Turning Point Therapeutics

Precision Oncology Medicines for Treatment Resistant Cancers

Company Overview

June 2019

Disclaimer

Forward-Looking Statements

Statements in this Presentation that are not statements of historical fact are forward-looking statements. Such forward-looking statements include, without limitation, statements regarding our research and clinical development plans, business strategy and plans, regulatory matters, objectives of management for future operations, industry trends, market size and opportunity and our ability to complete certain milestones. Words such as “believe,” “can”, “continue,” “anticipate,” “could,” “estimate,” “plan,” “predict,” “expect,” “intend,” “will,” “may,” “goal,” “upcoming,” “near term”, “milestone”, “potential,” “target” or the negative of these terms or similar expressions are intended to identify forward-looking statements, though not all forward-looking statements necessarily contain these identifying words. These forward-looking statements are based on the current beliefs of the Company's management as well as assumptions made by and information currently available to the Company. Such statements reflect the current expectations and projections of the Company with respect to future events and trends and are subject to known and unknown risks, including business, regulatory, economic and competitive risks, uncertainties, contingencies and assumptions about the Company, including, without limitation: our plans to research, develop and commercialize our drug candidates, including the timing of our planned Phase 2 portion of TRIDENT-1; the success, cost and timing of our product development activities and clinical trials, including whether the planned Phase 2 portion of TRIDENT-1 will support the approval of repotrectinib in *ROS1*+ advanced NSCLC and *NTRK*+ advanced solid tumors; our ability to obtain and maintain regulatory approval for repotrectinib or any of our other current or future drug candidates, and any related restrictions, limitations, and/or warnings in the label of an approved drug candidate; our expectations regarding the size of target patient populations for our drug candidates, if approved for commercial use, and any additional drug candidates we may develop; our ability to obtain funding for our operations; the commercialization of our drug candidates, if approved; our ability to attract collaborators with development, regulatory and commercialization expertise; our expectations regarding our ability to obtain, maintain, enforce and defend our intellectual property protection for our drug candidates; future agreements with third parties in connection with the commercialization of repotrectinib, or any of our other current or future drug candidates; the size and growth potential of the markets for our drug candidates, and our ability to serve those markets; the rate and degree of market acceptance of our drug candidates, as well as the reimbursement coverage for our drug candidates; regulatory and legal developments in the United States and foreign countries; the performance of our third-party suppliers and manufacturers; the success of competing therapies that are or may become available; our ability to attract and retain key scientific or management personnel; the accuracy of our estimates regarding expenses, capital requirements and needs for additional financing; and our use of the proceeds from the offering to which this Presentation relates. In light of these risks and uncertainties, the events or circumstances referred to in the forward-looking statements may not occur. The actual results may vary from the anticipated results and the variations may be material. These forward-looking statements should not be taken as forecasts or promises nor should they be taken as implying any indication, assurance or guarantee that the assumptions on which such forward-looking statements have been made are correct or exhaustive or, in the case of the assumptions, fully stated in the Presentation. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date this Presentation is given. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this Presentation.

This Presentation discusses product candidates that are under clinical study and which have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the use for which such product candidates are being studied.

Company Highlights:
Designing Small and Compact TKIs to Overcome Limitations of Conventional TKIs

Potential Best-in-Class ROS1 and TRK Inhibitor

- **Lead Program Repotrectinib has demonstrated proof of concept in ongoing TRIDENT-1 Phase 1/2 Study**
- *ROS1*+ advanced NSCLC in 33 subjects across 7 dose escalation cohorts[1]:
  - TKI-naïve 82% confirmed ORR (cORR) (83% cORR in patients treated at 160 mg QD or above)
  - TKI-pretreated 32% cORR (55% cORR in patients treated at 160 mg QD or above with 1 prior TKI)
  - CNS activity in both populations and manageable safety profile
- **Plan to enter TRIDENT-1 Phase 2 registrational portion in 2H 2019 in *ROS1*+ advanced NSCLC and *NTRK*+ advanced solid tumors**

Novel Structure Based Design Approach

- **Proprietary TKIs designed with small, compact, three-dimensional macrocyclic structure**
- Provides for favorable kinase selectivity and can overcome intrinsic and acquired resistance with current TKIs

Rapidly Advancing Pipeline

- **Multiple preclinical candidates:**
  - **TPX-0022 (MET/CSF1R/SRC):** Targets *MET*-driven tumor cells and modulates the TME through CSF1R inhibition
  - **TPX-0046 (RET/SRC):** Inhibition of RET and SRC kinases is believed to increase the therapeutic effect of RET inhibitors
  - **ALK inhibitor:** Inhibition against wildtype and a variety of mutated ALKs

Strong Cash Position

- **Well-funded**, cash position expected to be sufficient to fund current operations into the second half of 2021
  - **est. $90 million in cash as of March 31 and $175.2 million net IPO proceeds**

[1] *Data cut-off date of March 4, 2019*

Proven Management Team with Significant Precision Oncology Drug Development Experience

Athena M. Countouriotis, M.D.
President and Chief Executive Officer

15 years Oncology Drug Development

SPRYCEL dasatinib 100 mg tablets
SUTENT capsules sunitinib malate For my fight
Bosulif bosutinib tablets 500 mg | 400 mg | 100 mg
MYLOTARG gemtuzumab ozogamicin INJECTION FOR I.V. INFUSION 4.5 mg single-dose vial
Vanflyta Quizartinib

J. Jean Cui, Ph.D.
Scientific Founder, Chief Scientific Officer

20 years Oncology Drug Discovery

XALKORI CRIZOTINIB
SUTENT capsules sunitinib malate For my fight
LORBRENA LORLATINIB 100 mg tablets

Robert Xin, M.D., Ph.D.
Senior Vice President, Clinical Development

20 years Drug Discovery & Development

Zelboraf vemurafenib
Erivedge vismodegib
GAZYVA obinutuzumab injection
Rituxan Rituximab
SUTENT capsules sunitinib malate For my fight

Annette North, Esq.
Executive Vice President, General Counsel

25 years in Biotech

KURA ONCOLOGY
ambit
SGX Pharmaceuticals

Brian Baker, CPA
Vice President, Finance

22 years Accounting & Finance

CLEAVE BIOSCIENCES
pwc
PHENOMIX

Board of Directors with Deep Targeted Oncology Experience

Sheila Gujrathi, M.D.
Board Chair
President and CEO,
Gossamer Bio

Jacob Chacko, M.D., M.B.A.
CEO, ORIC Pharmaceuticals

Patrick Machado, J.D.
Co-Founder & Former CFO, Medivation

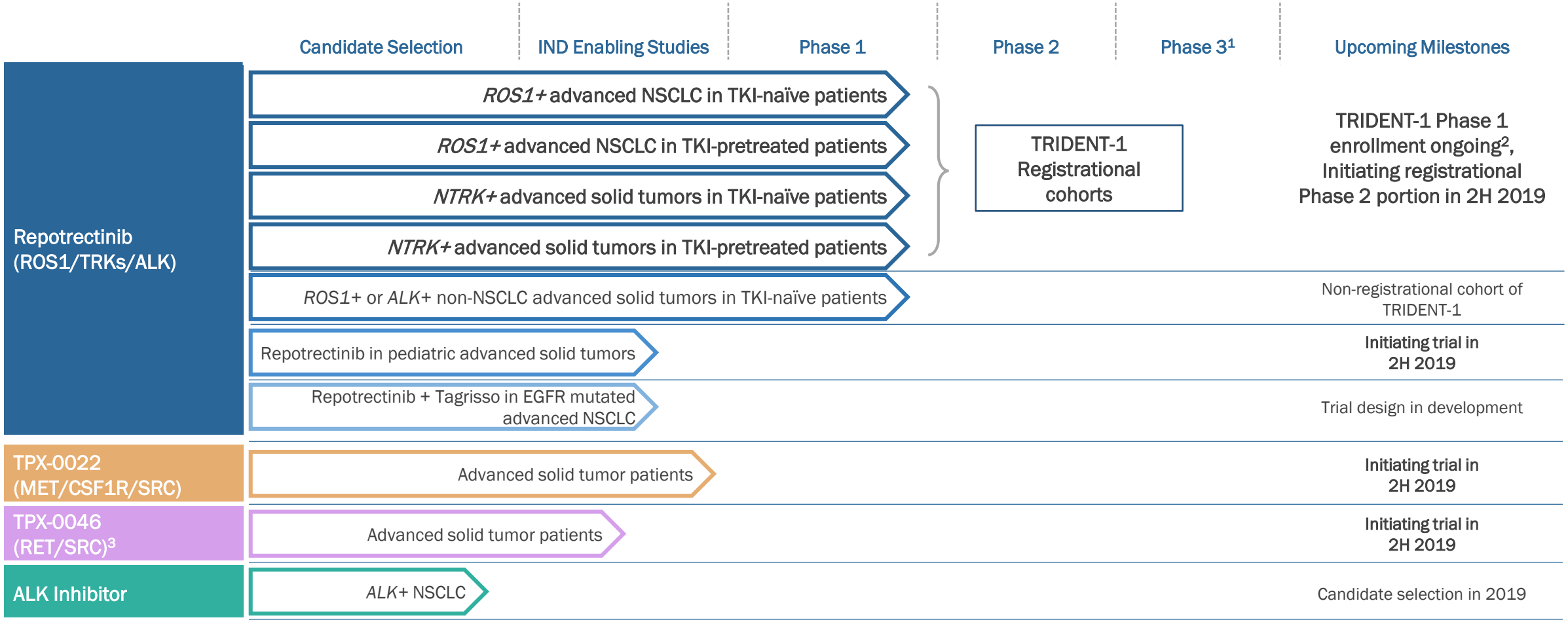
Athena Countouriotis, M.D.
President and Chief Executive Officer

Carl Gordon, Ph.D., CFA
Partner, OrbiMed Advisors, LLC

J. Jean Cui, Ph.D.
Scientific Founder and Chief Scientific Officer

Simeon George, M.D., M.B.A.
Partner, S.R. One, Limited

Extensive Pipeline with Lead Candidate Repotrectinib Approaching Registrational Phase 2 TRIDENT-1 Trial



[1] *Not required for Phase 2 registrational clinical trials*
[2] *Phase 1 Portion of TRIDENT-1 ongoing with anticipated data read outs within 2019*
[3] *Including NSCLC, thyroid, and other solid tumors with abnormal RET gene*

Overall Patient Population: Biomarker Prevalence

	Repotrectinib			TPX-0022			TPX-0046	
	Advanced NSCLC	Other Advanced Solid Tumors[1]		Gastric	Advanced NSCLC	EGFR Mutated TKI-Resistant Advanced NSCLC[3]	Advanced NSCLC	Thyroid[2]
U.S. Patients[4,5]	160,000	520,000		17,500	160,000	12,800	160,000	11,250
EU5 Patients[4]	117,000	557,900		36,680	117,000	6,230	117,000	11,030
Biomarker Prevalence[6]	2% (ROS1)	0.5% (NTRK)	0.5% (ROS1 / ALK)	4% (MET)	3% (MET Exon 14)	12.5% (MET Amplified)	2% (RET)	16% (RET)

[1] *Reflects other solid tumor indications including Brain, Breast, Colon, Melanoma, NSCLC, Pancreas, Sarcoma, and Thyroid, excluding ROS1+ and ALK+ for NSCLC*
[2] *Includes papillary and medullary thyroid tumors*
[3] *Does not include first line EGFR mutated advanced NSCLC patients. Assumes ~20%, 15%, 11%, 14%, 17% and 12% EGFR mutation prevalence for US, France, Germany, Italy, Spain and UK, respectively*
[4] *Estimates include Stage III unresectable and metastatic patient populations, adjusted for treatable population and those that are tested for the targeted biomarkers; assumes 85% biomarker testing rate*
[5] *Based on SEER 2015 5-year diagnosed prevalence, grown at 0.7% in line with U.S. population growth; estimated as of 2018*
[6] *Estimates based on publications and physician and payor interviews in the U.S.*

Turning Point Therapeutics

Novel Approach for Creating New Kinase Inhibitors and Overcoming Kinase Drug Resistance

Current Available TKIs Often Lead to Treatment Resistance

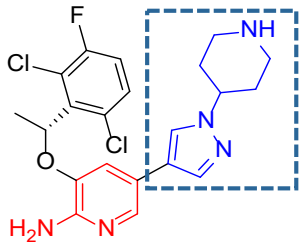
No Approved TKIs to address resistance that may arise after prior ROS1, TRK, or RET targeted agents

- *ROS1*+ NSCLC
  - Solvent Front Mutation (SFM: G2032R) reported in up to 41% of Xalkori treated patients[1]
- *NTRK*+ Metastatic Solid Tumors
  - Emerging SFMs reported after treatment with Vitrakvi and Entrectinib[2]
- RET NSCLC and Thyroid Cancer
  - While BLU-667 and LOXO-292 are effective against gatekeeper mutations, other potential resistant mutations may arise

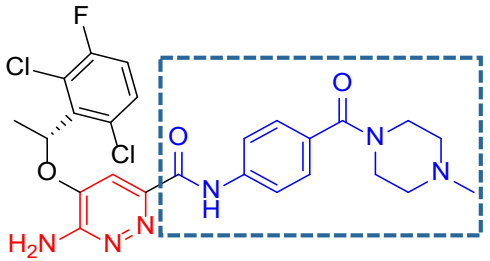
- Our small, compact TKIs have rigid three dimensional macrocyclic structures that bind to mutated kinases that sterically exclude conventional TKIs
- Our macrocycle platform has the ability to develop potential best-in-class therapies that may address limitations of today's TKIs and prevent common resistant mutations
- Activity of Repotrectinib against Solvent Front Mutations highlighted in Cancer Discovery[3]

[1] *Gainor JF et al., JCO Precis Oncol, 2017*
[2] *Drilon, et al, AACR, Jun 3, 2017; Drilon, et al, NEJM Feb 2018; Cocco et al, Nature Reviews, Dec 2018*
[3] *Drilon, et al, Cancer Discovery, Aug. 9, 2018*

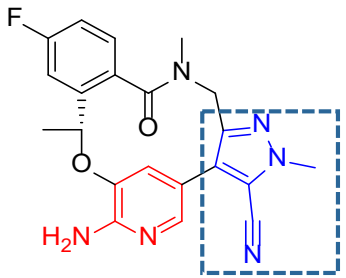
Common Structure Motifs May Lead to Solvent Front Mutations



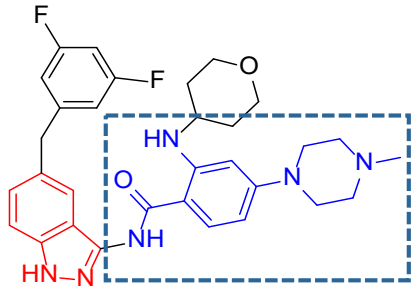
Crizotinib
MW 450.34



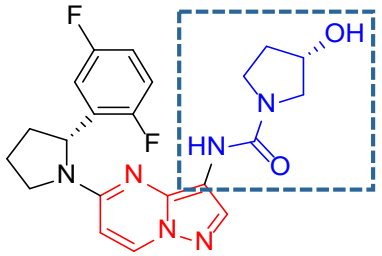
Ensartinib
MW 547.41



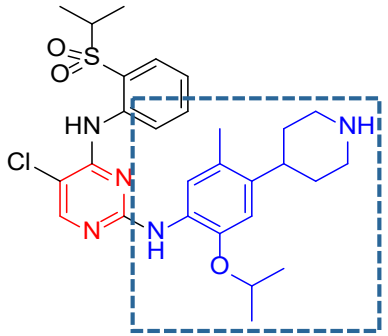
Lorlatinib
MW 406.42



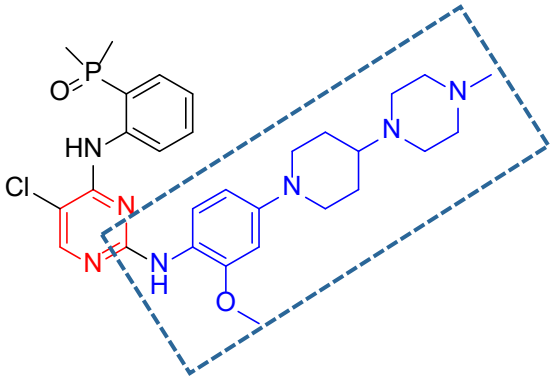
Entrectinib
MW 560.65



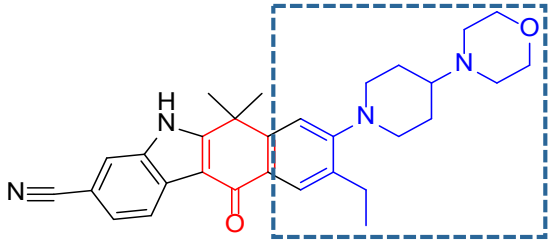
Larotrectinib
MW 428.44



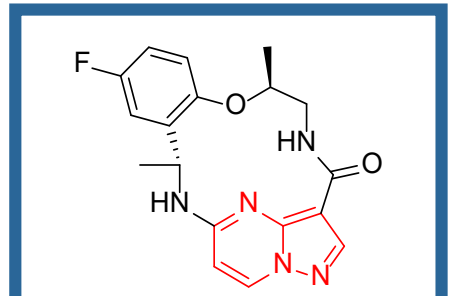
Ceritinib
MW 558.14



Brigatinib
MW 584.10



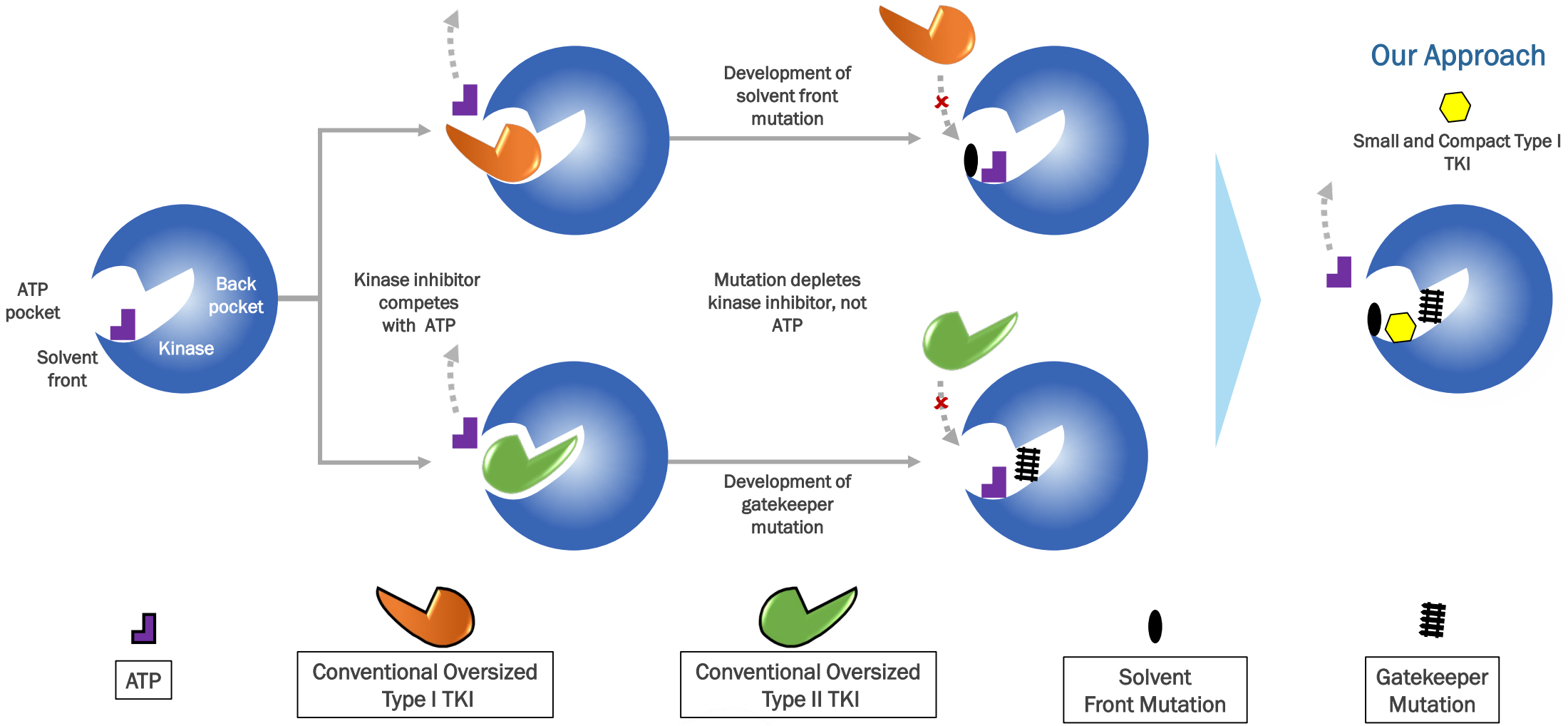
Alectinib
MW 482.64



Repotrectinib
MW 355.37

MW: molecular weight

Our TKIs Bind Completely Inside the ATP Pocket, Thereby Potentially Addressing Treatment Resistance



Turning Point Therapeutics

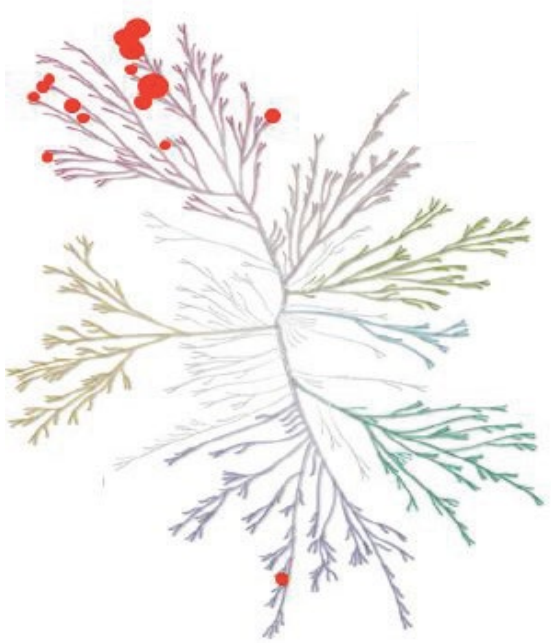
Repotrectinib (TPX-0005): A Highly Selective, Internally Designed Inhibitor Targeting Wildtype and Mutant ROS1/TRKs/ALK

Repotrectinib Inhibits ROS1, TRK and ALK Kinases

Mechanism of Action

In Preclinical Models:

- Potent inhibitor of **ROS1, TRK and ALK kinases**
- **Designed to circumvent the steric interference** from clinically resistant mutations, especially **solvent front and gatekeeper mutations**
- **Inhibits JAK2, SRC and FAK** in addition to the inhibition of ROS1, TRK and ALK kinases
  - **Leads to modulation of STAT3** (Signal Transducer and Activator of Transcription 3) signaling, one of the **major signaling pathways for both intrinsic and acquired resistance**
  - Potential to lead to **longer duration of response**



Kinase	Selectivity Index
ROS1	1 (IC_{50} 0.071 nM)
TRKA, TRKB, TRKC	1< SI <10
ALK, JAK2, FYN[1]	10< SI <20
LYN[1], YES1[1], FGR[1], SRC[1], TXK, ARK5, DDR1, FAK	20< SI <250

[1] *Denotes SRC family member. Kinome illustration reproduced courtesy of Cell Signaling Technology, Inc. (CSTI) (www.cellsignal.com). Each branch of the dendogram represents an individual human kinase. The foregoing website is maintained by CSTI*

Repotrectinib: Potential Best-in-Class ROS1 Inhibitor
Data Presented at Annual AACR Conference on April 1

- Crizotinib is approved for advanced *ROS1*+ NSCLC
  - Limitations include lack of CNS activity, acquired resistant mutations and safety profile
- Repotrectinib demonstrated high potency against fusion ROS1 and emerging resistant mutations
  - Designed to overcome TKI resistance mutations, especially solvent front ROS1 G2032R[1]

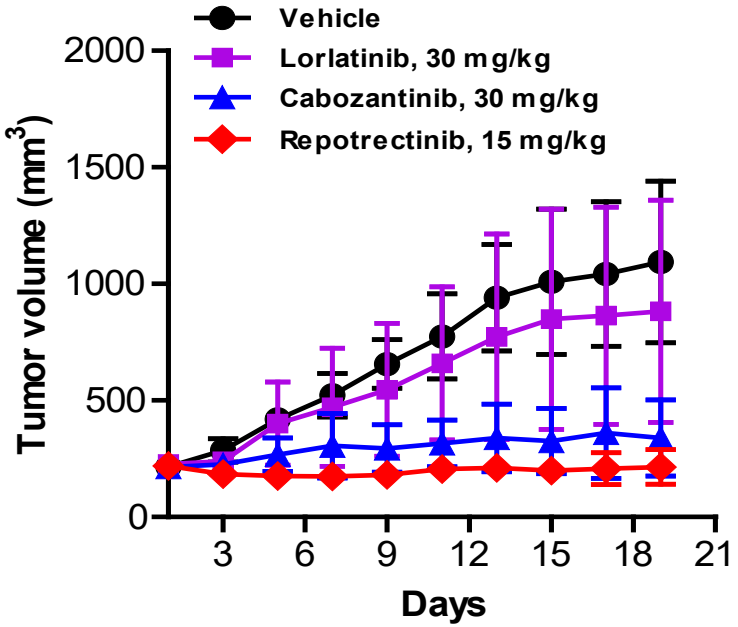
	Ba/F3 Cell Proliferation Assay IC_{50} (nM)									
	No Kinase Domain Mutation				ROS1 G2032R				ROS1 L2026M	
Inhibitor*	CD74-ROS1	SDC4-ROS1	EZR-ROS1	TPM3-ROS1	CD74-ROS1	SDC4-ROS1	EZR-ROS1	TPM3-ROS1	EZR-ROS1	TPM3-ROS1
Repotrectinib	**<0.2**	**0.2**	**<0.1**	**<0.1**	**3.3**	**3**	**5**	**16.3**	**<0.2**	**<0.1**
Crizotinib	14.6	19.6	19.4	31.1	266.2	4661	660	500.6	95.6	236.2
Lorlatinib	0.2	0.3	0.2	0.3	160.7	352.9	190.5	434.9	1.6	1.9
Entrectinib	10.5	ND	1.5	9.4	1813	ND	2947	1093	13.3	40.7
Cabozantinib	0.5	3	0.4	4.5	11.3	169.4	39.5	60.7	3.4	12.6

[1] *Drilon A et al., Cancer Discov 2018*

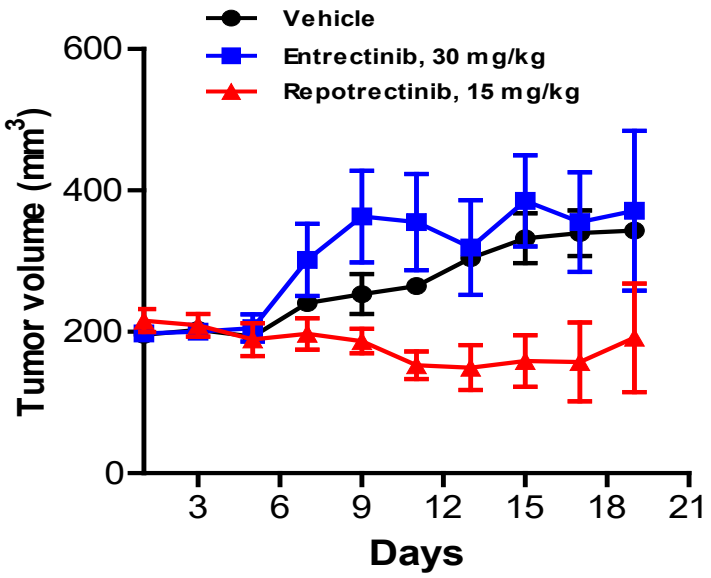
** Other than repotrectinib, data based on evaluation of comparable proxy chemical reagent purchased from commercial sources rather than obtained from the pharmaceutical company developing the kinase inhibitor*

Repotrectinib is Potent Against *ROS1* G2032R Mutation and Active in Intracranial Tumor Model

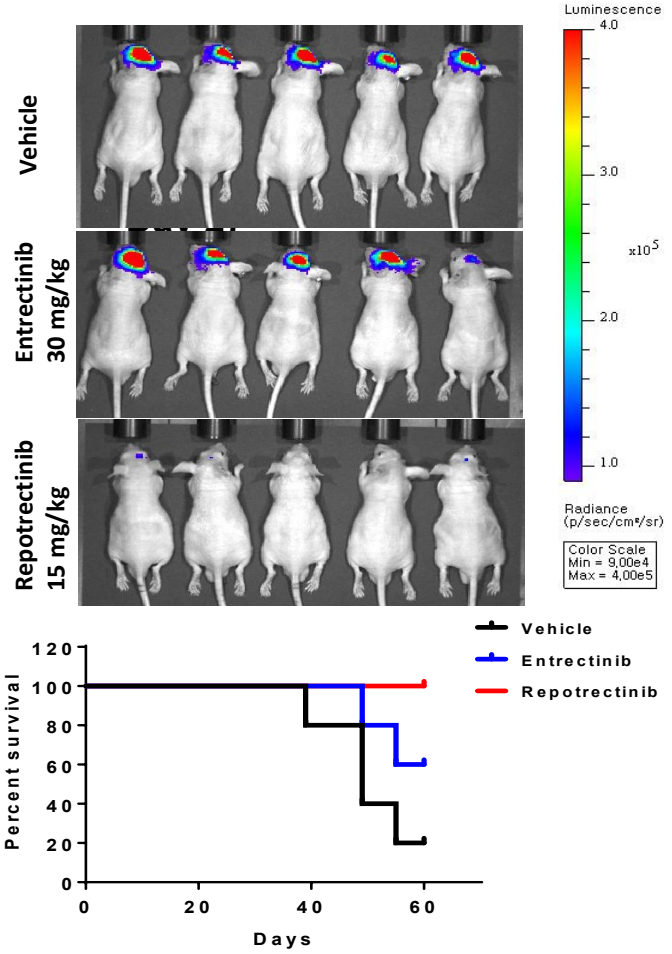
A. Antitumor activity in a NSCLC patient cell-derived mouse xenograft tumor model with CD74-*ROS1* G2032R mutation acquired after crizotinib treatment for 10 months



B. Antitumor activity in a NSCLC patient-derived xenograft mouse tumor model with CD74-*ROS1* G2032R mutation acquired after entrectinib treatment for 7 months



C. Antitumor activity in a *ROS1* fusion-driven intracranial tumor model



Kim DH et al AACR 2019 Abstract # 3071

Repotrectinib: Potential Best-in-Class TRK Inhibitor

Data Presented at Annual AACR Conference on March 31

	Ba/F3 Cell Proliferation Assay IC_{50} (nM)										
	LMNA-TRKA					ETV6-TRKB		ETV6-TRKC			
TRK Inhibitor*	WT	G595R	G667C	F589L	G595R/ F589L	WT	G639R	WT	G623R	G623E	F617I
Repotrectinib	<0.1	0.2	9.2	<0.2	13.7	<0.1	1.7	<0.2	1.0	0.6	<0.2
LOXO-195	4.6	15.1	**94.9**	**26.5**	480.8	1.4	20.8	4.0	23.9	36.1	**40.9**
Larotrectinib	18.9	2817	**1863**	**597**	>10000	28.2	2500	41.4	7500	1486	**4000**
Entrectinib	0.4	711	**186.7**	**<0.2**	1774	0.6	1577	0.8	1670	1500	**54.9**

** Other than repotrectinib, data based on evaluation of comparable proxy chemical reagent purchased from commercial sources rather than obtained from the pharmaceutical company developing the kinase inhibitor*

Key Areas of Differentiation for Repotrectinib

	Repotrectinib	Xalkori (Crizotinib)[1]	Entrectinib[1]	Vitrakvi (Larotrectinib)[1]
Development Stage	Plan to Enter Registrational Phase 2 for *ROS1*+ NSCLC and *NTRK*+ adv. solid tumors	Approved in *ROS1*+ NSCLC	PDUFA Date in August for *ROS1*+ NSCLC and *NTRK*+ adv. solid tumors	Approved for *NTRK*+ adv. solid tumors
TKI Naïve Activity (*ROS1*+ NSCLC)	✓	✓	✓	NA
TKI Pretreated Activity (*ROS1*+ NSCLC)	✓	✗	✗	NA
CNS Activity (*ROS1*+ NSCLC)	✓	✗	✓	NA
Grade 3 or 4 ALT or AST Elevation	Not reported to date	✓	✓	✓
TKI Pretreated Activity in *NTRK+* Adv. Solid Tumors (with solvent front mutations)	✓	NA	Not reported to date	Not reported to date

[1] *Based on published information and data*

Turning Point Therapeutics

TRIDENT-1:
An Ongoing Phase 1/2 Study of Repotrectinib in Patients with Advanced Solid Tumors Harboring *ROS1*, *NTRK1-3*, or *ALK* Rearrangements

Data Presented at ASCO 2019

(Presented May 31, 2019)

TRIDENT-1: A Phase 1/2 Study of Repotrectinib

Study Design/Eligibility (Phase 1)

- Advanced solid tumors harboring *ROS1/NTRK1-3/ALK* fusions
- No limit on prior lines of therapy
- Asymptomatic CNS metastases allowed

Phase 1 Primary Objective

- Determine the MTD and RP2D

Phase 1 Secondary Objectives

- Safety and tolerability
- Preliminary objective response rate and clinical benefit rate

	Number of patients per dose cohort									
	40 mg QD	80 mg QD	160 mg QD	240 mg QD	160 mg BID	200 mg BID[1]	120 mg QD w/ Food	160 mg QD w/ Food	160 mg QD/BID w/Food[2]	Total
Safety population (*ROS1+, NTRK1-3+, ALK+* solid tumors)	13	12	23	10	12	2	3	5	3	83**
Efficacy population (*ROS1*+ NSCLC)	5	5	10	2	6	0	2	3	0*	33

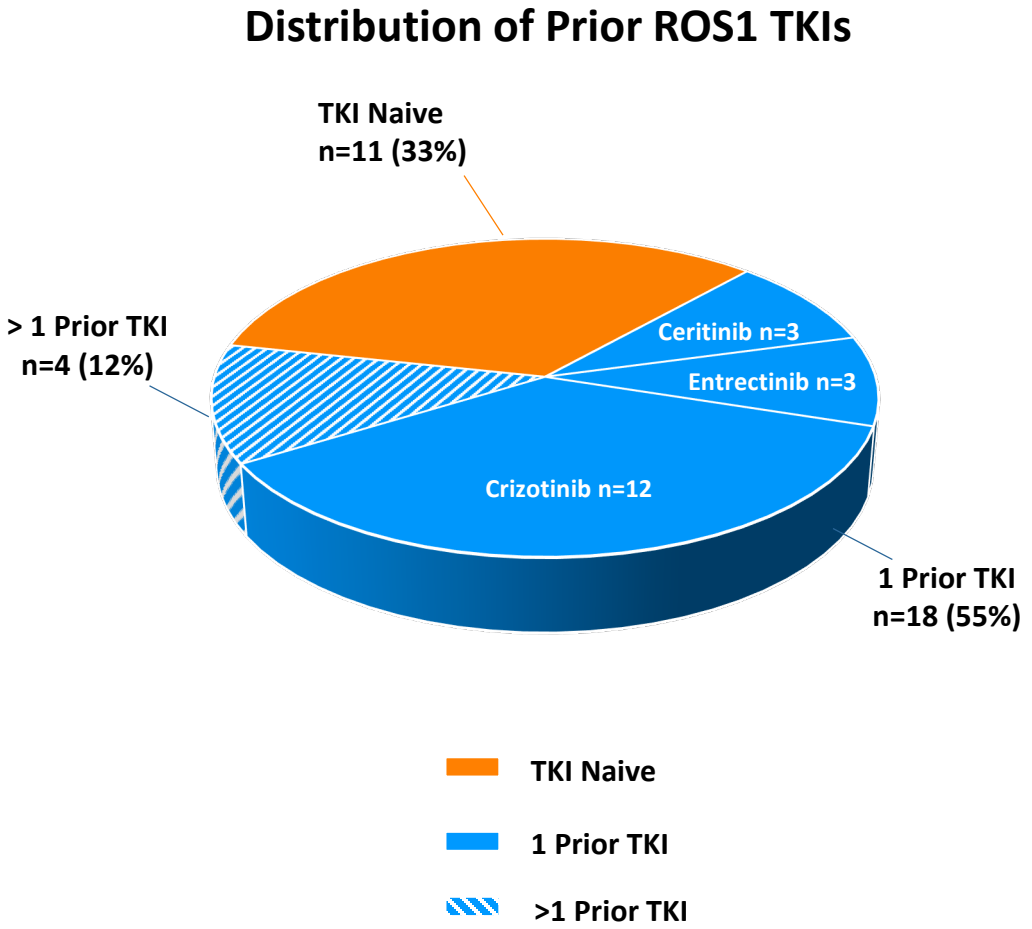
[1] 2 ALK patients enrolled
[2] 160 mg QD for one week followed by 160 mg BID
* Not yet evaluable for efficacy by BICR
** N=83 patients: 31 were *ALK*+, 9 were *NTRK*+, and 43 were *ROS1*+ (of which 33 *ROS1*+ NSCLC were evaluable for efficacy by BICR)
BICR: Blinded Independent Central Review

Data cut-off date of March 4, 2019

TRIDENT-1: *ROS1*+ NSCLC Patient Demographics

Characteristics	N=33
Age, median (range)	57 (30, 79)
Sex, female n (%)	23 (70)
Race, Asian n (%)	20 (61)
Median lines of prior systemic therapy (range)	2 (1, 8)
Prior chemotherapy, n (%)	28 (85)
CNS metastases at baseline n (%)*	18 (55)
Median # of prior ROS1 TKIs (range)	1 (0, 3)
TKI Naive, n (%)	11 (33)
TKI Pretreated, n (%)	22 (67)
1 prior TKI	18 (55)
Crizotinib only	12 (67)
Ceritinib or entrectinib	6 (33)
>1 prior TKI	4 (12)

*Assessed by Investigator



Data cut-off date of March 4, 2019

Preliminary Efficacy of Repotrectinib in TKI Naive *ROS1*+ NSCLC by BICR

TKI Naive (N=11)	
Confirmed ORR, n/N (%)	**9/11 (82%)**
95% CI (%)	(48 – 98)
ORR at 160mg QD or above	**5/6 (83%)**
Duration of response (DOR), months	
Median	**Not reached**
Range	5.6 – 17.7+
Intracranial ORR (IC-ORR)[1], n/N (%)	**3/3 (100%)**
95% CI (%)	(29 – 100)
Clinical benefit rate, n/N (%)	**11/11 (100%)**
95% CI (%)	(72 – 100)
Median follow-up time, months	**16.4**
Range	3.5+ – 19.4+

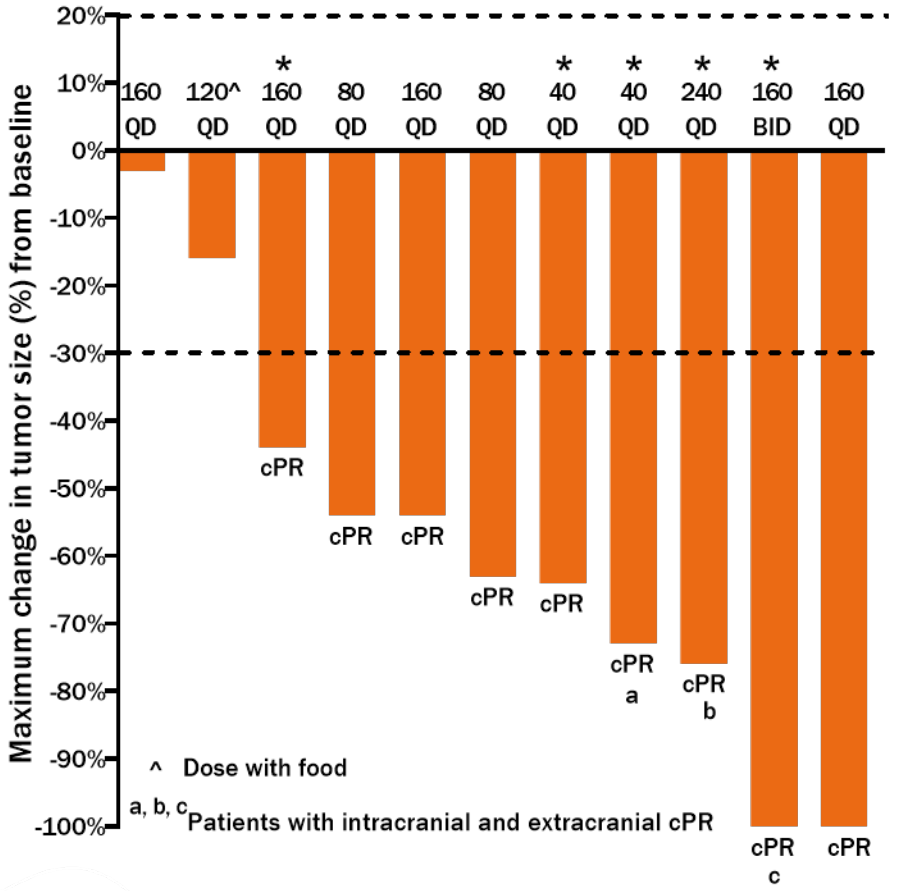
***5 of 9 patients remain in cPR from 10.9+ to 17.7+ months. 3 patients with IC-ORR remain in cPR for 10.9+, 12.1+, and 17.6+ months.**

[1] For patients with CNS measurable disease at baseline

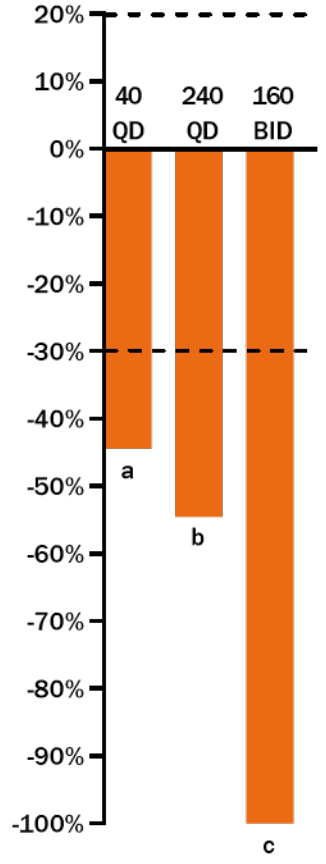
BICR: Blinded Independent Central Review

Clinical Benefit Rate: CR + PR + SD ≥ 2 Cycles

Overall Response (N=11)



Intracranial Response (N=3)



Data cut-off date of March 4, 2019

Preliminary Efficacy of Repotrectinib in TKI Pretreated *ROS1+* NSCLC by BICR

Pretreated with 1 TKI (N=18**)	
Confirmed ORR, n/N (%)	**7/18 (39%)**
95% CI (%)	(17 – 64)
ORR at 160 mg QD or above	**6/11 (55%)**
• **Crizotinib as ONLY prior TKI**	**4/7 (57%)**
IC-ORR[1], n/N (%)	**3/4 (75%)**
95% CI (%)	(19 – 99)
Clinical benefit rate, n/N (%)	**14/18 (78%)**
95% CI (%)	(52 – 94)
Median follow-up time, months	**14.6**
Range	1.4 – 14.6+

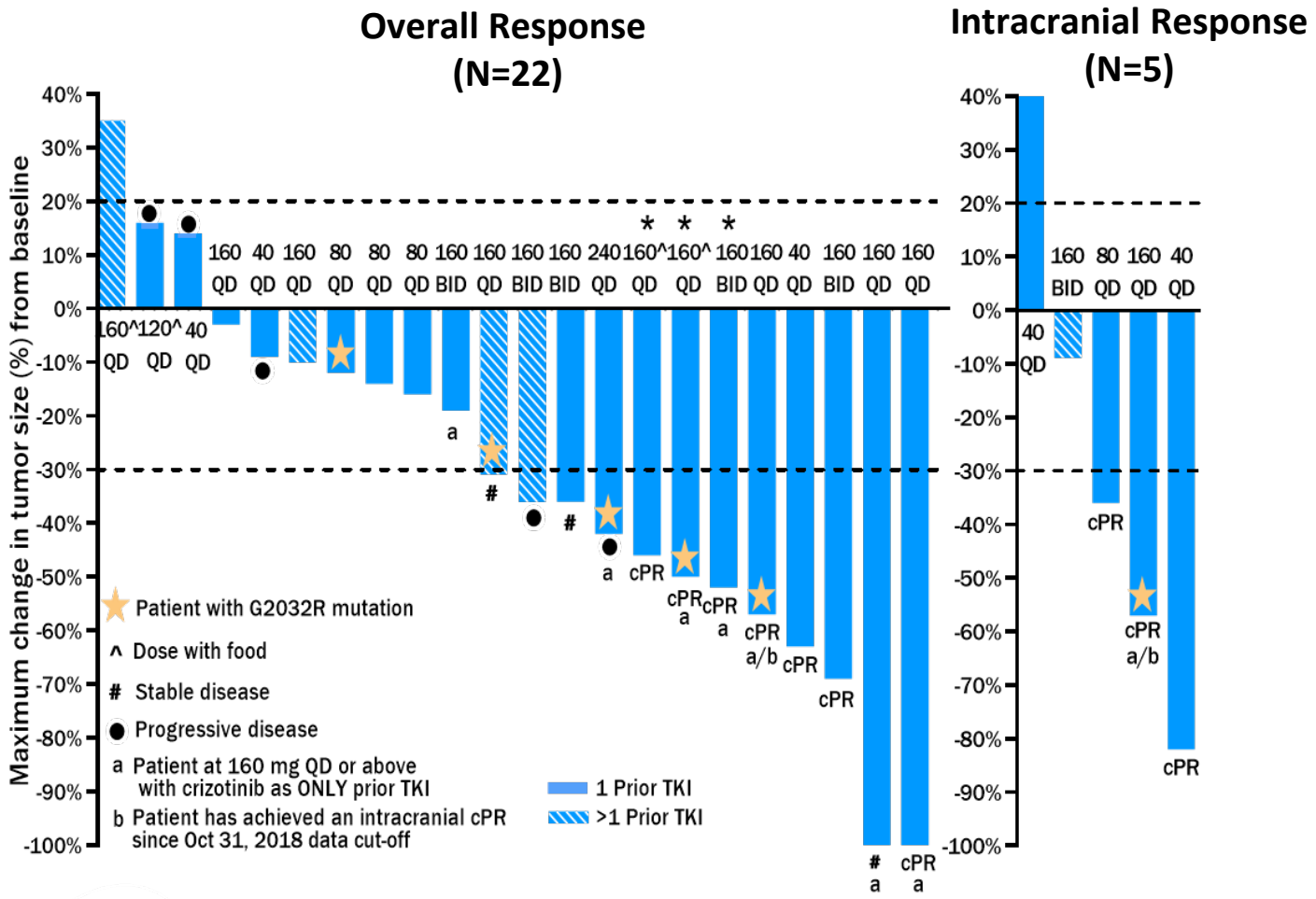
***3 of 7 patients remain in cPR from 1.0+ to 7.6+ months**

** 4 patients treated with > 1 prior TKI not included (3 of 4 had tumor regressions)

[1] For patients with CNS measurable disease at baseline

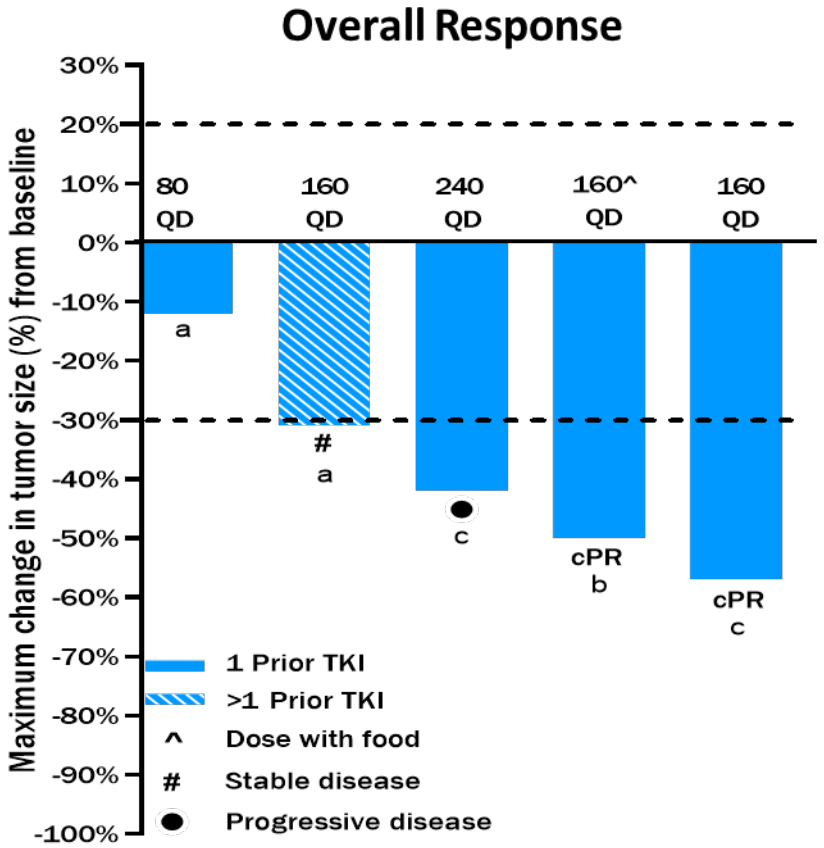
BICR: Blinded Independent Central Review

Clinical Benefit Rate: CR + PR + SD ≥ 2 Cycles



Data cut-off date of March 4, 2019

Preliminary Clinical Activity of Repotrectinib Against *ROS1* G2032R Solvent Front Mutation

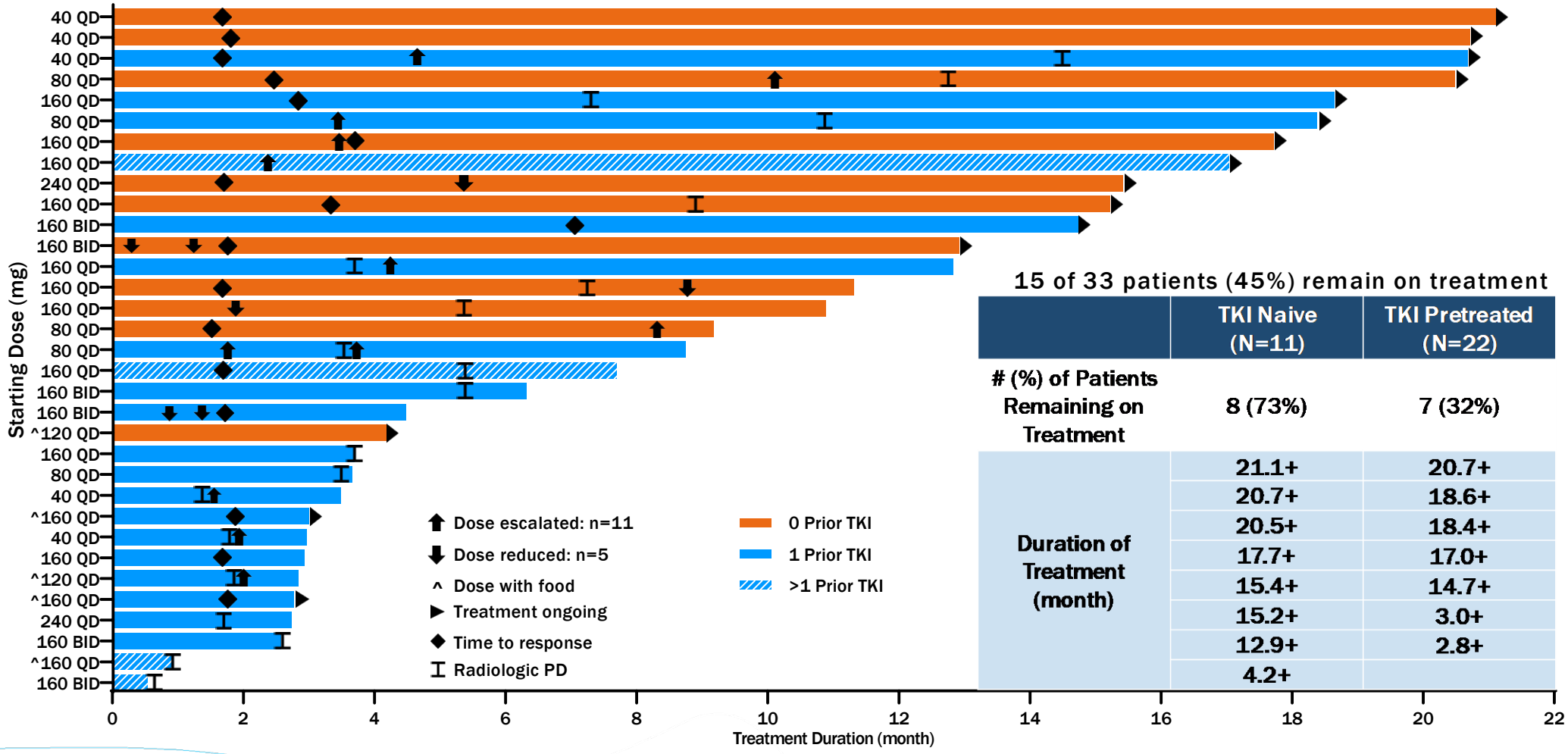


G2032R identified in (a) plasma cfDNA; (b) tumor tissue; (c) plasma cfDNA and tumor tissue

- *ROS1* G2032R identified by plasma cfDNA or tissue NGS test in 5 patients who had prior crizotinib treatment
- All 5 patients experienced tumor regressions on repotrectinib
- **<u>Confirmed ORR</u>: 2/5 (40%)**
  - 2/3 (67%) for 160 mg QD and above with 1 prior TKI
    - 1 cPR at 160 mg QD with food (DOR 1.0+ months and remains on treatment at 3.0+ months)
    - 1 cPR at 160 mg QD (DOR 4.4 months and remains on treatment at 18.6+ months)

Data cut-off date of March 4, 2019

Duration of Repotrectinib Treatment in N=33 *ROS1*+ NSCLC by BICR



15 of 33 patients (45%) remain on treatment

	TKI Naive (N=11)	TKI Pretreated (N=22)
# (%) of Patients Remaining on Treatment	8 (73%)	7 (32%)
Duration of Treatment (month)	21.1+	20.7+
	20.7+	18.6+
	20.5+	18.4+
	17.7+	17.0+
	15.4+	14.7+
	15.2+	3.0+
	12.9+	2.8+
	4.2+	

Data cut-off date of March 4, 2019

Safety Summary: Treatment-Emergent and Treatment-Related AEs

Adverse Event	All Treated Patients (N=83)				
	TEAEs (≥10% of patients)			TRAEs	
	All Grades n(%)	Grade 3 n(%)	Grade 4* n(%)	Grade 3 n(%)	Grade 4 n(%)
Dizziness	47 (56.6)	2 (2.4)	---	2 (2.4)	---
Dysgeusia	42 (50.6)	---	---	---	---
Dyspnea	25 (30.1)	5 (6.0)	1 (1.3)	1 (1.2)	---
Fatigue	25 (30.1)	2 (2.4)	---	---	---
Constipation	24 (28.9)	---	---	---	---
Paresthesia	24 (28.9)	---	---	---	---
Anemia	23 (27.7)	10 (12.0)	---	3 (3.6)	---
Nausea	19 (22.9)	2 (2.4)	---	---	---
Cough	17 (20.5)	---	---	---	---
Pyrexia	16 (19.3)	---	---	---	---
Headache	14 (16.9)	1 (1.2)	---	---	---
Vomiting	13 (15.7)	---	---	---	---
Upper respiratory tract infection	11 (13.3)	---	---	---	---
Ataxia	10 (12.0)	---	---	---	---
Pain in extremity	10 (12.0)	1 (1.2)	---	---	---
Abdominal pain	9 (10.8)	---	---	---	---
Muscular weakness	9 (10.8)	1 (1.2)	---	---	---

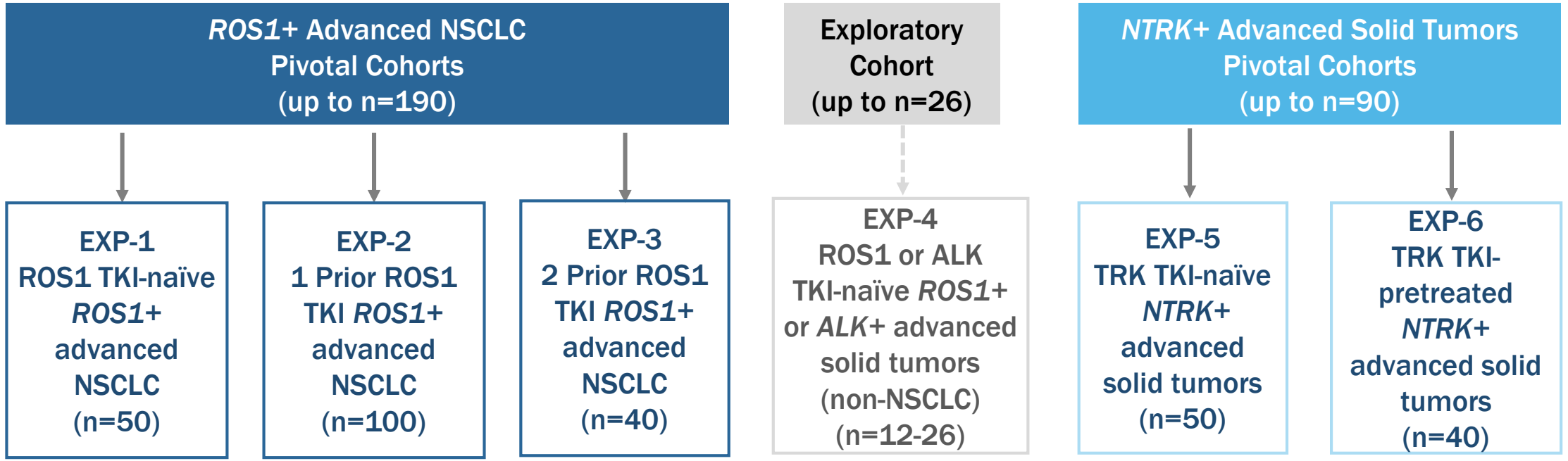
- **Repotrectinib was generally well tolerated**
- **Majority of treatment emergent adverse events (TEAEs) were Grade 1 or Grade 2**
  - No Grade 3 or Grade 4 ALT or AST elevations
  - No cases of dizziness have led to treatment discontinuation
- **Four DLT events**:
  - Grade 2 or 3 dizziness
    - 160 mg BID (n=2)
    - 240 mg QD (n=1)
  - Grade 3 dyspnea and hypoxia
    - 160 mg BID (n=1)
- **Four TEAE Grade 5 events^**
- **Treatment related adverse events (TRAEs) leading to dose modifications**
  - Dose reduction: n=8 (9.6%)
  - Dose interruption: n=2 (2.4%)
  - Drug discontinuation: n=2 (2.4%)

**Add'l Grade 4 TEAEs: cerebrovascular accident, dyspnea, influenza, hyperkalemia, bacterial pneumonia (n=1 each), respiratory failure (n=2); None were determined to be related to treatment*

^ Grade 5 TEAEs: respiratory failure (n=2), sepsis, sudden death (n=1 each); Only the case of sudden death was determined to be possibly related to treatment

Data cut-off date of March 4, 2019

Pivotal Phase 2 Portion of TRIDENT-1: Plan to Initiate in 2H 2019 (n=~310)



- **Phase 2 Primary Objective**
  - cORR by BICR in each expansion cohort
- **Phase 2 Secondary Objectives**
  - DOR, PFS, and OS
  - IC-ORR and CNS-PFS

Conclusions

- **TRIDENT-1 Phase 1 data support repotrectinib as a potential best-in-class *ROS1* agent in advanced NSCLC**
- **Preliminary clinical activity demonstrated across 7 dose cohorts in *ROS1+* NSCLC patients**
  - TKI-naive:
    - cORR 82% (9/11); median DOR not yet reached
  - TKI-pretreated:
    - 1 prior TKI: cORR 39% (7/18)
      - cORR 57% (4/7) in crizotinib-pretreated patients at 160 mg QD and above
  - CNS activity observed in both TKI-naïve and TKI-pretreated patients
- **Repotrectinib is a next-generation ROS1/TRKA-C/ALK inhibitor designed to overcome TKI resistant mutations**
  - All 5 *ROS1+* NSCLC patients with the G2032R SFM experienced tumor regressions with a cORR of 40%
- **Repotrectinib was well tolerated with a manageable safety profile**
  - Dizziness is an on-target AE associated with TRK inhibition and manageable
  - Most AEs managed without dose modification and rarely led to discontinuation
- **Pivotal Phase 2 portion of TRIDENT-1 planned to initiate in 2H 2019**

Data cut-off date of March 4, 2019

Turning Point Therapeutics

Interim Data Over Time, Proof of Concept in NTRK

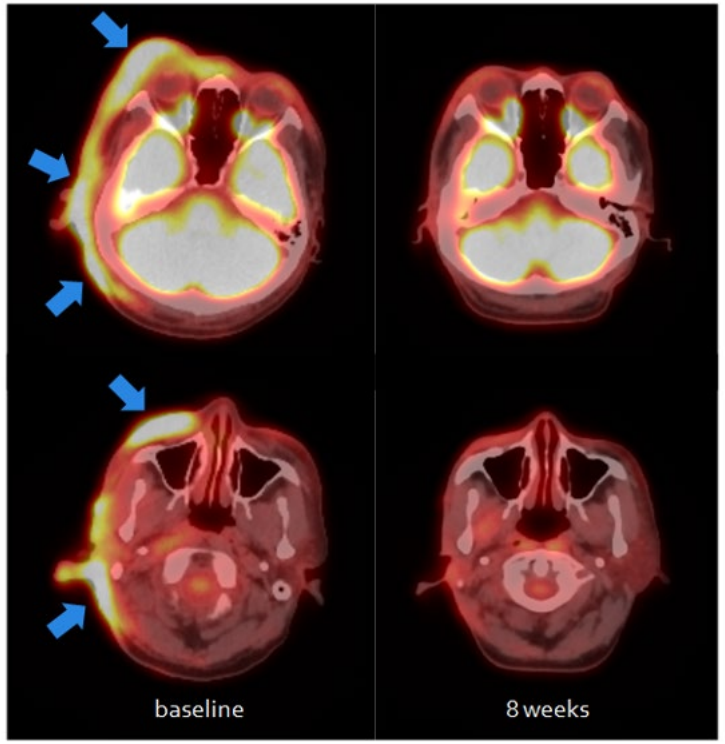
Preliminary Clinical Activity of Repotrectinib in TKI-Naïve *ROS1+* NSCLC

	2018 ASCO Presentation (by PI 4/17/2018 data cutoff)	2018 WCLC Oral Presentation (by BICR 7/13/2018 data cutoff)	ASCO Abstract 2019 (by BICR 10/31/2018 data cutoff)	2019 ASCO Presentation (by BICR 3/4/2019 data cutoff)
ALL ROS1 NSCLC	N=29	N=27	N=28	N=33
TKI - Naive	N=10	N=10	N=10	N=11
ORR (%) (95%CI)	7/10 (70)	8/10 (80) (44 - 97)	9/10 (90) (56 - 100)	9/11 (82) (48 – 98)
ORR at 160 mg QD or above			5/6 (83)	5/6 (83)
IC ORR n (%) (95% CI)		3/3 (100) (29 - 100)	3/3 (100) (29 - 100)	3/3 (100) (29 - 100) All 3 remain in cPR
CBR (%), (95%CI)		10/10 (100) (69 - 100)	10/10 (100) (69 - 100)	11/11 (100) (72 – 100)
mDOR (month), (95%CI)			Not Reached (5.6 – NR) 5 of 9 remain in cPR (5.5+ to 14.9+ months)	Not Reached (5.6 – NR) 5 of 9 in cPR (10.9+ to 17.7+ months)
Median FU time Range			16.4 months (5.3 – 16.6+)	16.4 months (3.5+ - 19.4+)

Preliminary Clinical Activity of Repotrectinib in Pretreated *ROS1+* NSCLC

		2018 ASCO Presentation (by PI 4/17/2018 data cutoff)	2018 WCLC Oral Presentation (by BICR 7/13/2018 data cutoff)	ASCO Abstract 2019 (by BICR 10/31/2018 data cutoff)	2019 ASCO Presentation (by BICR 3/4/2019 data cutoff)
ALL ROS1 NSCLC		29	27	28	33
TKI - pretreated		N=19	N=17	N=18	N=22
ORR (%) (95%CI)		2/19 (11)	3/17 (18) (4 - 44)	5/18 (28) (10 - 53)	7/22 (32) (14 – 55)
ORR	1 prior TKI		3/14 (21)	5/15 (33)	7/18 (39)
	1 prior TKI 160 mg QD or above		2/8 (25)	4/9 (44)	6/11 (55)
	1 prior TKI Crizotinib only 160mg QD and above		2/5 (40)	3/6 (50)	4/7 (57)
	2 or more prior TKIs		0/3 (0)	0/3 (0)	0/4 (0)
IC ORR n (%) 1 Prior TKI			1/4 (25) 1/3 (33)	2/4 (50) 2/3 (67)	3/5 (60) 3/4 (75)
CBR (%), (95% CI)			13/17 (76) (56 - 97)	14/18 (78) (52 - 94)	16/22 (73) (50 – 89)
SFMs		1 PR reported	1/4 (25)	1/4 (25)	2/5 (40)
DOR (month) # still in response (+)			Not reported	1 of 5 responders still in cPR at 1.9+ months	3 of 7 responders still in cPR at 1.0+ to 7.6+ months
Median Follow Up Time Range				12.9 months (0.6 – 14.5)	14.6 months (1.4 – 16.6+)

Repotrectinib Demonstrated Durable Activity Against TRKC G623E Solvent Front Mutation (Highlighted in Cancer Discovery[1])



- *ETV6-NTRK3*+ mammary analogue secretory carcinoma with acquired TRKC G623E solvent front substitution
- Prior TKI treatment: crizotinib, entrectinib and entrectinib+trametinib
- Confirmed PR with DOR of 9.8 months and DOT 17.9 months at time of data cut-off

[1] *Drilon, et al, Cancer Discovery, Aug. 9, 2018*

Turning Point Therapeutics

Pipeline Programs:

TPX-0022 MET/CSF1R/SRC Inhibitor

TPX-0046 RET/SRC Inhibitor

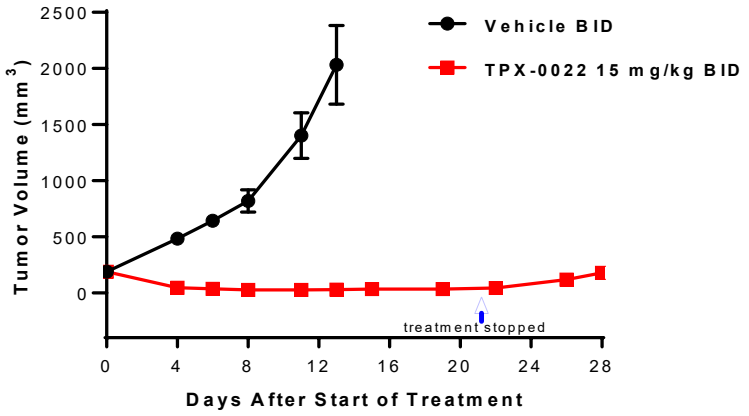
TPX-0022 MET/CSF1R/SRC Inhibitor: IND Cleared in May 2019

Target Patient Populations	• Advanced solid tumors with abnormal MET/HGF or CSF1R/CSF1 signaling
Mechanism of Action	• Selective multi-targeted kinase inhibitor of MET/CSF1R/SRC • Novel MOA by simultaneously targeting *MET*-driven tumor cells and modulating the TME • Highly potent towards MET mutations/rearrangements such as exon 14 skipping mutations
Preclinical Data	• Demonstrated high potency in inhibiting MET, SRC and CSF1R in enzymatic and cell-based assays • Anti-tumor activity and inhibition of MET phosphorylation in xenograft tumor models
Development Status	• IND cleared in May 2019; initiate Phase 1 trial in 2H 2019

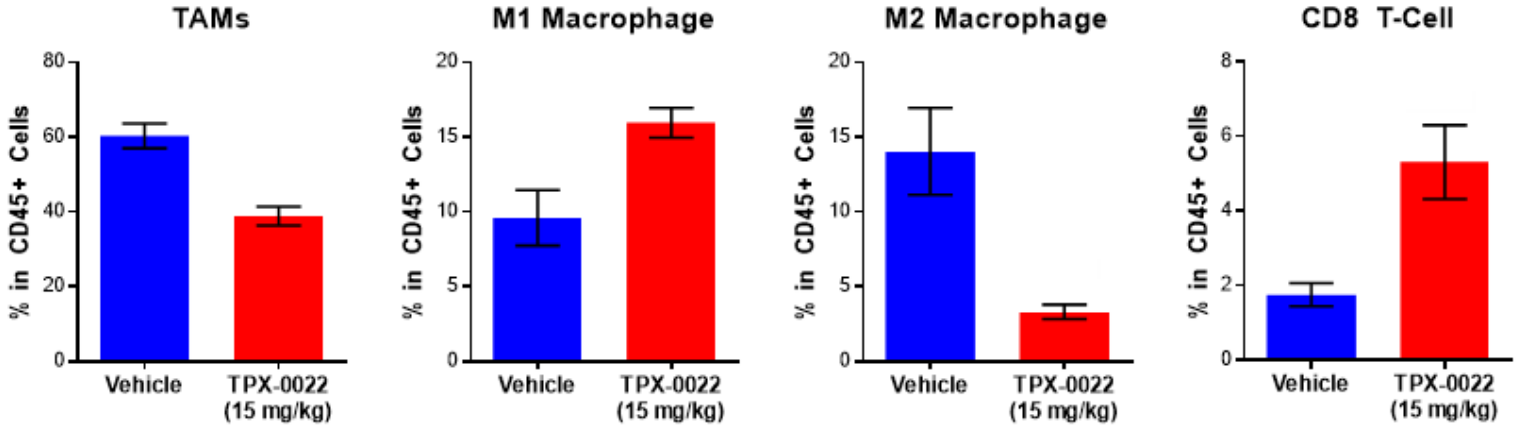
TPX-0022: A Potent MET/CSF1R/SRC Inhibitor

	Enzymatic IC_{50} (nM) at 10 μM ATP			Cell Proliferation IC_{50} (nM)		
Inhibitor*	MET	SRC	CSF1R	Gastric MKN-45 (MET)	Gastric SNU5 (MET)	Ba/F3 ETV6-CSF1R
TPX-0022	0.14	0.12	0.71	<0.2	<0.2	14
Capmatinib	0.20	ND	ND	<0.2	<0.2	ND
Crizotinib	4.0	ND	ND	10.5	2.8	ND
PLX-3397	ND	ND	ND	ND	ND	581

Antitumor effect of TPX-0022 in the LU2503 patient-derived xenograft tumor model of lung cancer with MET gene amplification and Exon 14 deletion



TPX-0022 Modulated Tumor Associated Macrophages to Promote a Pro-inflammatory Anti-tumor Microenvironment in MC38 Syngeneic Mouse Tumor Model



** Data based on evaluation of corresponding proxy chemical compound purchased from a commercial source rather than from the pharmaceutical company commercializing or developing the inhibitor*

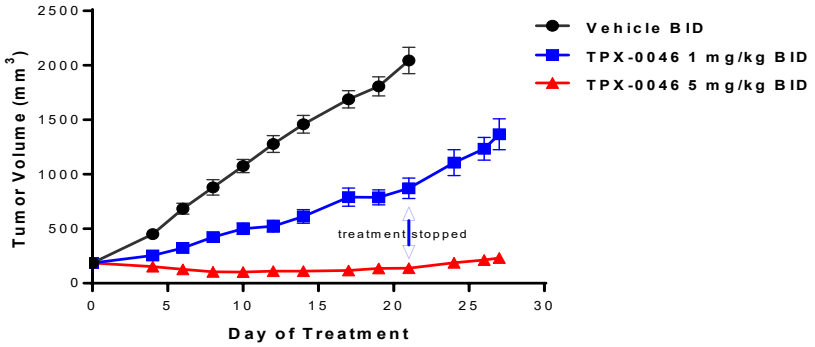
TPX-0046 Novel RET/SRC Inhibitor: In IND Enabling Studies

Target Patient Populations	• Advanced solid tumors with abnormal RET genes (both TKI-pretreated and TKI-naïve)
Mechanism of Action	• Selective multi-targeted kinase inhibitor of RET and SRC • Novel structure compared to other RET inhibitors to potentially address resistant mutations that may emerge • Inhibition of SRC has the potential to reduce bypass resistance and increase the effect seen with RET inhibitors
Preclinical Data	• Strong potency against wildtype (WT) and many mutated RETs including solvent front mutation G810R • Showed dose-dependent inhibition in cancer cell- and patient-derived tumor models
Development Status	• In IND-enabling studies; plan to submit IND and initiate Phase 1 trial in 2H 2019

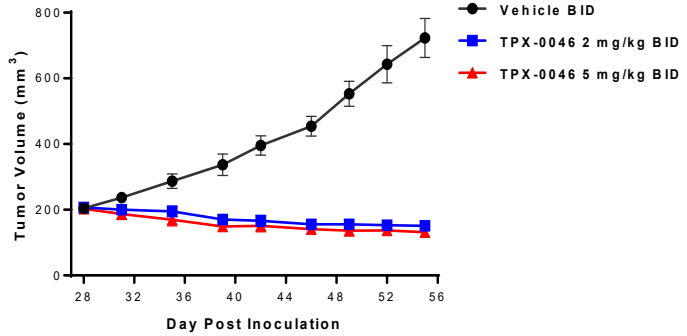
TPX-0046 Potently Inhibited RET and SRC in Enzymatic and Cell Assays and Demonstrated Dose-Dependent Tumor Growth Inhibition in Xenograft Models

	Enzymatic Kinase Activity at 10 μM ATP IC_{50} (nM)				Cell Proliferation IC_{50} (nM)				
Inhibitor	RET	RET-CCDC6	SRC	VEGFR2	Ba/F3 KIF5B-RET WT	Ba/F3 KIF5B-RET G810R (solvent front mutation)	TT[1] (RET C634W)	LC2/ad[2] (CCDC6-RET)	Ba/F3 KIF5B-RET V804M (gatekeeper mutation)
TPX-0046	1.01	0.691	1.46	>1000	1.2	15.3	0.9	1	444
Cabozantinib[3]	ND	ND	ND	ND	142	1344	399	500	3400

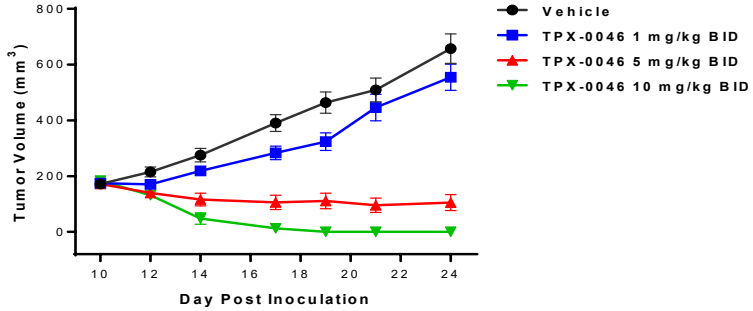
Antitumor effect of TPX-0046 in the CR1520 patient-derived xenograft model of colorectal cancer with the NCOA4-RET fusion gene



Antitumor effect of TPX-0046 in a TT cell-derived xenograft tumor model of medullary thyroid carcinoma with the RET C634W mutation



Antitumor effect of TPX-0046 in a Ba/F3 cell-derived xenograft tumor model with the KIF5B-RET fusion harboring the G810R mutation



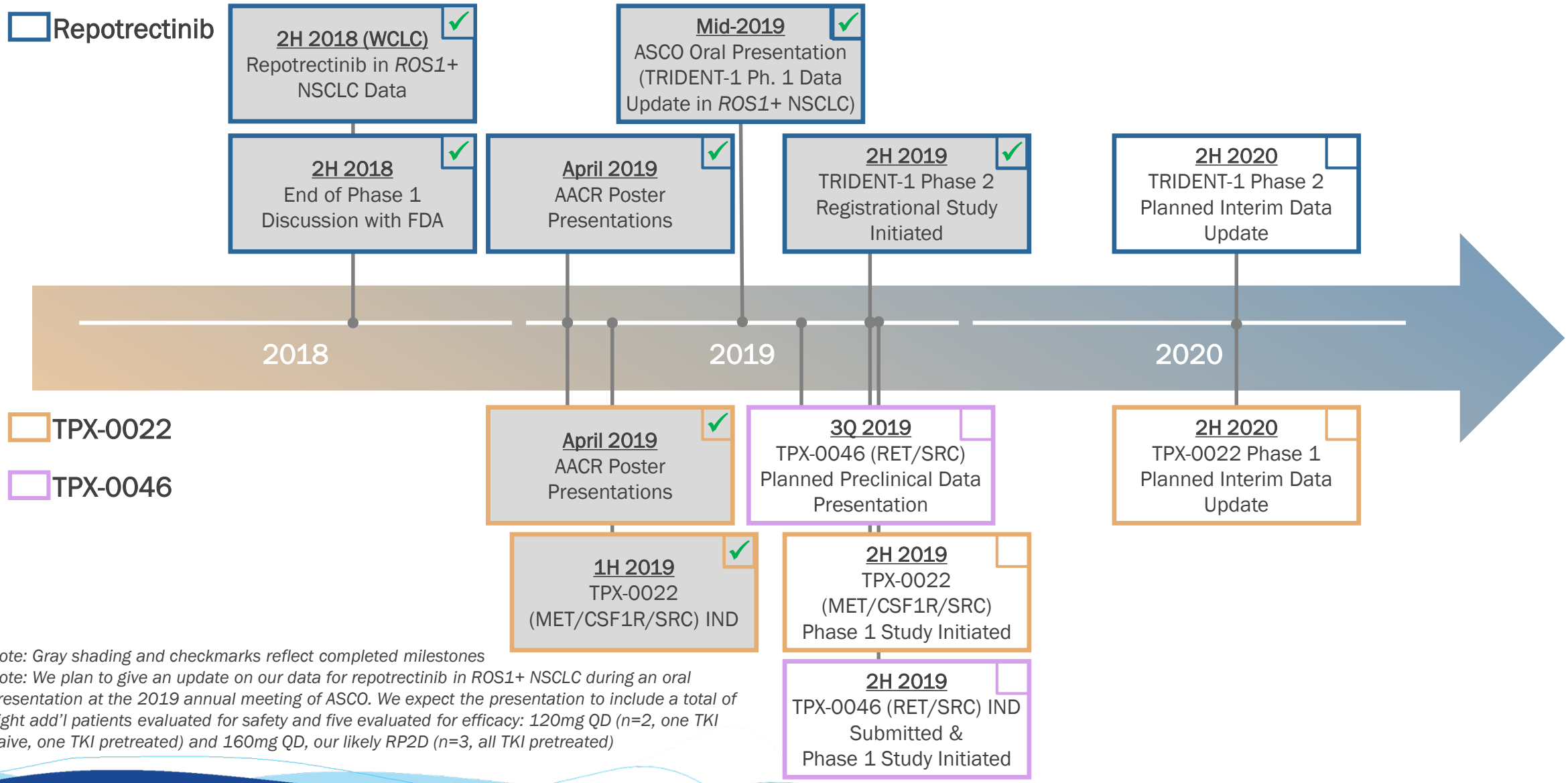
ND: Not determined

[1] TT is a stable cancer cell line derived from a human medullary thyroid carcinoma with a C634W mutation

[2] LC2/ad is a stable cancer cell line derived from a human lung adenocarcinoma with the CCDC6-RET fusion gene

[3] Data based on evaluation of corresponding proxy chemical compound purchased from a commercial source rather than from the pharmaceutical company commercializing or developing the kinase inhibitor

Key Milestones



Note: Gray shading and checkmarks reflect completed milestones

Note: We plan to give an update on our data for repotrectinib in ROS1+ NSCLC during an oral presentation at the 2019 annual meeting of ASCO. We expect the presentation to include a total of eight add'l patients evaluated for safety and five evaluated for efficacy: 120mg QD (n=2, one TKI naive, one TKI pretreated) and 160mg QD, our likely RP2D (n=3, all TKI pretreated)

Turning Point Therapeutics

Precision Oncology Medicines for Treatment Resistant Cancers

Company Overview

June 2019