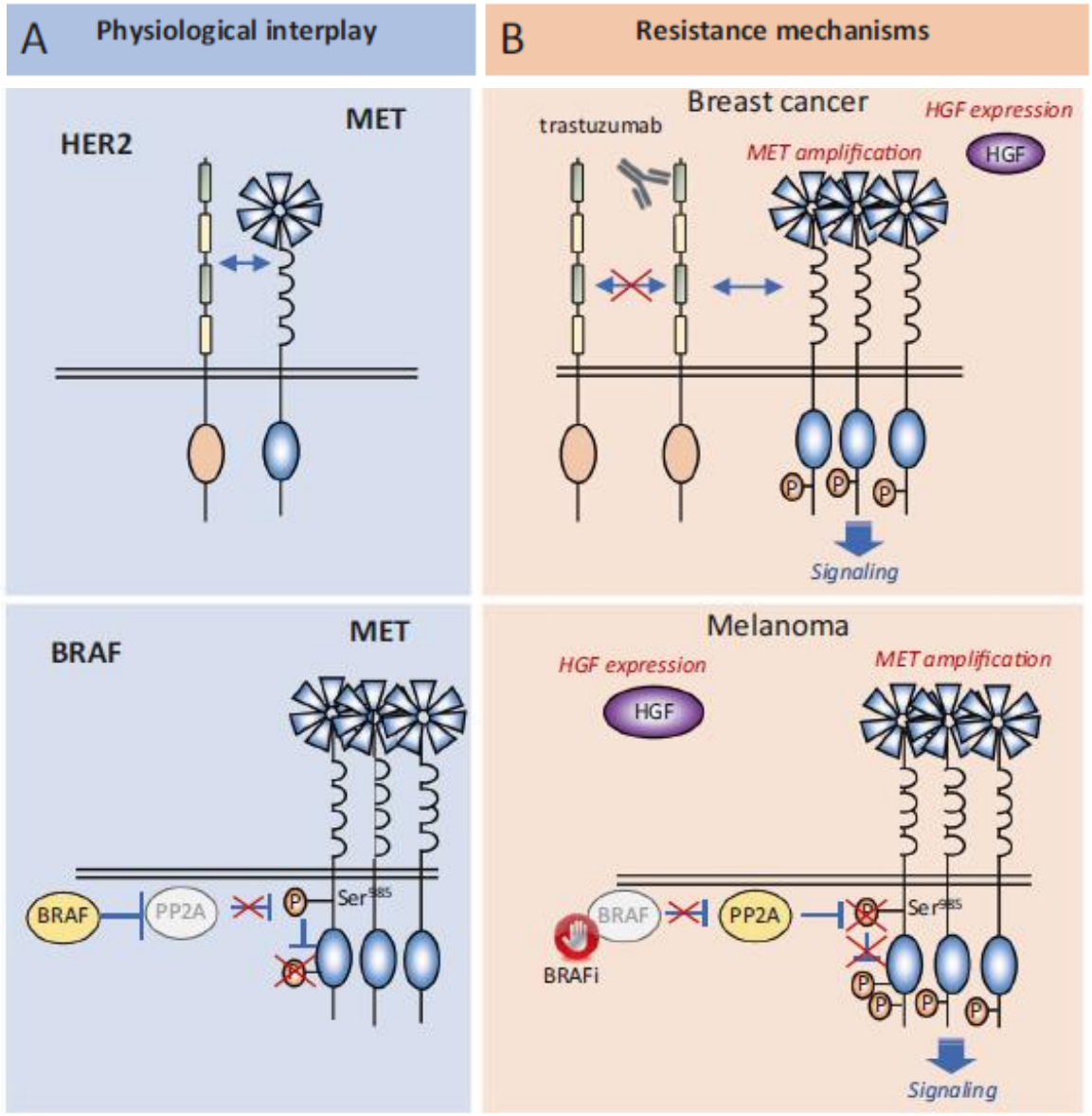
MET driven Resistance Mechanisms



- Tumors resistant to anti-HER2 treatments showed higher MET expression
- MET amplification is observed in approximately 1/4th of HER2+ breast cancer cases and is associated with a higher risk of trastuzumab therapy failure
- HGF overexpression has also been detected in trastuzumab resistant tumors

- HGF-induced resistance in melanoma was dependent on MET
- BRAF activation leads to inhibition of MET activity, and MET amplification remains functionally dormant. Treatment with a BRAFi reactivates this alteration.

Fernandes et al 2021

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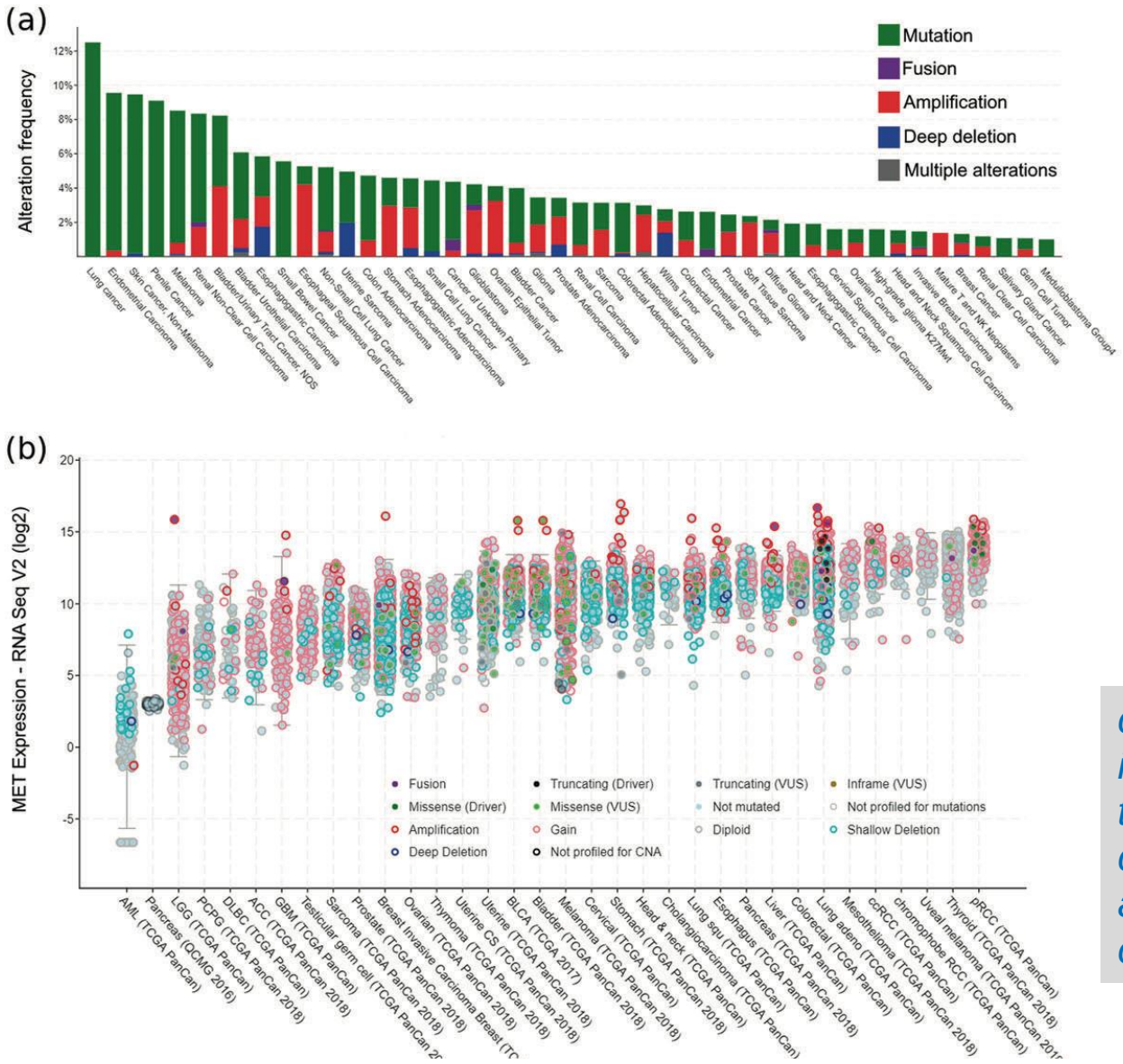
c-Met is a Target Relevant Across Multiple Tumor Types



c-Met is involved in proliferation, motility, migration and invasion. Although c-MET is important in the control of tissue homeostasis under normal physiological conditions, it has also been found to be aberrantly activated in human cancers via mutation, amplification or protein overexpression.

Koch et al; Oncogene (2020) 39:2845–2862

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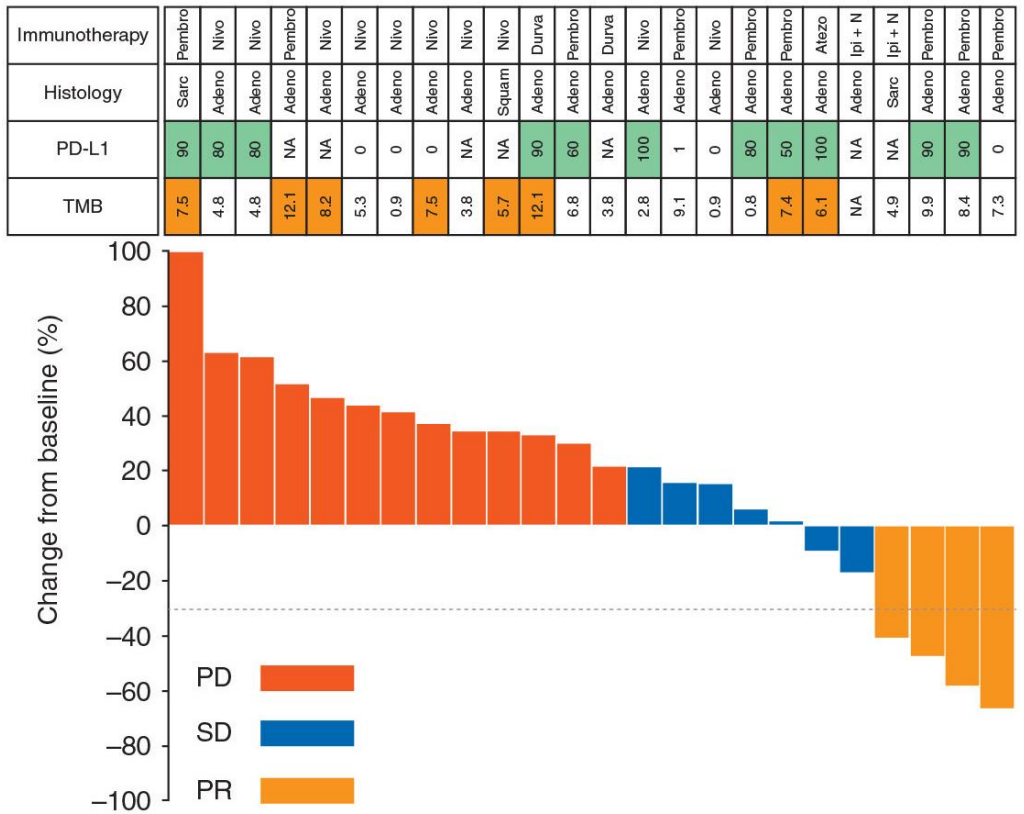
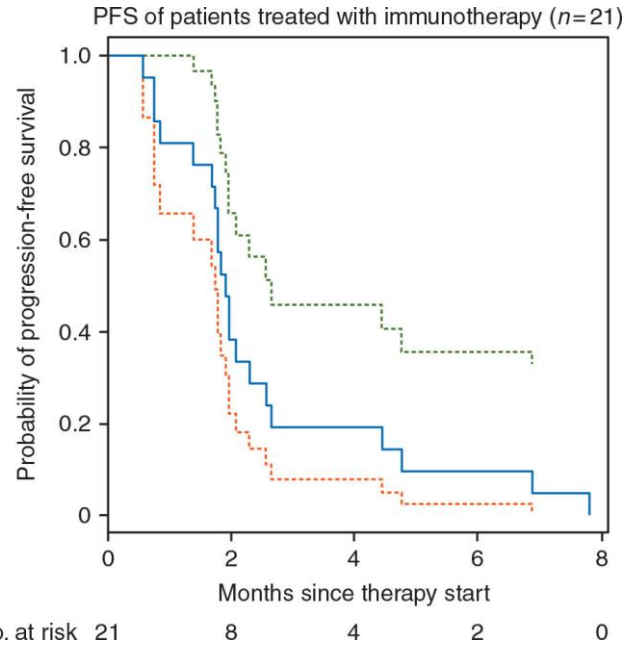
Immune Checkpoint Inhibitors in Met Ex14 NSCLC

- Outcomes achieved with single-agent immunotherapy in this setting are poor
- ORR with immune checkpoint inhibition was low at 17% (low n)
- Response was not associated with PD-L1 expression





Sabari et al; Annals of Oncology 2018

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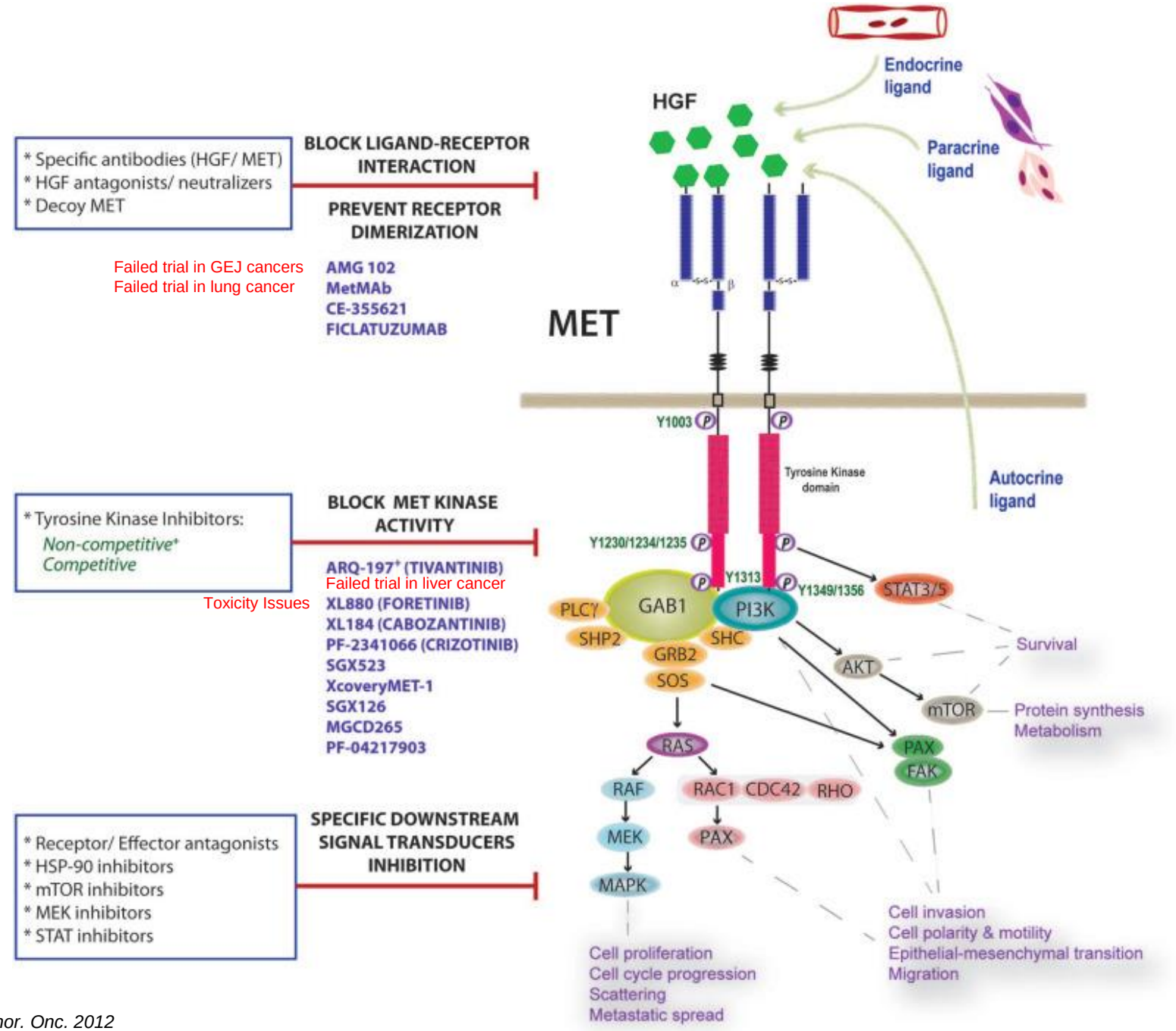
Early Challenges Inhibiting the MET-HGF Pathway

- cMet expression as a biomarker
- Multi targeted TKIs



Yan Feng et al J. Thor. Onc. 2012

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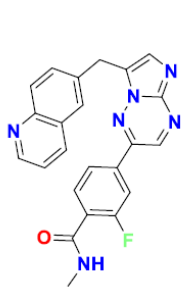
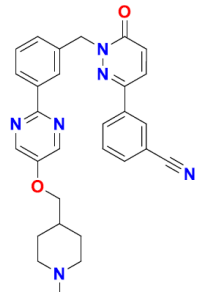
Recent therapeutic approaches

- Highly specific tyrosine kinase inhibitors (small molecule)
  - Capmatinib
  - Tepotinib
  - Savolitinib

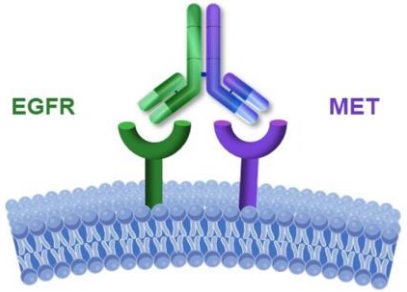


Capmatinib

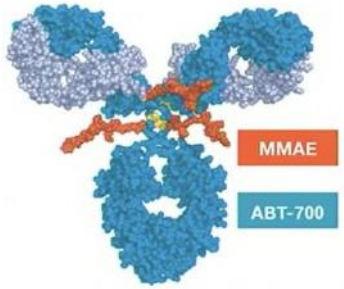


Tepotinib

- Bispecific Antibodies
  - Amivantamab bispecific antibody
  - Approved in Exon 20 EGFRm NSCLC
  - Trials ongoing in Met dysregulated cancers



- cMet ADCs
  - Telisotuzumab vedotin
  - In MET expressing NSCLC
  - Breakthrough Therapy designation in Met-high NSCLC



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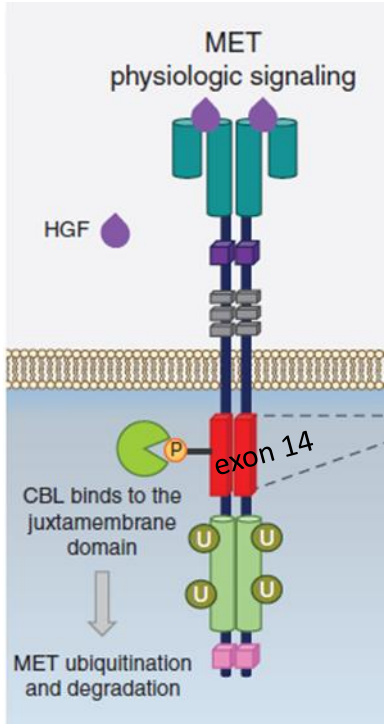
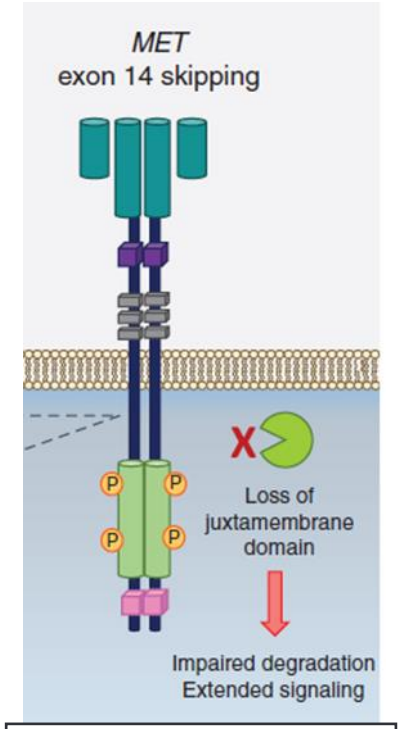
HGF/Met Pathway is activated in multiple dysregulations

Wild Type
HGF-dependent



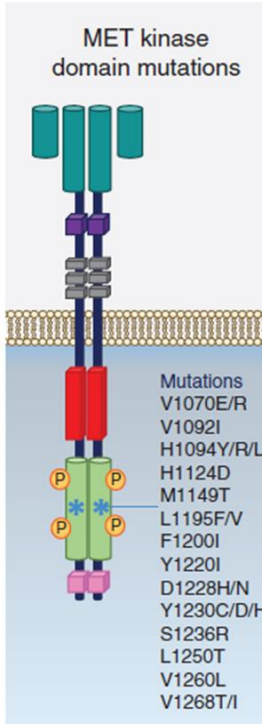
Genomic Alteration
HGF-independent



Clinically validated
- NSCLC –mono

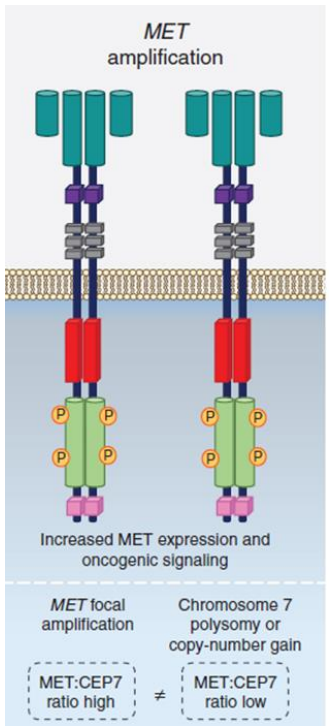
To be continued
- Brain Mets
- R/R patients

Genomic Alteration
HGF-independent



Open Clinical hypothesis
- Pan-cancer

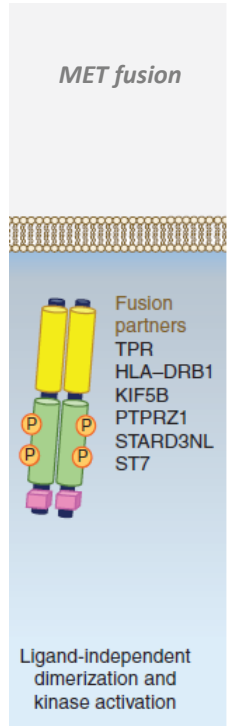
Gene Amplification
HGF-independent



Ongoing Clinical validation
- NSCLC (Driver-): mono
- NSCLC-(Driver+): combo

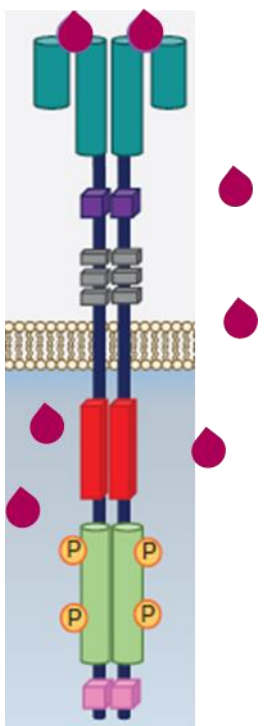
Open Clinical hypothesis
- Pan-cancer

Gene Rearrangement
HGF-independent



Ongoing Clinical validation
- NSCLC -mono
- GBM -mono
- Pan-cancer -mono

Autocrine HGF



Open Clinical hypothesis
- Pan-cancer

G. Recondo., J. Che., P.A. Jänne., M.M Awad; Cancer Discovery 2020

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GEOMETRY *mono-1*: Study Design

Patients with stage IIIB or IV NSCLC[a]
- Aged ≥ 18 years
- Any histology
- EGFR wild type
- ALK-negative
- MET dysregulation by central assessment
- ECOG PS ≤ 1
- ≥ 1 measurable lesion (as per RECIST 1.1)

Capmatinib 400 mg BID tablet

Cohort 1: Pretreated
- Subcohort 1a: *MET* GCN ≥ 10
- Subcohort 1b: *MET* GCN ≥ 6 and < 10

Cohort 2: Pretreated patients with *MET* GCN ≥ 4 and < 6

Cohort 3: Pretreated patients with *MET* GCN < 4 (n = 30)

Cohort 4: Pretreated patients with *MET* mutation regardless of *MET* GCN

Cohort 5: treatment-naive
- Subcohort 5a: *MET* GCN ≥ 10 and no *MET* mutation
- Subcohort 5b: *MET* exon mutation regardless of *MET* GCN

Cohort 6: Pretreated (1 prior line of therapy)
- Group 1: *MET* GCN ≥ 10 and no *MET* mutation
- Group 2: *MET* exon mutation regardless of *MET* GCN

Cohort 7: Treatment-naive patients with *MET* exon 14 mutation regardless of *MET* GCN

[a] Patients were allocated based on MET central molecular prescreening.

METex14 mutation positive cohort | *MET* amplified cohort

Wolf et al AACR 2020

Geometry Trial – Met Ex14 & Amp+ NSCLC

Table 2. Responses to Capmatinib Treatment, as Assessed by the Independent Review Committee.*

Response	NSCLC with *MET* Exon 14 Skipping Mutation		NSCLC with *MET* Amplification				
	Cohort 4 (N=69)	Cohort 5b (N=28)	Cohort 1a (N=69)	Cohort 5a (N=15)	Cohort 1b (N=42)	Cohort 2 (N=54)	Cohort 3 (N=30)
Best response — no. (%)							
Complete response	0	1 (4)	1 (1)	0	0	0	0
Partial response	28 (41)	18 (64)	19 (28)	6 (40)	5 (12)	5 (9)	2 (7)
Stable disease	25 (36)	7 (25)	28 (41)	4 (27)	17 (40)	20 (37)	14 (47)
Noncomplete response or nonprogressive disease	1 (1)	1 (4)	1 (1)	0	1 (2)	0	0
Progressive disease	6 (9)	1 (4)	12 (17)	4 (27)	15 (36)	21 (39)	6 (20)
Unknown or could not be evaluated	9 (13)	0	8 (12)	1 (7)	4 (10)	8 (15)	8 (27)
Overall response†							
No. of patients with overall response	28	19	20	6	5	5	2
Percent of patients (95% CI)	41 (29–53)	68 (48–84)	29 (19–41)	40 (16–68)	12 (4–26)	9 (3–20)	7 (1–22)
Disease control‡							
No. of patients with disease control	54	27	49	10	23	25	16
Percent of patients (95% CI)	78 (67–87)	96 (82–100)	71 (59–81)	67 (38–88)	55 (39–70)	46 (33–60)	53 (34–72)
Duration of response							
No. of events/no. of patients with response	23/28	11/19	15/20	6/6	3/5	4/5	2/2
Median duration of response (95% CI) — mo	9.7 (5.6–13.0)	12.6 (5.6–NE)	8.3 (4.2–15.4)	7.5 (2.6–14.3)	24.9 (2.7–24.9)	9.7 (4.2–NE)	4.2 (4.2–4.2)
Progression-free survival							
Progression or death — no. of patients	60	17	58	15	34	50	22
Median progression-free survival (95% CI) — mo	5.4 (4.2–7.0)	12.4 (8.2–NE)	4.1 (2.9–4.8)	4.2 (1.4–6.9)	2.7 (1.4–3.1)	2.7 (1.4–4.1)	3.6 (2.2–4.2)

Wolf et al NEJM 2020

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Clinical Data in Met Exon 14 skip NSCLC

	Capmatinib (marketed, Phase II data[1]) Full Approval		Tepotinib (marketed, Phase II data[2]) Accelerated Approval		Savolitinib (marketed, Phase II data[3]) Conditional Approval	
Indication	Metastatic NSCLC with exon 14 skipping mutation		Metastatic NSCLC with exon 14 skipping mutation		Metastatic NSCLC with exon 14 skipping mutation	
	Naïve (N=60)	Previously Treated (N=100)	Naïve (N=69)	Previously Treated (N=83)	Naïve (N=28)	Previously Treated (N=42)
ORR (Objective Response Rate)	68%	44%	43%	43%	46%	41%
mDOR (median Duration of Response)	16.6 months	9.7 months	10.8 months	11.1 months	5.6 months	5.6 months
DCR (Disease Control Rate)	96%	78%				
mPFS (median Progression-Free Survival)	12.4 months	5.4 months				
mOS (median Overall Survival)	20.8 months	13.6 months				

Note: 1. NCT02414139, ORR time frame: at least 18 weeks; Patients: 97(28 naïve patients; 69 previously treated patients). Source: FDA
Locations: United States, Argentina, Austria, Belgium, France, Germany, Israel, Italy, Japan, Korea(Republic of), Lebanon, Mexico, Netherlands, Norway, Russian Federation, Singapore, Spain, Sweden, Taiwan, United Kingdom
2. NCT02864992, ORR time frame: baseline up to 20 months; Patients: 152(69 naïve patients; 83 previously treated patients)
Locations: United States, Austria, Belgium, China, France, Germany, Israel, Italy, Japan, Korea(Republic of), Netherlands, Poland, Spain, Switzerland, Taiwan. Source: FDA
3. Savolitinib Approval in China

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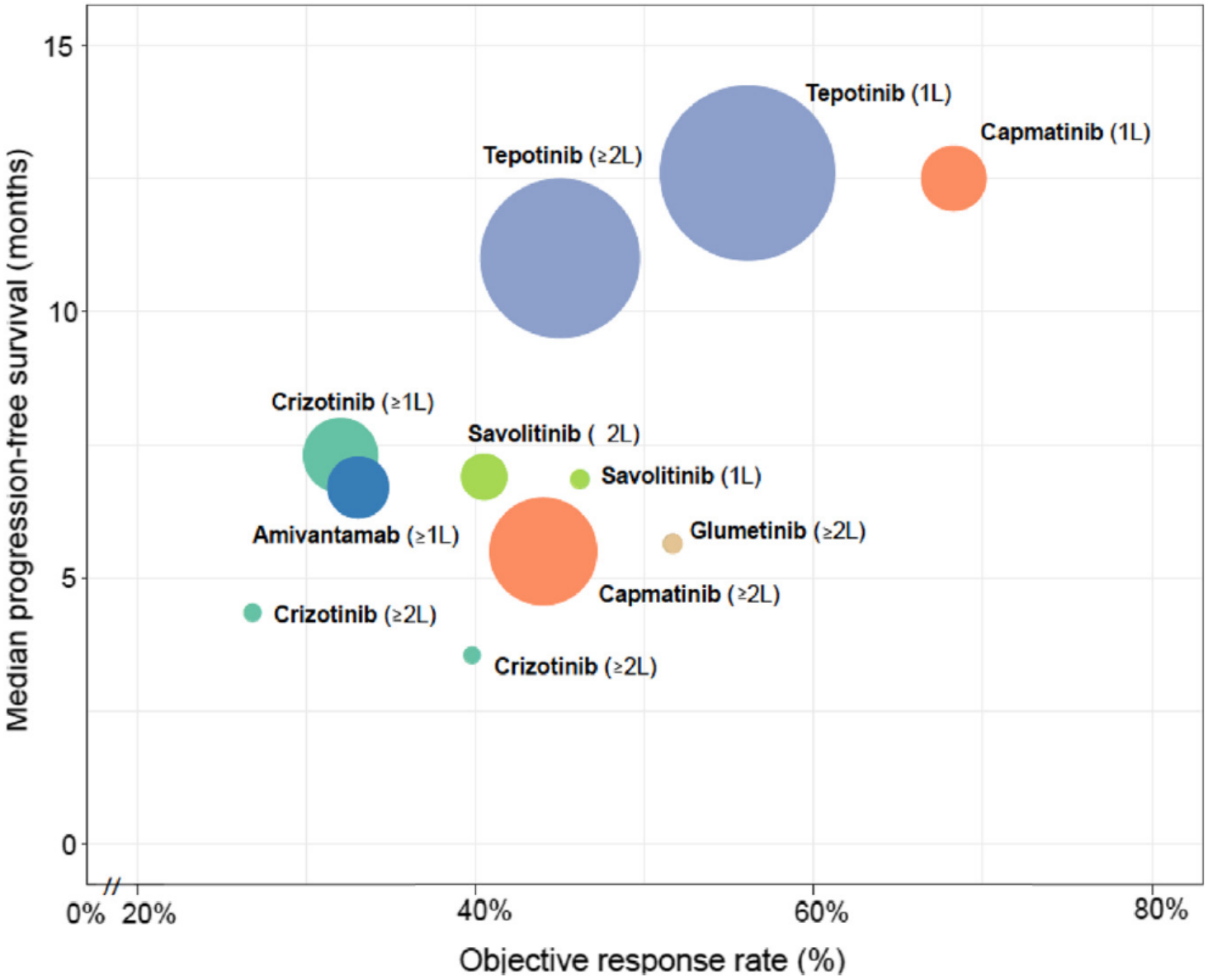
Efficacy of Met TKIs in Met Exon 14 skip NSCLC



Remon et al J. Thor. Onc.2022

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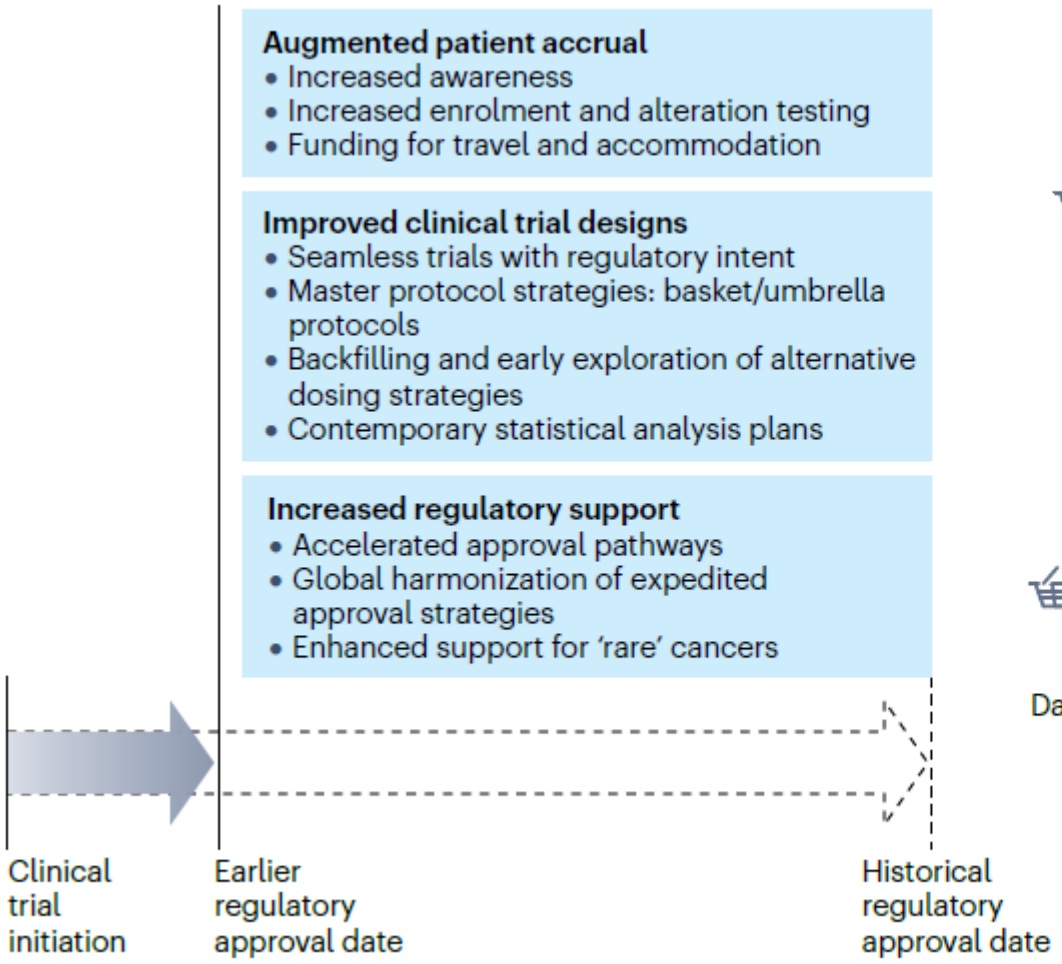
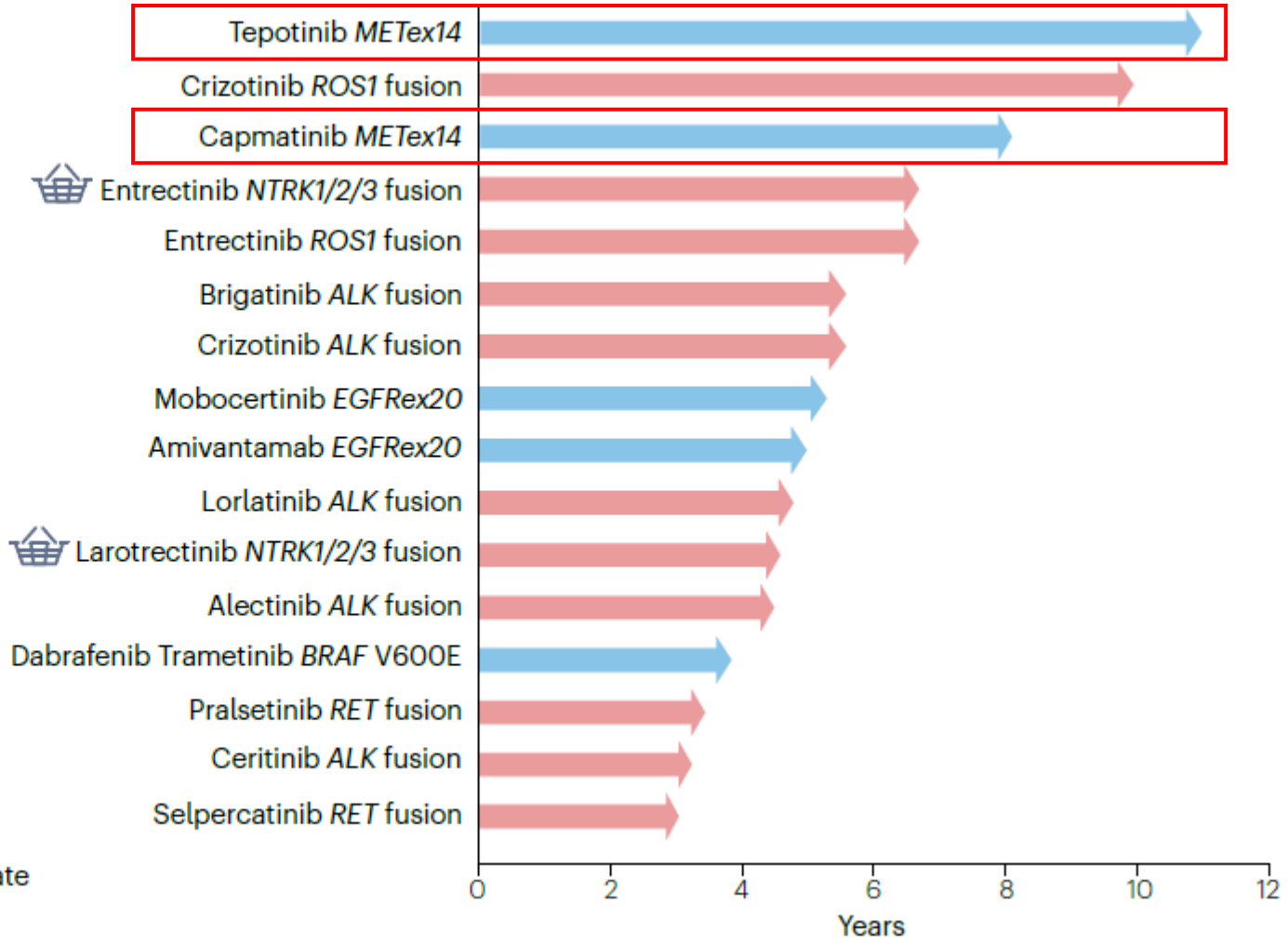
Development of TKIs in NSCLC molecular subtypes





Drilon et. al. Nature Reviews Clin. Oncol. 2023

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Apollomics: Innovative biopharma company

dedicated to leaving no cancer patient behind

Precision Medicine

Targeting difficult to treat cancers

Vebreltinib

Highly specific c-Met inhibitor with 3 near term NDA/sNDA opportunities

Uproleselan

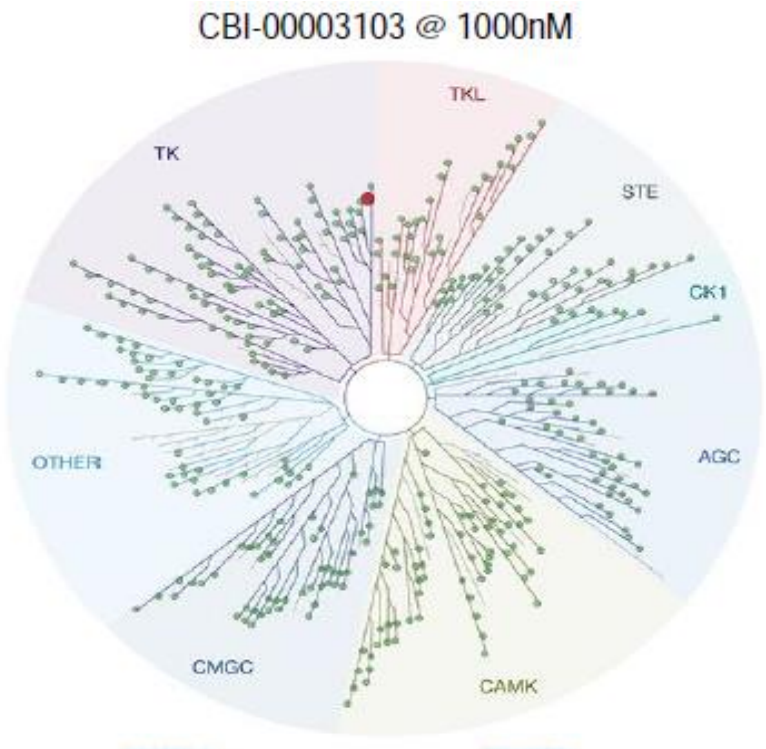
E-selectin antagonist in late-stage trials in acute myeloid leukemia

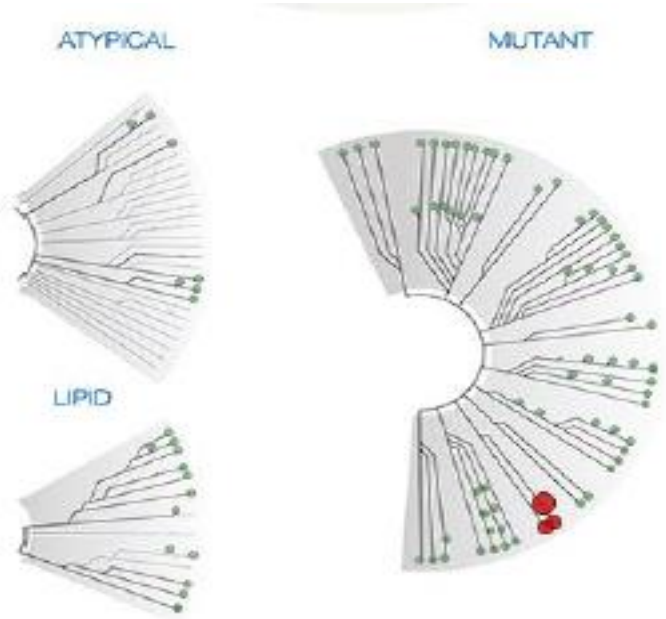
Vebreltinib (APL-101/PLB1001)

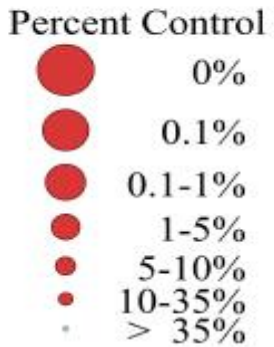
Specific Type 1b c-Met Inhibitor

Only inhibits c-Met
out of 473 total kinases (IC50 = 31 nM)
Intracellular Assay (IC50 = 0.5 nM)

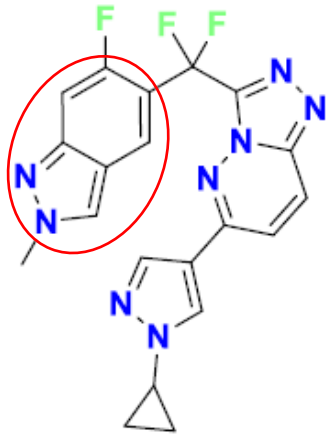
Central Nervous System Activity







Vebreltinib



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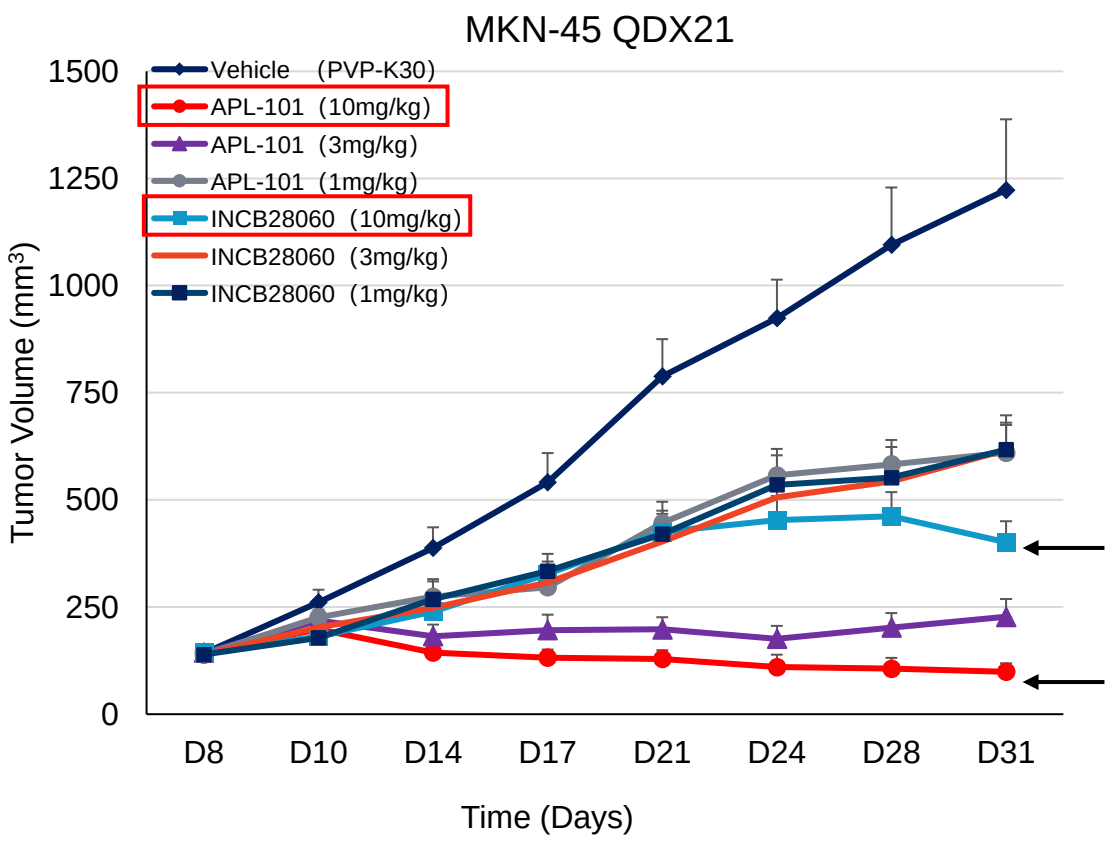
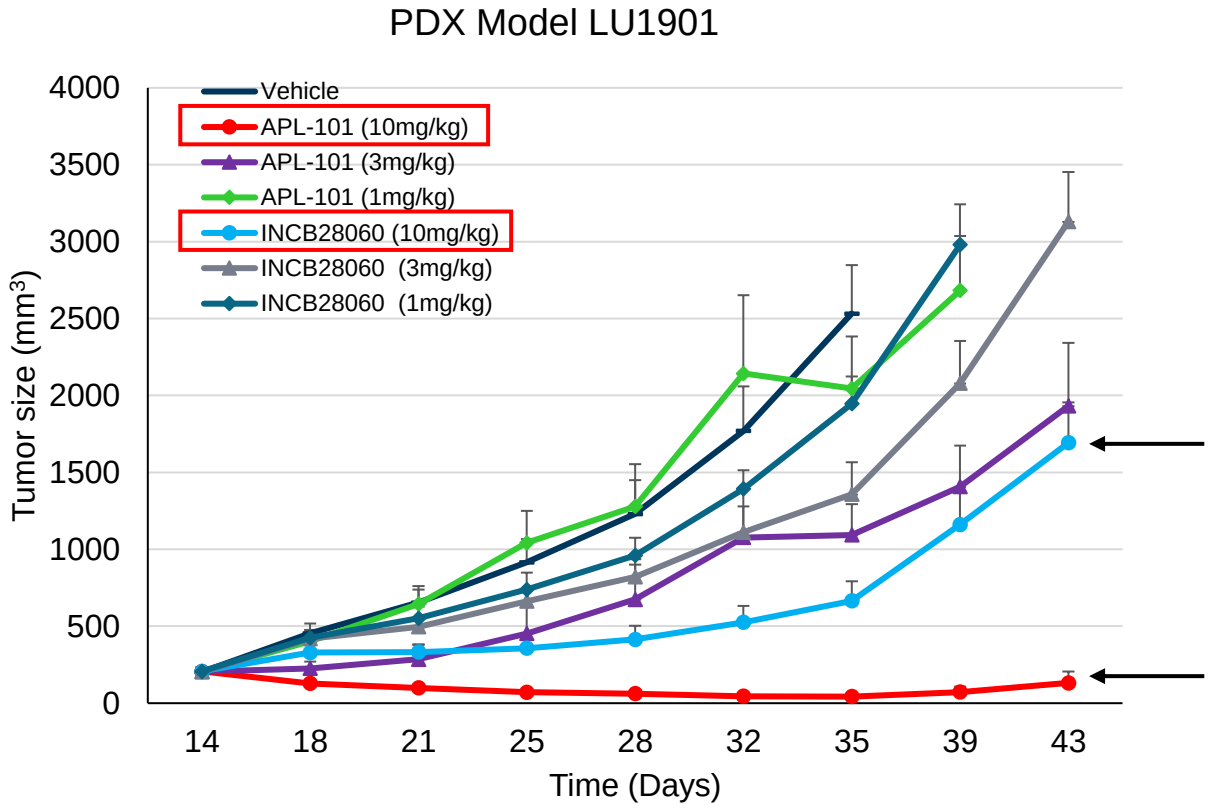
Vebreltinib – Preclinical differentiation

Compares favorably to capmatinib*

Favorable to Capmatinib in a Gastric Cancer MKN45 – Met amplified



Favorable to Capmatinib in a LUNG PDX Model LU1901 – Met amplified



Poster #2096 AACR 2017

* capmatinib= INC-B28060

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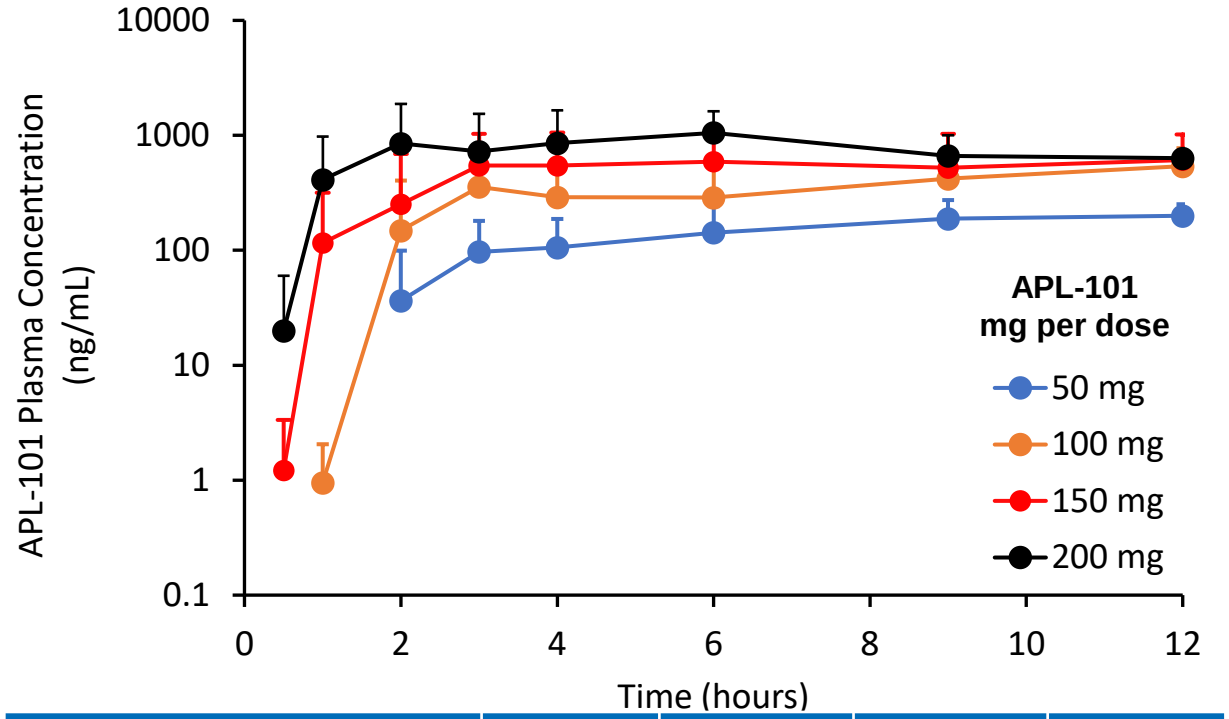
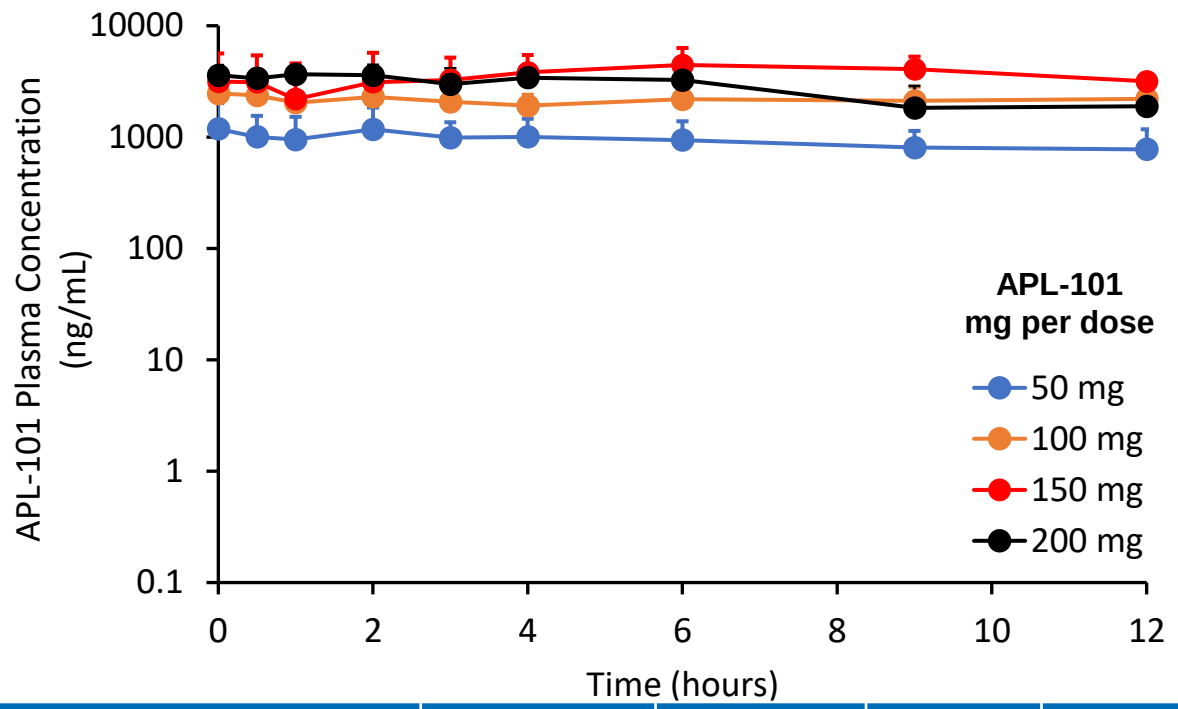
Plasma Pharmacokinetics

Cycle 1 Day 1; After single oral administration



Cycle 2 Day 1; After 28 days of twice daily oral administration



Dose level (QD)	50 mg (n=3)	100 mg (n=4)	150 mg (n=3)	200 mg (n=5)
C_{max} (ng/mL) Mean (SD)	235 (42.5)	581 (206)	833 (326)	1218 (721)
T_{max} (hr) Median (Min,Max)	12 (9, 48)	7.5 (3, 48)	6.0 (2, 36)	2.0 (0, 9)
$AUC_{(0-12)}$ (ng•hr/mL) Mean (SD)	1512 (820)	3824 (2323)	5661 (4837)	10611 (nc)
$T_{1/2}$ (hr) Mean (SD)	24.0 (11.5)	16.0 (5.0)	16.2 (3.6)	38.0 (5.0)

Dose level (BID)	50 mg (n=3)	100 mg (n=4)	150 mg (n=2)	200 mg (n=4)
C_{max} (ng/mL) Mean (SD)	1375 (739.5)	2950 (735.3)	4650 (nc)	5380 (1658)
T_{max} (hr) Median (Min,Max)	2.0 (0, 3)	4.0 (0, 12)	7.5 (6, 9)	1.0 (0, 2)
$AUC_{(0-12)}$ (ng•hr/mL) Mean (SD)	11115 (5208.4)	23910 (4864)	44670 (nc)	31095 (nc)*

*nc, not calculated (n = 1)

Kizilbash et al. EORTC NCI AACR Meeting 2020

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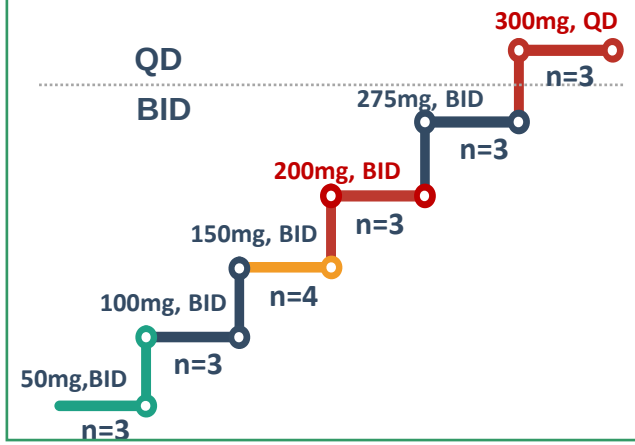
Study Design and patient Characteristics

Key Inclusion Criteria

- Metastatic or locally advanced NSCLC;
- c-Met overexpressed (IHC), *MET* amplified (FISH/NGS) or MET ΔEx14 (NGS) ;
- Never recieved c-Met inhibitor or HGF target therapy;
- ≥1 measurable lesions (RECIST);
- ECOG 0-2

Dose escalation n=19



Dose expansion n=18

- 300mg QD (n=3)
- 200mg BID (n=15)

RP2D 200mg BID

Endpoint

Primary Endpoint :

- Safty : Incidence and severity of adverse events, clinically significant abnormal laboratory results, ECG, and vital signs.

Secondary Endpoint :

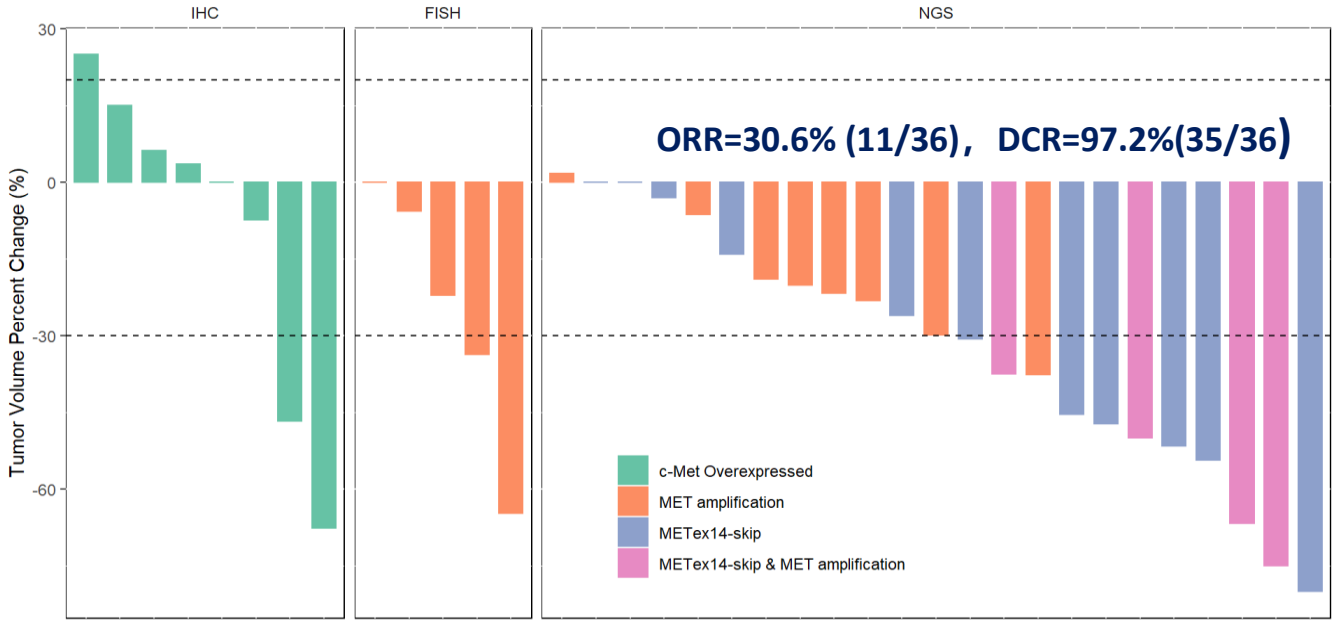
- PK parameters;
- Pharmacodynamics index evaluation;
- efficacy

Characteristic (n=37)	
Age, years	
Median (Min, Max)	62 (36-77)
Sex, n (%)	
Male	24 (64.8%)
Female	13 (35.2%)
Smoking history , n (%)	
Yes	24 (64.8%)
No	13 (35.2%)

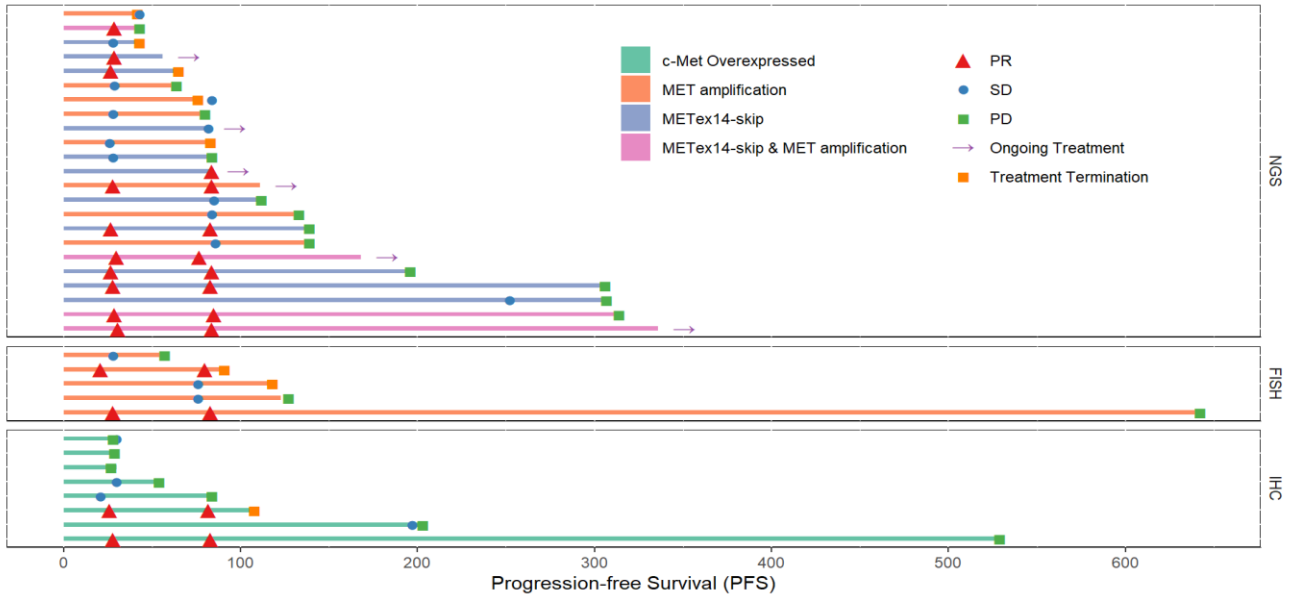
Characteristic (n=37)	
ECOG PS, n (%)	
1	1(2.8%)
2	34 (91.9%)
Numbers of prior therapies, n (%)	
0	17 (45.9%)
>=1	20 (50.1%)
Histology , n (%)	
Adenocarcinoma	34 (91.9%)
Other NSCLC	3 (8.1%)

Characteristic (n=37)	
Brian metastasis, n (%)	
Yes	7 (18.9%)
No	30 (81.1%)
c-Met alteration , n (%)	
c-Met overexpression	8 (21.6%)
MET amplification	13 (35.1%)
MET exon14 skipping	13 (35.1%)
MET exon14 skipping &amplification	3 (8.1%)
Stage, n (%)	
IIIB-IIIC	5 (13.5%)
IVA	14 (37.8%)

Duration of treatment and best tumor response



Treatment Duration



c-Met alteration (n=36)	PR	SD	ORR	DCR
c-Met overexpression (n=14)	5	8	35.7%	92.9%
MET amp (-) exon14 skipping (-) (n=8)	2	5	25%	87.5%
With MET amp (n=6)	3	3	50%	100%
With MET exon14 skipping (n=1)	1	0	100%	100%
***MET* amp (n=17)**	7	10	41.2%	100%
Accessed by FISH (n=5)	2	3	40%	100%
Accessed by NGS (n=12)	5	7	41.6%	100%
MET exon14 skipping (+) (n=8)	1	7	12.5%	100%
***MET* exon14 skipping (n=15)**	10	5	66.7%	100%
With MET amp (+) (n=3)	4	0	100%	100%

- **Preliminary clinical activity was observed with an ORR of 30.6% and DCR of 97.2%.**
- **Patients with exon 14 skipping determined by NGS had a significantly higher ORR (p=0.017) .**
- **11 patients treated with RP2D had an ORR of 72.7% and DCR of 100%.**

APL-101-01 SPARTA Phase 2 Study Design

Primary Endpoint: Overall Response Rate

Eligibility

- ≥ 18 years of age
- ECOG or KPS PS 0 – 1
- Measurable disease
- NSCLC & solid tumors with MET dysregulation^

Phase 2 RP2D (200mg BID) MET Dysregulation Inclusion Criteria

- MET amplification
  - Met/Cep-7 ratio of ≥ 2.2 or GCN of ≥ 6
  - MET/Cep-7 ratio of ≥ 5 or GCN ≥ 10 gene copies
- Mutation (EXON 14 skipping mutation)
- MET fusions per protocol

Cohort A1
EXON 14 Skipping NSCLC (MET inhibitor naïve)
1L (Stage 1=15, Stage 2=31)

Cohort A2
EXON 14 Skipping NSCLC (MET inhibitor naïve)
2L/3L (N=60)

Cohort B
EXON 14 Skipping NSCLC (MET inhibitor experienced)
(Stage 1=10, Stage 2=19)

Cohort C
Basket of tumor types except primary CNS tumors, MET amplification (MET inhibitor naïve)
(Stage 1=10, Stage 2=50)

Cohort C-1
NSCLC harboring MET amplification and wild-type EGFR (MET inhibitor naïve)
(Stage 1=10, Stage 2=36)

Cohort D
Basket of tumor types except primary CNS tumors, harboring MET gene fusions (MET inhibitor naïve)
(Stage 1=10, Stage 2=36)

Cohort E
Primary CNS tumors with MET alterations (MET inhibitor naïve)
(Stage 1=10, Stage 2=30)

Tx Term & 30-day FU & OS

NCT03175224

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Generating Clinical Evidence for Treating Three Indications

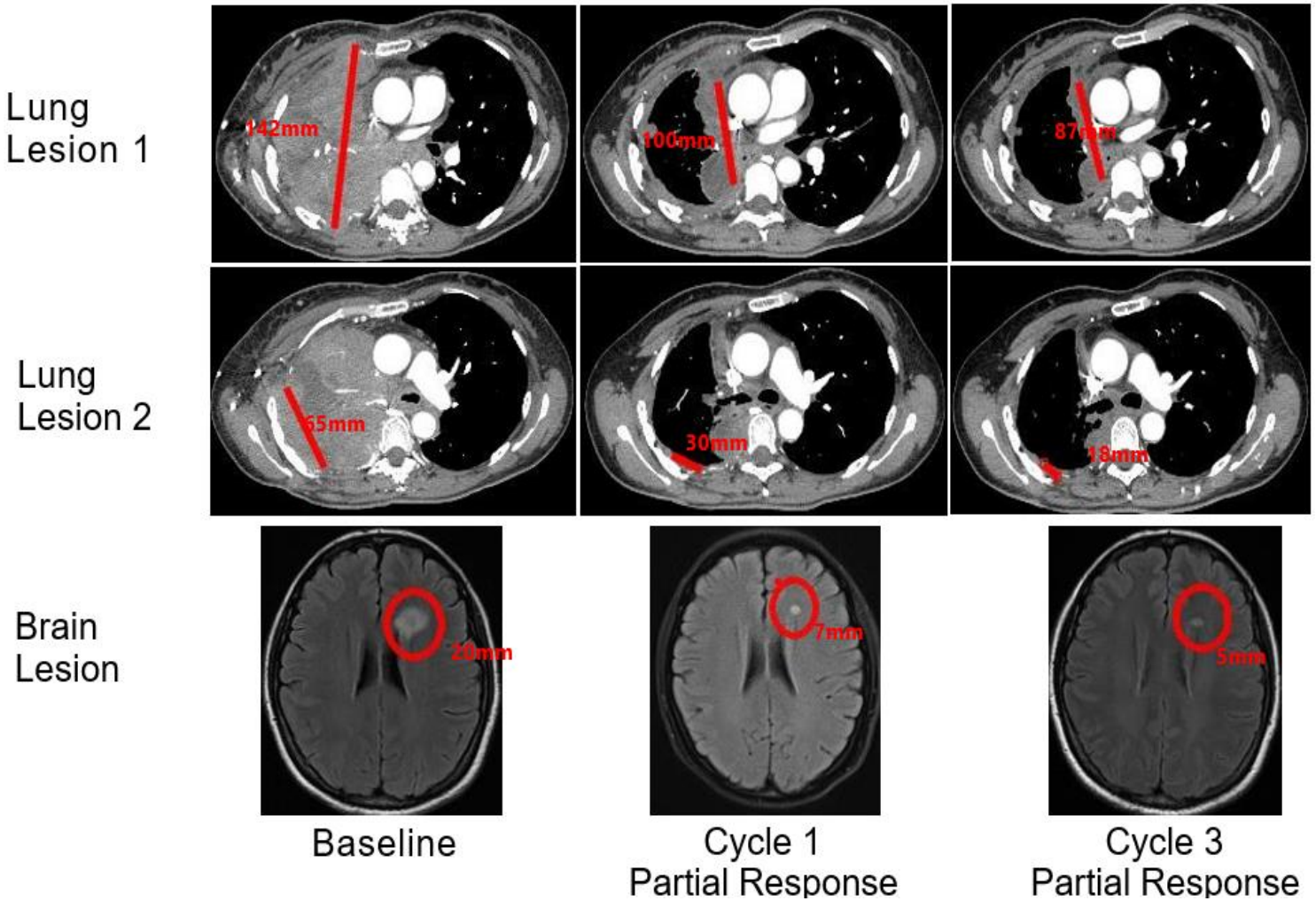
- **NSCLC with Met Ex14 skip phase 2 (potentially registrational)**:
  - **China submission** – efficacy based on China study: efficacy results appear more favorable than approved c-MET TKIs
    - NDA submission (for conditional approval) Sept'22,, under "priority review" by NMPA.
  - **US submission –** based on both China study and US/global study results; basis of FDA evaluation: "totality of data"
    - NDA submission- timing pending meeting results

- **NSCLC with cMet amplification**
  - China study enrollment ongoing
  - SPARTA cohort and China data to support a US submission

- **GBM**
  - Phase 2/3 Study in China – enrollment near completion;
  - SPARTA cohort and China Study may support a US submission

Activity in a Patient with Primary NSCLC Lesions and Brain Metastasis

NSCLC with c-Met amplification



Yilong Wu et al CSCO 2019

NSCLC – Non-Small Cell Lung Cancer

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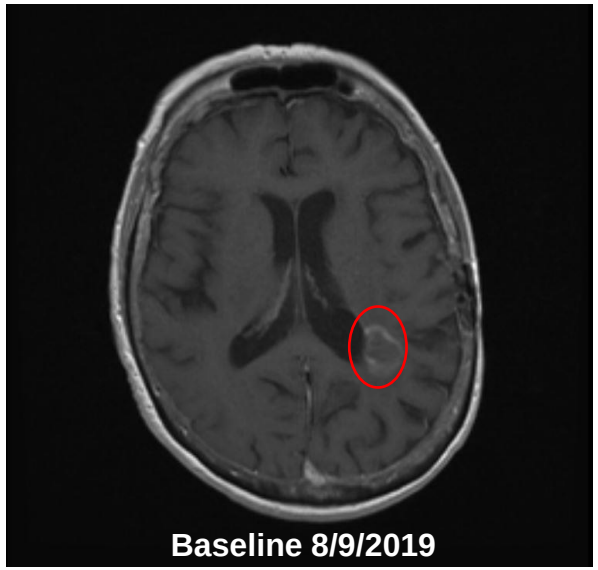
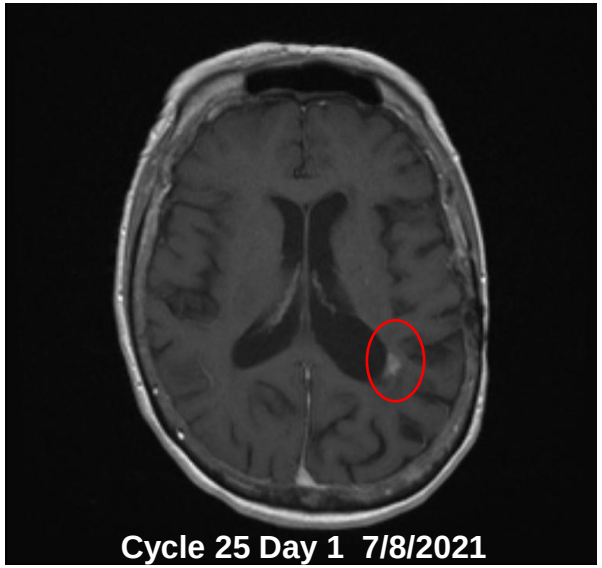
Activity in a Glioblastoma Patient with c-MET Amplification

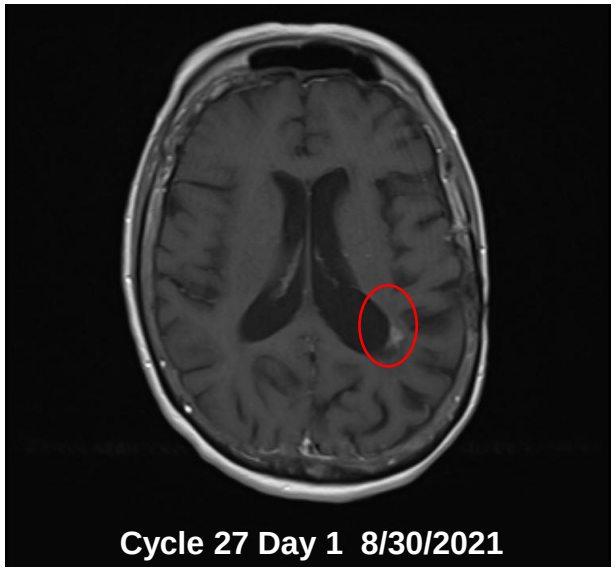
On treatment for 2+Years

- 78-yr old female, GBM since May 2015, c-Met Amplification, target lesion Lt Subependymal
- Received 3 prior lines of therapies (Temodar 2015-2017, Avastin 2017-2018, Nivolumab 2018-2019)
- C1D1: 04Sep2019; 2+ yr treatment, durable response

Visit	Product of Perpendicular Diameters
Screening	**285**
Cycle 3 Day 1	285
Cycle 5 Day 1	300
Cycle 7 Day 1	252
Cycle 9 Day 1	119
Cycle 11 Day 1	96
Cycle 13 Day 1	98
Cycle 15 Day 1	96
Cycle 17 Day 1	75
Cycle 19 Day 1	56
Cycle 21 Day 1	96
Cycle 23 Day 1	60
Cycle 25 Day 1	60
Cycle 27 Day 1	**25**







Longest Axis	**19**	**12**	**05**
Perpendicular Measurement	**15**	**05**	**05**
Product of Perpendicular Diameters	**285**	**60**	**25**

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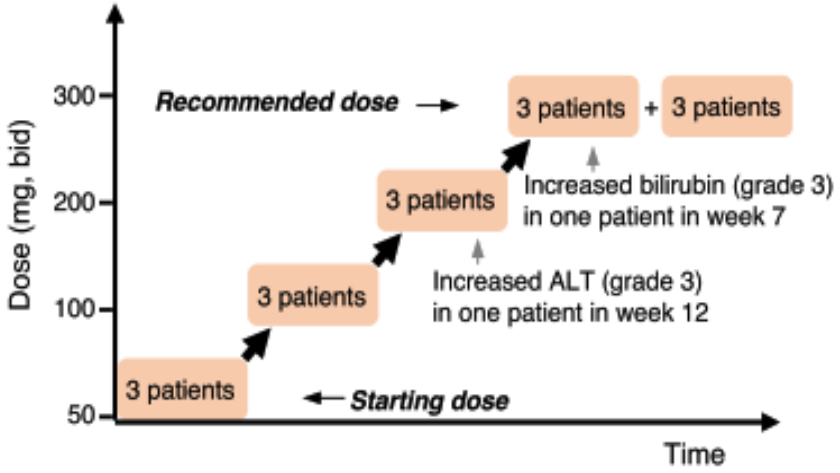
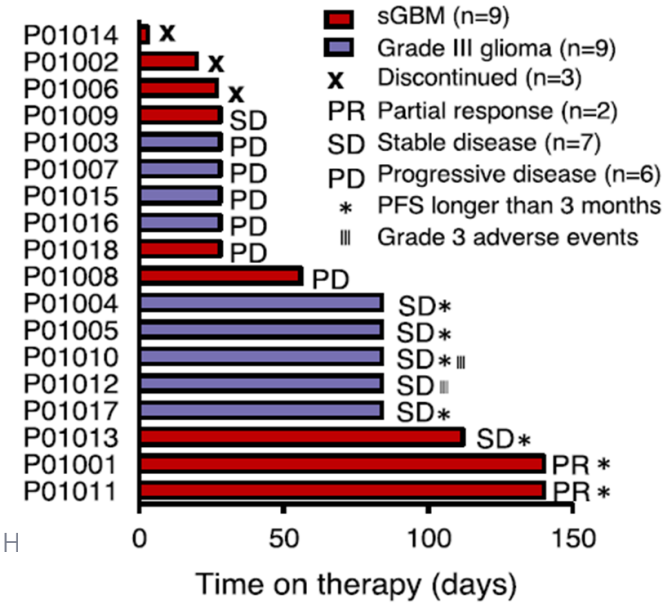
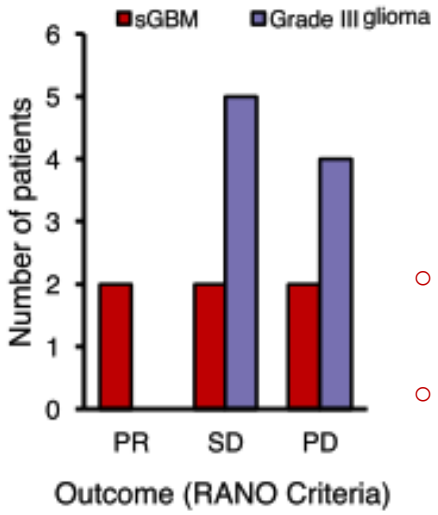
Glioblastoma Phase I/II (Pearl)

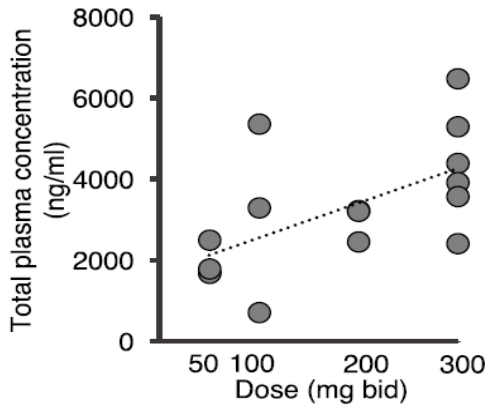
Study of a c-Met Inhibitor PLB1001 in Patients With PTPRZ1-MET Fusion Gene Positive Recurrent High-grade Gliomas. NCT02978261



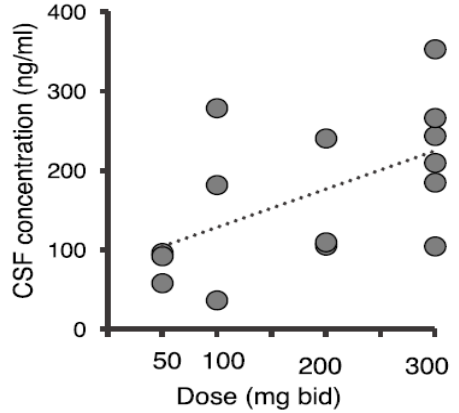




Plasma



Cerebrospinal fluid (CSF)



APL-101 concentration in the cerebrospinal fluid (CSF) collected on day 15 was ~3%–8% of that in plasma and showed an increasing trend with the drug dosage

- Among six sGBM patients treated with APL-101, two achieved PR, two achieved SD, and two had PD
- Among the nine grade III glioma patients, five achieved SD and four had PD

Hu et. al. Cell 2018

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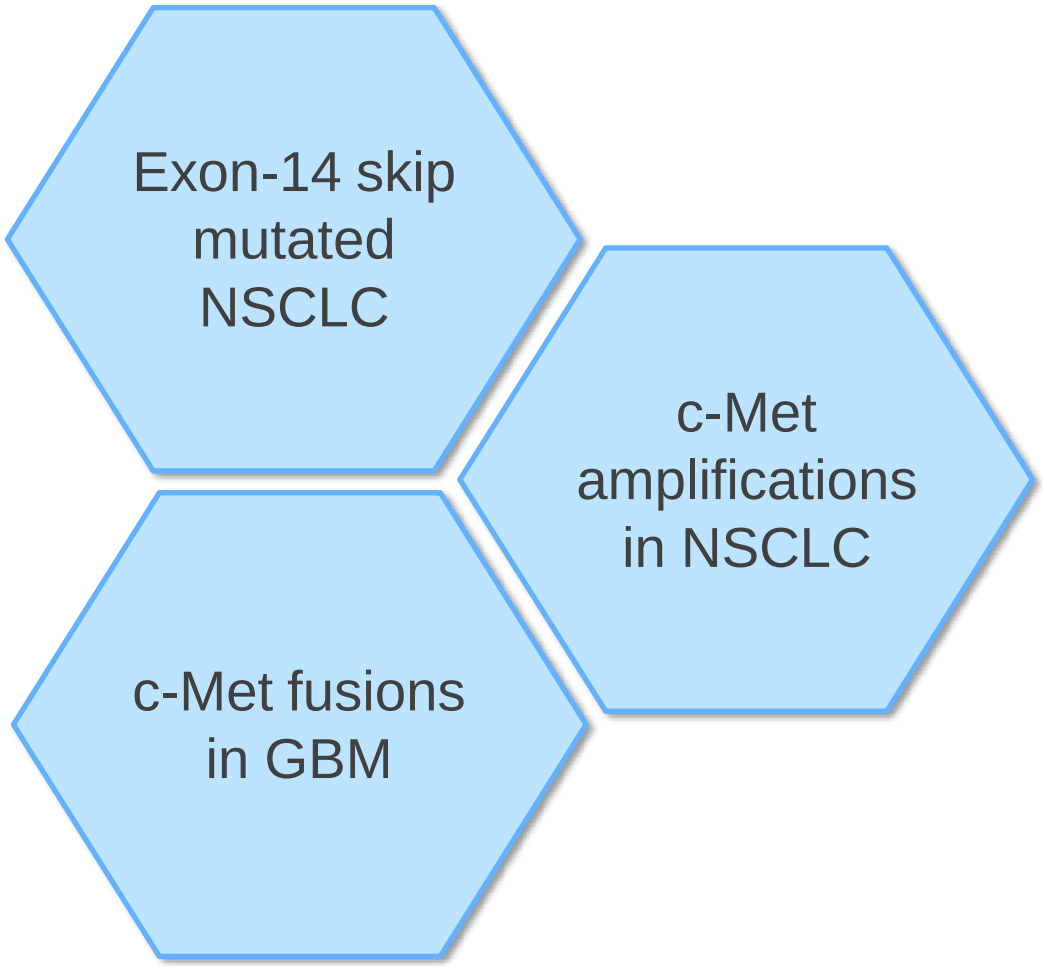
Vebreltinib: 3 Indications for near-term NDA/sNDAs



Vebreltinib

Global Multicohort Phase 2 – Non-Small Cell Lung cancer, Glioblastoma ("GBM"), various solid tumors with c-Met dysregulation

- ✓ **Highly specific c-Met inhibitor**
- ✓ **Brain penetration**
- ✓ **Safety data available from over 370 patients worldwide**

- ✓ **Biomarkers to target c-Met patients**
- ✓ **Strong IP**

- ✓ **Orphan drug designation by FDA**
- ✓ **~ 140 patients treated in Apollomics SPARTA trial ongoing in 13 countries and 90+ sites**
- ✓ **Registrational Phase 2 study in NSCLC with exon 14 skip or c-Met amplification (China)**
- ✓ **Phase 2/3 GBM with PTPRZ1-MET fusion (China)**

- ✓ **Potential combo therapy w/EGFR inhibitors, etc., with huge potential**
- ✓ **Potential other tumors: Gastrointestinal, renal, thyroid, etc.**

NSCLC – Non-Small Cell Lung Cancer
GBM – Glioblastoma Multiforme

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Vebreltinib - Additional Indications

- EGFR resistance & c-Met amplification – potential role for c-Met TKI
- Potential Vebreltinib Indications beyond Lung & Brain Tumors
  - Gastrointestinal cancers: colon, stomach, pancreatic, liver, cholangiocarcinoma
  - Renal cell cancer
  - Thyroid cancer
  - Prostate cancer
  - Breast cancer
  - Ovarian, and other female reproductive tract

MET amplification - a driver of resistance in~**15%** of the TKI-treated population across various oncogene-driven **NSCLCs**
Opportunity for Combination Therapy W/ APL-101 TO OVERCOME RESISTANCE

Table 1. Summary of key clinical studies identifying *MET* amplification as a mechanism of resistance in oncogene-driven NSCLC

Molecular subset of NSCLC	Number of lung cancer samples + Type	Prior targeted therapy	Incidence of MET amplification	Method of MET amplification testing	Reference
EGFR	Following second-line osimertinib: range 10%-22%				
	83 Plasma		19% (14/83)	NGS	52
	32 Tumor tissue		22% (7/32)	FISH	8
	42 Tumor tissue		14% (6/42)	FISH and/or NGS	54
	41 Tumor tissue		10% (4/41)	NGS and FISH	53
EGFR	Following first-line osimertinib: range 7%-15%				
	91 Plasma		15% (14/91)	NGS	55
	27 Tumor tissue		7.4% (2/27)	NGS	51
ALK	Post-treatment tissue ($n = 101$) or Plasma ($n = 106$)	Crizotinib, or next-generation ALK inhibitors (e.g. lorlatinib)	11 (13%)	FISH and/or NGS	77
RET	23	Selpercatinib or pralsetinib	15%	FISH or NGS	82
ROS1	17	Lorlatinib	6%	NGS and FISH	85
KRAS	10 Tumor tissue and/or plasma	Adagrasib	20%	NGS	95

NGS, next-generation sequencing; NSCLC, non-small-cell lung cancer.

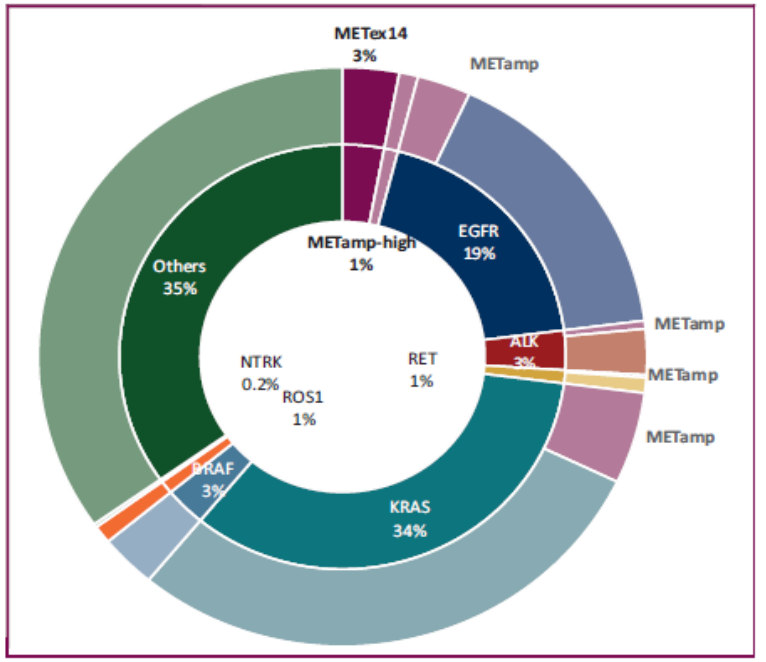

Figure 2. Frequency of MET dependency in lung cancer.
The inner ring represents known primary oncogenic driver alterations in metastatic lung cancers, such as *EGFR, ALK, RET, KRAS,* and *BRAF*. The outer ring illustrates known resistance mechanisms in these oncogenic-driven NSCLC subsets: frequency of *MET* amplification (red) is ~15% in *EGFR, KRAS,* and *ALK*- and *RET*-fusion-positive NSCLC. Taken together, these data highlight that ~7%-10% of NSCLC tumors are *MET* dependent, including *de novo* METex14 and high MET amplification. EGFR, epidermal growth factor receptor; NSCLC, non-small-cell lung cancer.

Ref: Coleman, etc; Beyond epidermal growth factor receptor: MET amplification as a general resistance driver to targeted therapy in oncogene-driven non-small-cell lung cancer; *J ESMO Open*, Nov 2021; 6(6)

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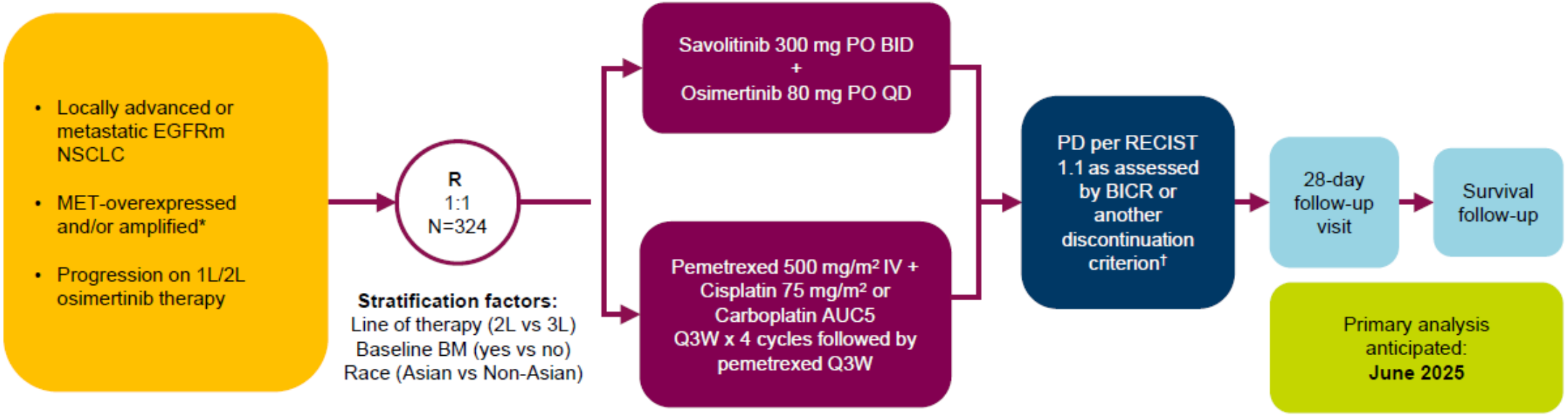
Figure 1. SAFFRON study design

- SAFFRON is a global, multicentre, randomised, open-label Phase III study aiming to determine the efficacy and safety of savolitinib in combination with osimertinib vs platinum doublet chemotherapy in patients with EGFRm and MET-overexpressed and/or amplified, locally advanced or metastatic NSCLC who have progressed on 1L or 2L osimertinib treatment
- Patients treated with osimertinib in the adjuvant setting can be included if disease progression occurred <6 months after the last dose
- Patients may continue to receive savolitinib plus osimertinib or osimertinib monotherapy beyond progression if they are deriving clinical benefit, as judged by the investigator
- Tumour assessments using computed tomography or magnetic resonance imaging will be collected every 6 weeks ±7 days, up to 54 weeks, and then every 9 weeks ±7 days until disease progression



*MET overexpression is defined as ≥90% of tumour cells staining at strong 3+ intensity as detected by immunohistochemistry; MET amplification is defined as ≥10 copies of MET gene in tumour cells as detected by fluorescence in situ hybridisation. †Other discontinuation criteria include unacceptable toxicity or patient withdrawal; cross-over from chemotherapy to the combination treatment is not permitted. 1/2/3L, first/second/third-line; AUC5, area under the plasma drug concentration-time curve of 5mg/mL/min; BICR, blinded independent central review; BID, twice daily; BM, brain metastases; EGFRm, epidermal growth factor receptor-tyrosine kinase inhibitor sensitising mutation; IV, intravenous; NSCLC, non-small cell lung cancer; PD, progressive disease; PO, oral; Q3W, every 3 weeks; QD, once daily; RECIST, Response Evaluation Criteria in Solid Tumors

Lu et al. WCLC 2022

Summary

- Inhibiting the HGF/c-Met pathway remains a promising target in multiple cancers.
- MET dysregulations include a heterogeneous group of diseases that include mutations, gene amplifications as well as fusions in NSCLC as well as multiple other cancers
- Inhibiting the pathway with TKIs has shown a meaningful benefit in MET Exon 14 skipping NSCLC
- MET gene amplified cancers as well as MET fusions still remains a pathway under investigation
- Acquired resistance due to MET amplification post TKI dosing is one of the drivers in several cancers, primarily in EGFR mutated NSCLC
- Elevated levels of HGF expression is also a mechanism of resistance in other cancers such as breast cancer and melanoma.
- New modalities – such as MET-EGFR bispecific antibodies and MET-ADCs will expand the landscape of inhibiting the HGF/c-Met pathway

Thank you