



CORPORATE OVERVIEW

February 2021

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OUR VISION

We aspire to conquer cancer by addressing the unmet medical needs of our patients with paradigm-shifting therapeutics

Vincerx Highlights



MANAGEMENT TEAM

- Cohesive, accomplished management team
- Highly engaged scientific advisory board and chair
- Proven track record of successful drug development & approvals, company creation, fundraising and value creation



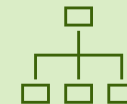
ASSETS

Clinical small molecule:

- Highly selective PTEFb [CDK9] inhibitors (oral and IV) in Phase 1; signs of clinical activity in double-hit DLBCL

Preclinical bioconjugation platform:

- SMDC for solid tumors
- CXCR5 ADC for B-cell malignancies
- CD123 ADC for AML



BUSINESS STRATEGY

- Develop oncology therapies to address unmet patient needs with accelerated approval potential
- Bayer support in the start-up process
- Develop each asset to POC and optimize commercial value of each asset



INNOVATIVE PROPRIETARY PLATFORMS

- Modular bioconjugation platform
- Small Molecule Drug Conjugate (SMDC) for solid tumors
- Next generation ADC with novel linker and warhead

Vincerx Founders



AHMED HAMDY, MD
CEO

- Cofounder of Acerta Pharma
- Former CMO of Pharmacyclics, leading developer of Imbruvica®
- Proven track record for assembling experienced teams that deliver from INDs to NDAs



RAQUEL IZUMI, PhD
COO

- Cofounder of Acerta Pharma
- Former Sr Director of Clinical Development of Pharmacyclics
- Extensive drug development experience from preclinical stages through NDA submission



TOM THOMAS, JD

- Outside general counsel
- Extensive experience in venture, finance, M&A & IPO
- Partner, Pillsbury Winthrop Shaw Pittman LLP



STUART HWANG, PhD
CBO

- Biotech corporate development executive
- Leadership roles at multiple startups, Astex and Agilent in licensing, fundraising & M&A
- Led drug and diagnostic R&D teams at SuperGen, Cor Tx(Millennium), Celera and UCSF/LBNL



JOHN BYRD, MD

- Scientific Advisor: World-renowned leader in drug development in hematologic malignancies (CLL, AML)
- D Warren Brown Chair of Leukemia Research at OSU, Comprehensive Cancer Center
- CMO, Beat AML LLC, Leukemia & Lymphoma Society

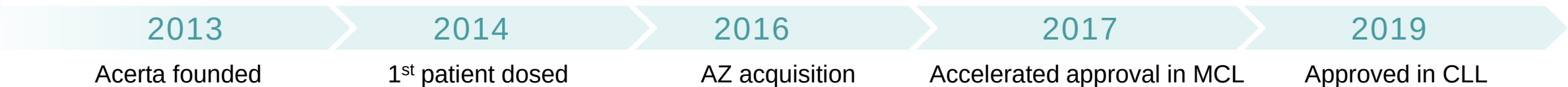
Management Team Experience - Proven Track Record



\$7B acquisition by AstraZeneca in 2016 for acalabrutinib in Phase 3

Management Team's Contribution

- Founded Acerta with acalabrutinib at preclinical stage
- Accelerated approval in 4 years



\$975M partnership with Janssen in 2011 [\$150 upfront, \$825M in milestones]

\$21B acquisition of Pharmacyclics by AbbVie in 2015

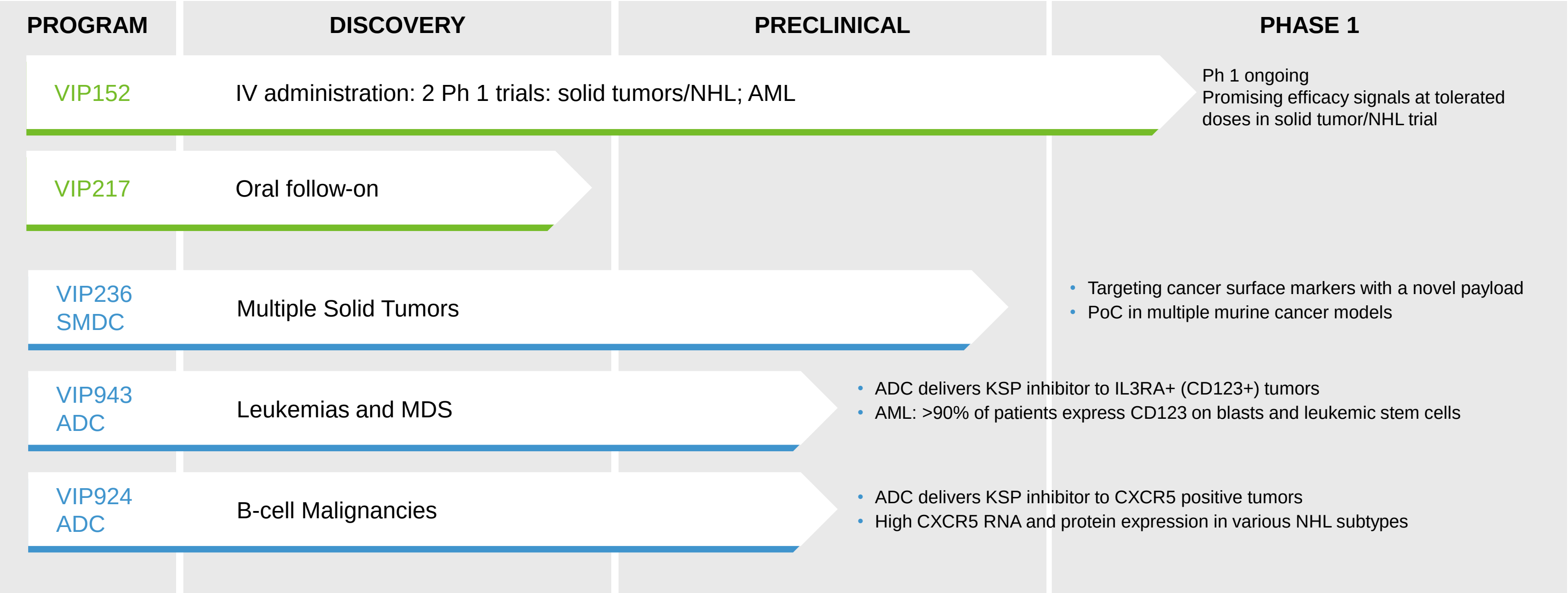
Management Team's Contribution

- Developed ibrutinib from preclinical through Phase 2 in < 3 years
- All three Phase 2 studies garnered Break-Through Designation and Accelerated Approvals

Vincerx Pipeline

PTEFb

Bioconjugation

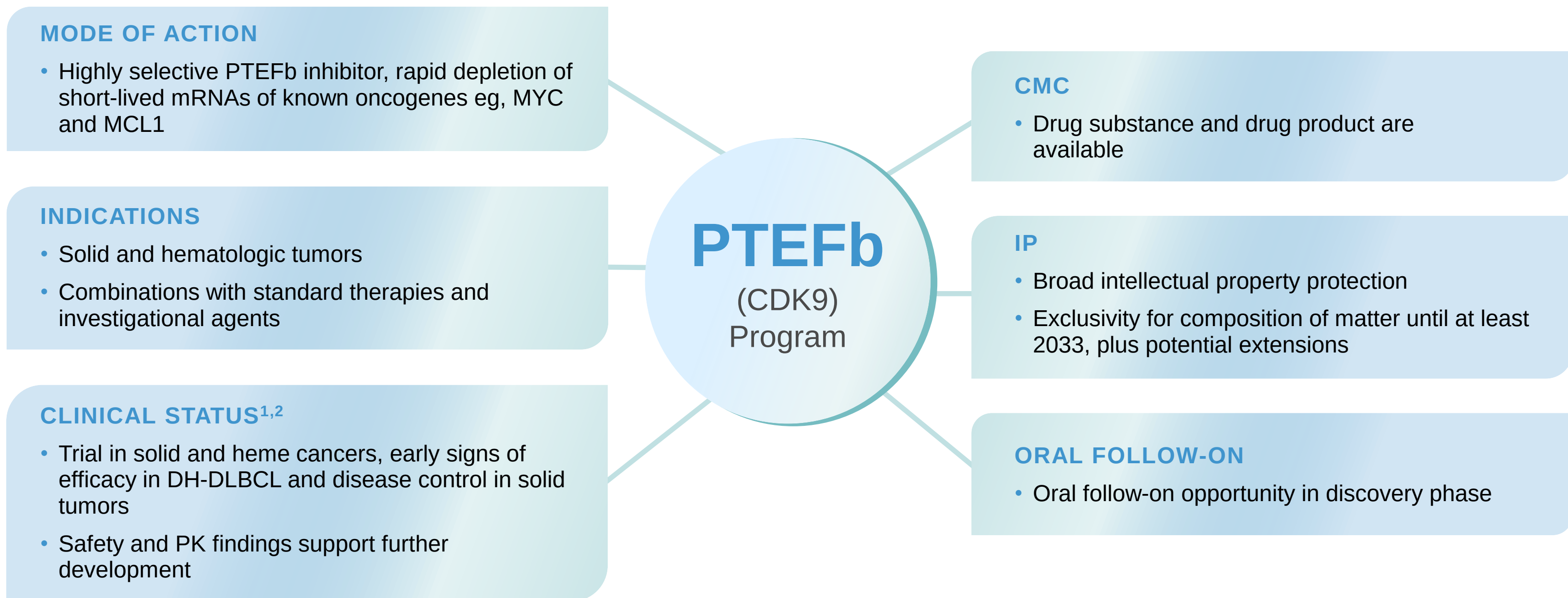


ADC = antibody-drug conjugate; AML = acute myeloid leukemia; MDS = myelodysplastic syndromes; NHL = nonHodgkin lymphoma; PoC = proof of concept; PTEFb = positive transcription elongation factor b; SMDC= small molecule drug conjugate

PTEFb PROGRAM

**VIP152 IV (Phase 1)
VIP217 Oral (Discovery)**

Summary of PTEFb (CDK9) Portfolio – Clinical & Discovery

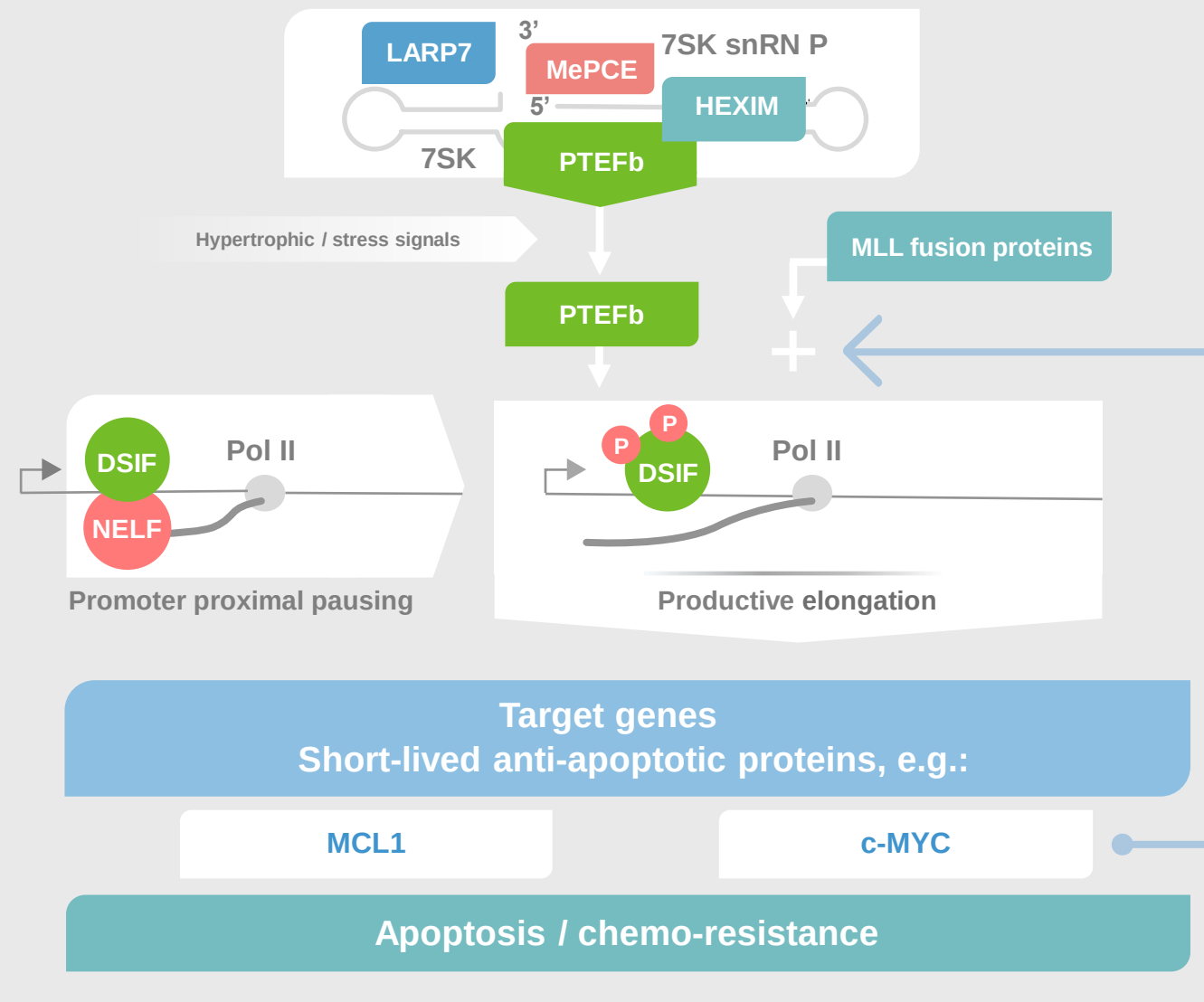


1. Blood (2018) 132 (Supplement 1): 4055.

2. JCO (2018) 36 (15): 2507.

PTEFb: A Novel Target for Oncology

After its release from an inhibitory complex, PTEFb starts the elongation of transcription by phosphorylation of RNA pol II



PTEFb [CDK9]

- Positive transcription elongation factor beta is a key regulator of transcription through phosphorylation of RNA polymerase II
- A key target to address transcriptional addiction in cancer
- Inhibition causes rapid depletion of short-lived mRNA transcripts of known oncogenes eg, MCL1 and MYC

Role of MCL1

- Drives tumor growth and resistance to apoptosis in various heme and solid tumor entities
- Potential PD biomarker: Induction of apoptosis
- Inhibitors currently in Phase 1

Role of MYC

- Aberrations like translocation, amplification and overexpression may lead to MYC dependency in oncogenesis
- Frequently (>40%) observed in heme and solid tumor indications
- Difficult to target

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CDK9 is a Clinically Validated Target

	VIP152 Vincerx	Dinaciclib Merck	Alvocidib (Flavopiridol) Tolero		
Patients	Double hit DLBCL [MYC driven]	r/r CLL [MCL1 driven]	r/r CLL [MCL1 driven]	Untreated AML	r/r AML, MCL1 dependent
Treatment	VIP152 monotherapy	Dinaciclib monotherapy vs ofatumumab	Alvocidib monotherapy	Alvocidib + cytarabine + mitoxantrone vs 7+3	Alvocidib + cytarabine + mitoxantrone
Trial	Phase 1/1b dose escalation and dose expansion	Randomized Phase 3 (stopped early)	Two Phase 2's	Randomized Phase 2	Phase 2
Response	ORR: 29% (2/7), both PET-negative CRs	Dinaciclib ORR: 40% (8/20) Ofatumumab ORR: 8% (2/24)	Study 1 ORR: 54% (34/64) Study 2 ORR: 25% (41/164)	Alvo/cy/mit CR: 70% (76/109) 7+3 CR: 46% (26/56)	CR/CRi: 57% (13/23)
Durability	2.3 to 3.6 years	Dinaciclib mPFS of 13.7 mo Ofatumumab mPFS of 5.9 mo	Study 1: mPFS of 8.6 mo Study 2: mPFS of 7.6 mo	No difference in survival	mDoR of 8.5 mo for patients achieving CR/CRi

VIP152 is the Most Selective CDK9 Inhibitor in the Clinic

Programs	VIP152 Vincerx	AZD4573 AZ	Dinaciclib Merck	CYC065 Cyclacel	Alvocidib (Flavopiridol) Tolero	Voruciclib MEI Pharma
Selectivity	CDK9	CDK1/9	CDK1/2/5/9	CDK2/3/5/9	Pan CDK	Pan CDK
Development Stage	P1	P1	P3 mono P2 combo	P1	P2	P1 mono and combo BCL2
Type of tumor	Hematologic & Solid tumors	Hematologic	CLL stopped Solids combo with IO	AML, CLL, ALL Solid tumors	AML/MDS Combos	B-cell malignancies and AML
IC₅₀ on CDK9	3 nM	14 nM	1-4 nM	26 nM	6 nM	1 nM
Half life	4 h	<3 h	3 h	~1 h	2-4 h	30 h
Route of admin	IV	IV	IV	Oral & IV	IV	Oral

VIP152 Highly Selective and Potent CDK9 Inhibitor

					Activity against all non-CDK kinases with <50x higher KDs	
Assay	VIP152	Kinase	Kd [nM] @ DiscoverRx	IC ₅₀ [nM] @ Millipore	Kinase	Activity [nM]
IC ₅₀ CDK9 [nM] low ATP	3	CDK9	1.3	13**	CDK9	1.3
IC ₅₀ CDK9 [nM] high ATP	4	CDK1	n.a.	192	GSK3a	7.4
High potency is independent of [ATP]		CDK2	710	158	IRAK1	61
		CDK3	540	318	High selectivity over other CDKs, incl CDK2 Favorable non-CDK kinase selectivity profile	
		CDK4- cyclinD1	120	n.d.		
		CDK4- cyclinD3	68	n.d.		
		CDK5	4900	286		
		CDK6	n.a.	1048		
		CDK7	24*	>10000		
		CDK8	25000	n.d.		
		CDK11	not active	n.d.		

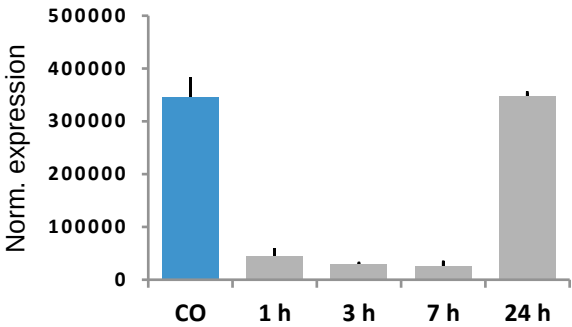
* No cyclin co-expression

** Probably lower limit of quantification

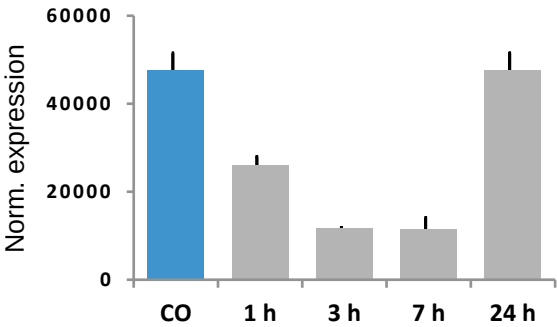
MoA – VIP152 Inhibits the Transcription of MYC and MCL1

Reduction of MYC & MCL1 mRNA levels

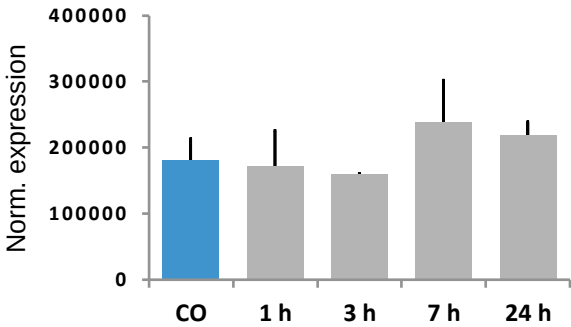
MYC mRNA



MCL1 mRNA

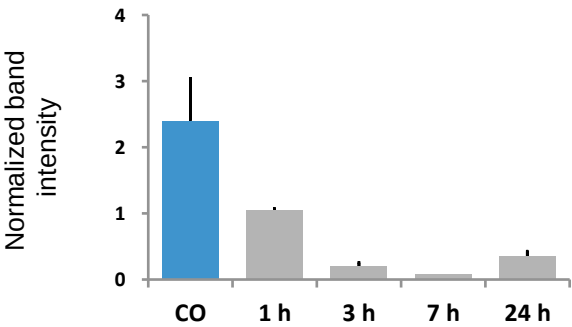


BCL2 mRNA



Durable reduction of MYC protein levels

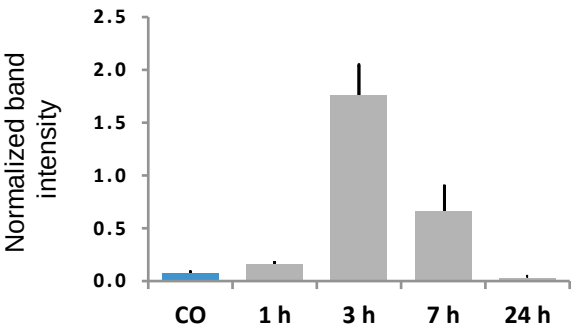
MYC Protein



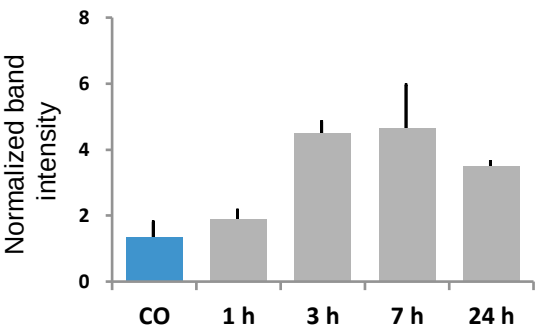
In vivo MoA in JJN3 multiple myeloma xenografts in mice upon a single dose of 15 mg/kg VIP152 IV

Induction of apoptosis

Cleaved Caspase 3 p17



Cleaved PARP



VIP152 (IV) - Clinical Trial Design & Status

Two Phase 1 clinical trials

FIRST-IN-HUMAN STUDY (17496; NCT02635672)

Dose escalation (N=31)

MTD

Expansion cohort (ongoing; N=6)

- Once weekly IV; 30-min infusion
- 21-day cycles
- No biomarker selection patient population (ie, all-comer advanced cancer)

- At recommended Phase 2 dose of 30 mg
- Double-hit DLBCL

AML study (18117; NCT02745743)

Dose escalation (N=21)

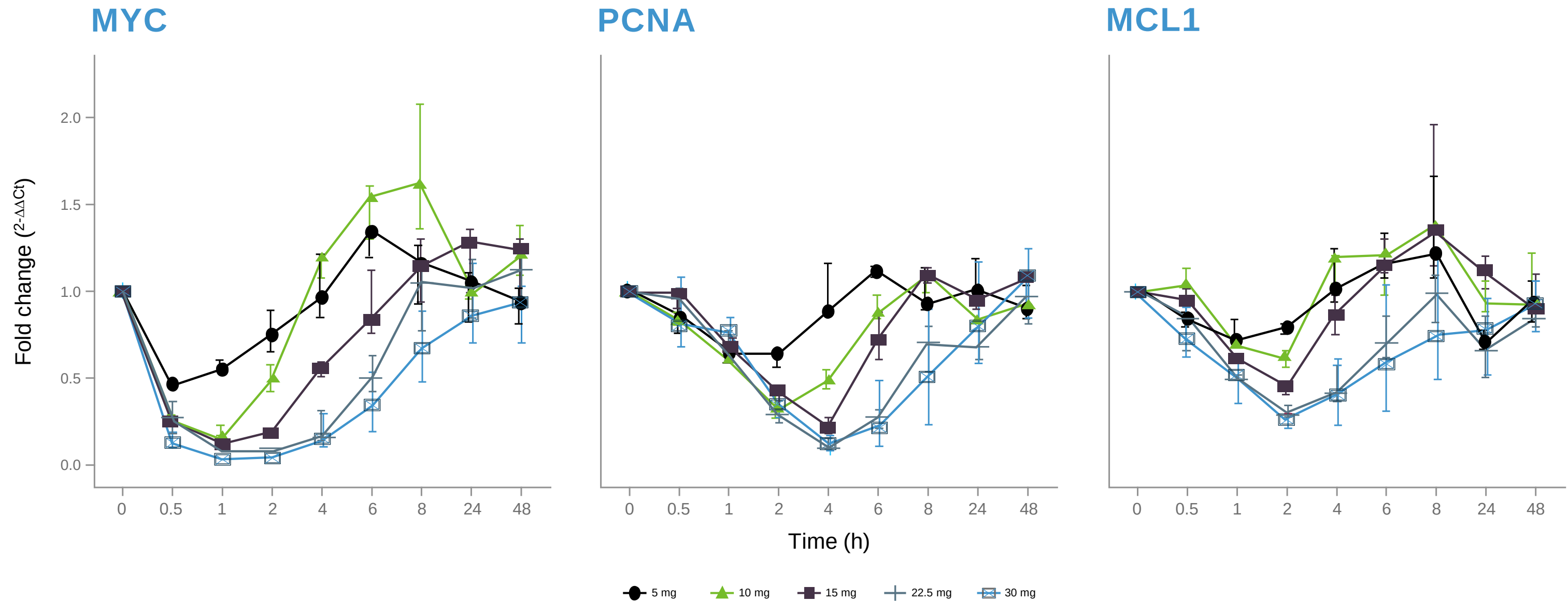
Completed

- Once weekly IV; 30-min infusion
- 21-day cycles
- No biomarker selection in patients with AML

VIP152 Pharmacodynamic Activity in Patient Samples

PD biomarker assessment: mRNA expression in whole blood, cycle 1, day 1

Inhibition of MYC, MCL1 and cell proliferation (PCNA)



Manageable Safety Profile

Neutropenia manageable; Long-term CRs highlight tolerability profile

Adverse Events (>15%)	Grade 1	Grade 2	Grade 3	Grade 4	All (n=31)
Nausea	17 (55)	9 (29)	0 (0)	0 (0)	26 (84)
Vomiting	15 (48)	5 (16)	0 (0)	0 (0)	20 (65)
Anemia	6 (19)	5 (16)	3 (10)	0 (0)	14 (45)
Neutropenia	0 (0)	3 (10)	5 (16)	4 (13)	12 (39)
Fatigue	2 (6)	8 (26)	0 (0)	0 (0)	10 (32)
Diarrhea	8 (26)	1 (3)	0 (0)	0 (0)	9 (29)
Constipation	4 (13)	2 (6)	0 (0)	0 (0)	6 (19)
Thrombocytopenia	4 (13)	2 (6)	0 (0)	0 (0)	6 (19)
Abdominal pain	0 (0)	2 (6)	3 (10)	0 (0)	5 (16)
Anxiety	4 (13)	1 (3)	0 (0)	0 (0)	5 (16)
Fever	4 (13)	0 (0)	1 (3)	0 (0)	5 (16)

No patients
withdrew
due to toxicity

Early Signs of Monotherapy Efficacy in Phase 1 with VIP152

Dose escalation trial (solid tumors and NHL)

- 31 patients, ≥ 3 prior systemic chemotherapies in 97% of patients
- No biomarker selection

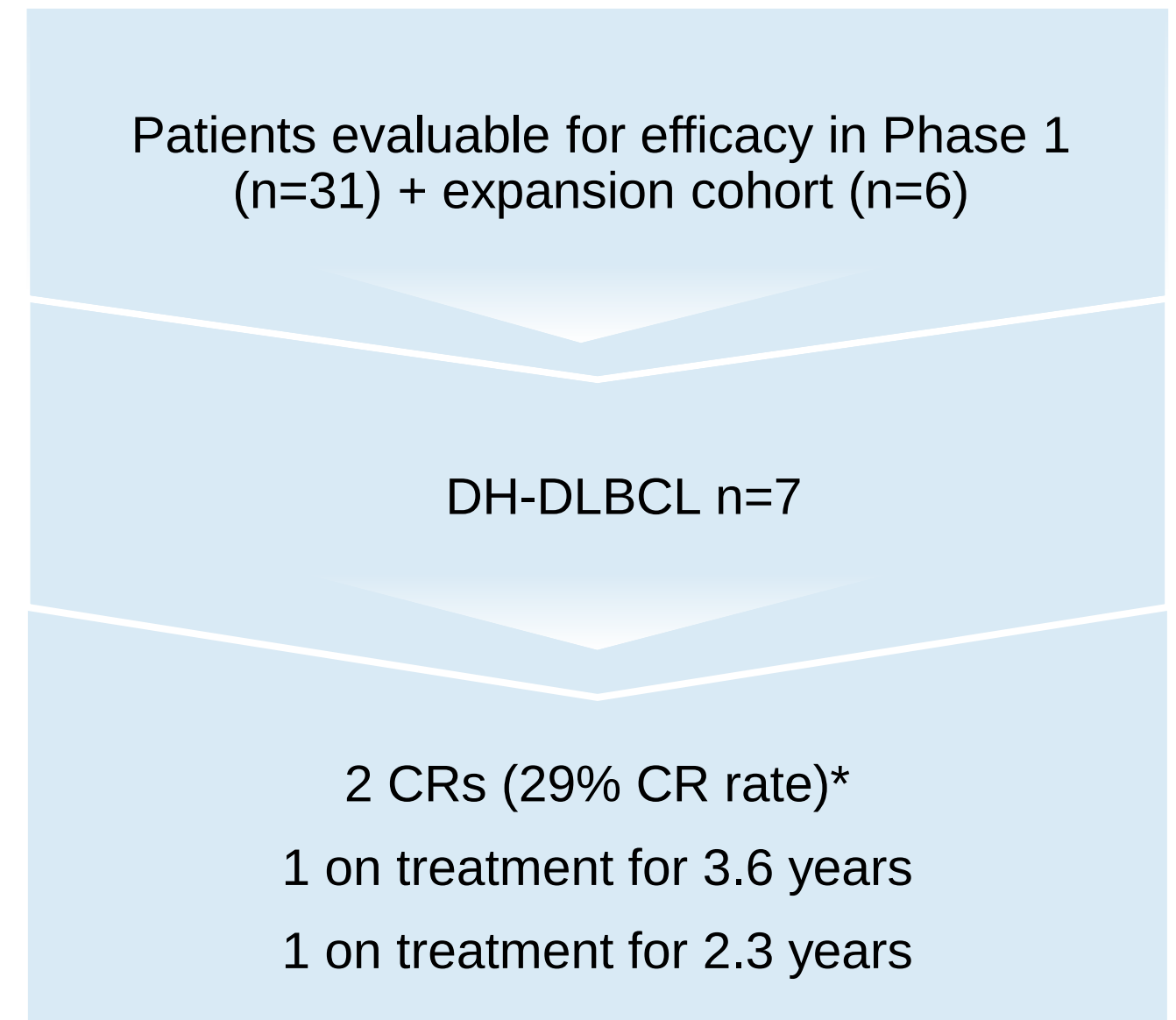
Early clinical signs of efficacy in DH-DLBCL

- 1 patient with DH-DLBCL in dose escalation achieved a PET-negative CR*
- DH-DLBCL patients have MYC rearrangements and either BCL2 or BCL6 rearrangements

Expansion cohort ongoing in DH-DLBCL

- 1/6 patients in the expansion cohort achieved a PET-negative CR*

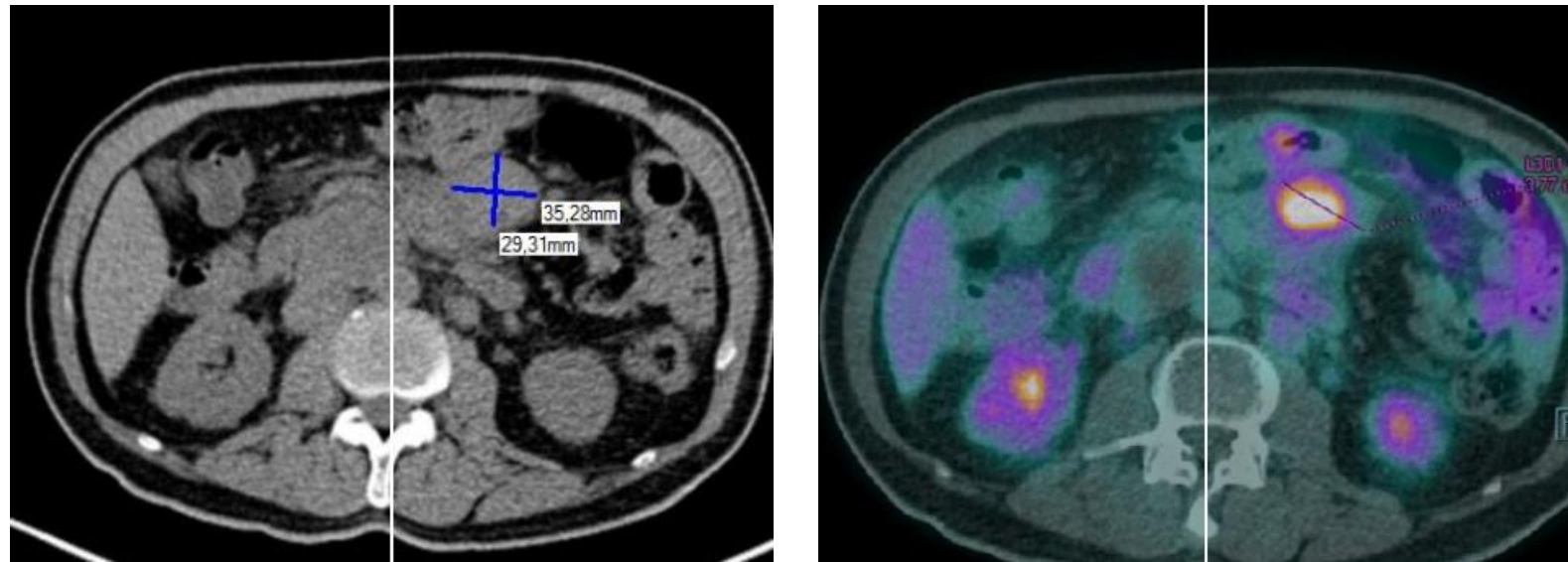
Disease control observed in heavily pretreated solid tumor patients (1 pancreatic cancer and 1 salivary gland cancer pt)



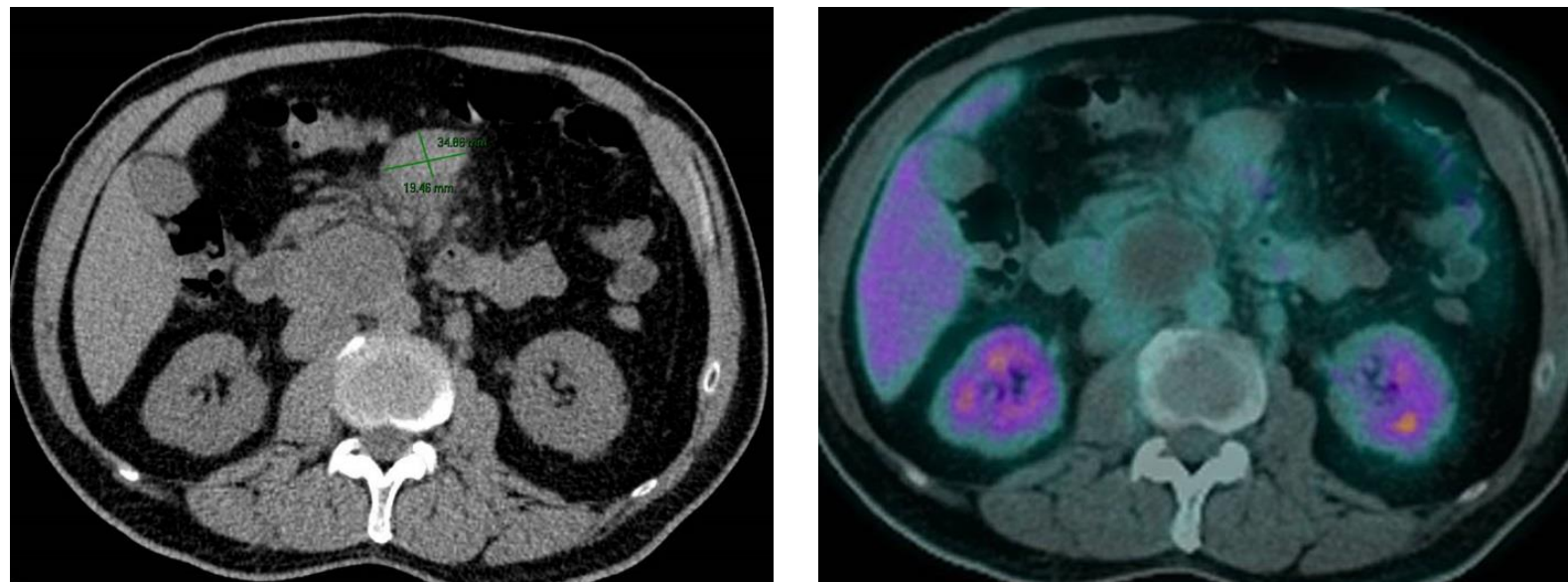
*Per investigator assessment

Complete Metabolic Response Observed in Patient with Treatment-refractory Double-hit DLBCL

Baseline PET/CT



PET/CT cycle 10



IHC, immunohistochemistry; PET / CT, positron emission tomography/computed tomography; TTP, time to progression

Treatment summary

- GCB molecular subtype
- *BCL2* and *MYC* rearrangement positive
- BCL2, BCL6, CD10, CD20 IHC positive; MYC IHC >80% positive
- Foundation Medicine FoundationOne®Heme panel sequencing: *IGH-BCL2* fusion, mutated *CREBBP*, *EZH2*, *PCLO*, *TP53*

Prior Therapies

1. R-EPOCH with partial response (153 days)
2. R-DHAP with progressive disease
3. Palliative radiotherapy 30 Gy to abdomen; best response of progressive disease

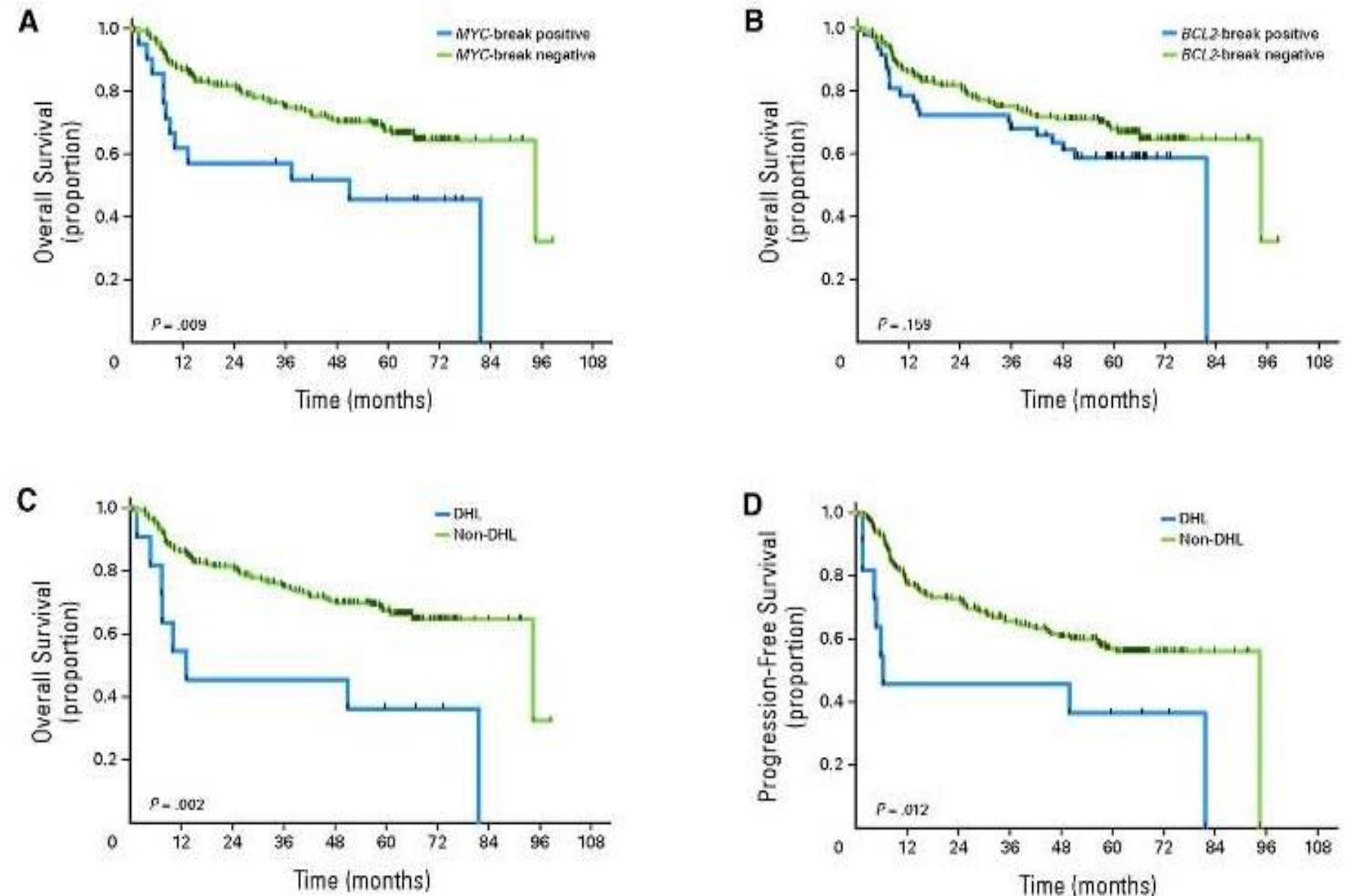
Poor Prognosis in Double-hit Lymphoma

Double-hit (DH)-DLBCL

- Activation of MYC and BCL2/BCL6 genes
- Rearrangements
- Overexpression
- 25% of r/r-DLBCL¹
 - Median PFS 11 months²
 - Median OS 22 months²

R-CHOP in unselected DLBCL pts:
>80% reach a PFS of 6-year⁽³⁾

1. Tumati et al Int J Radiation Oncol Biol Phys 2018;100:1126-32
2. Petrich et al Blood 2014;124:2354-61
3. Pfreundschuh et al Lancet Oncol 2011;12:1013-22



Overall survival (OS) and progression-free survival (PFS) after treatment with rituximab, cyclophosphamide, vincristine, doxorubicin, and prednisone in patients with diffuse large B-cell lymphoma (DLBCL) harboring gene breaks in MYC, BCL2, or both. Kaplan-Meier curves of (A) OS in 21 patients with DLBCL who were positive for MYC breaks versus 168 patients with DLBCL who were negative for MYC breaks show this cytogenetic aberration to be significantly associated with inferior OS ($P = .009$). Kaplan-Meier curves of (B) OS in 47 patients with DLBCL who were positive for BCL2 breaks versus 144 patients with DLBCL who were negative for BCL2 breaks show no significant association with OS ($P = .159$). Kaplan-Meier curves of OS (C) and PFS (D) in 11 patients with double-hit lymphoma (DHL) versus 180 patients with non-DHL DLBCL show that combined breaks in MYC and BCL2 are significantly associated with inferior OS ($P = .002$) and PFS ($P = .012$).

Published in: Green et al JCO 2012;30: 3460-67 Copyright © 2012 by American Society of Clinical Oncology

Potential Indications

MYC and MCL1 overexpression is a hallmark of multiple aggressive, resistant tumors representing a wide-ranging unmet medical need

B-cell Lymphoma MYC dependent (DH-DLBCL, Transformed FL, RS, blastoid MCL)

- Broad sensitivity to VIP152 across NHL cell panel & clinical activity in DH-DLBCL
- Opportunity for significantly improving responses by combining BTK (acalabrutinib) or BCL-2 (venetoclax)

Leukemias MCL1 Dependent (CLL, AML, MDS)

- Initial indication double refractory CLL (potential AA); potential front-line with BTK/BCL2 inhibition
- Potential combinations (eg, BCL2 or FLT3 inhibitors) in AML

Myeloma highly expresses and is dependent on MCL1 & CDK9 for survival (MM)

- Opportunity for significantly improving responses by combining with SOC

Solid Tumors (ovarian, TNBC, CRPC incl NEPC)

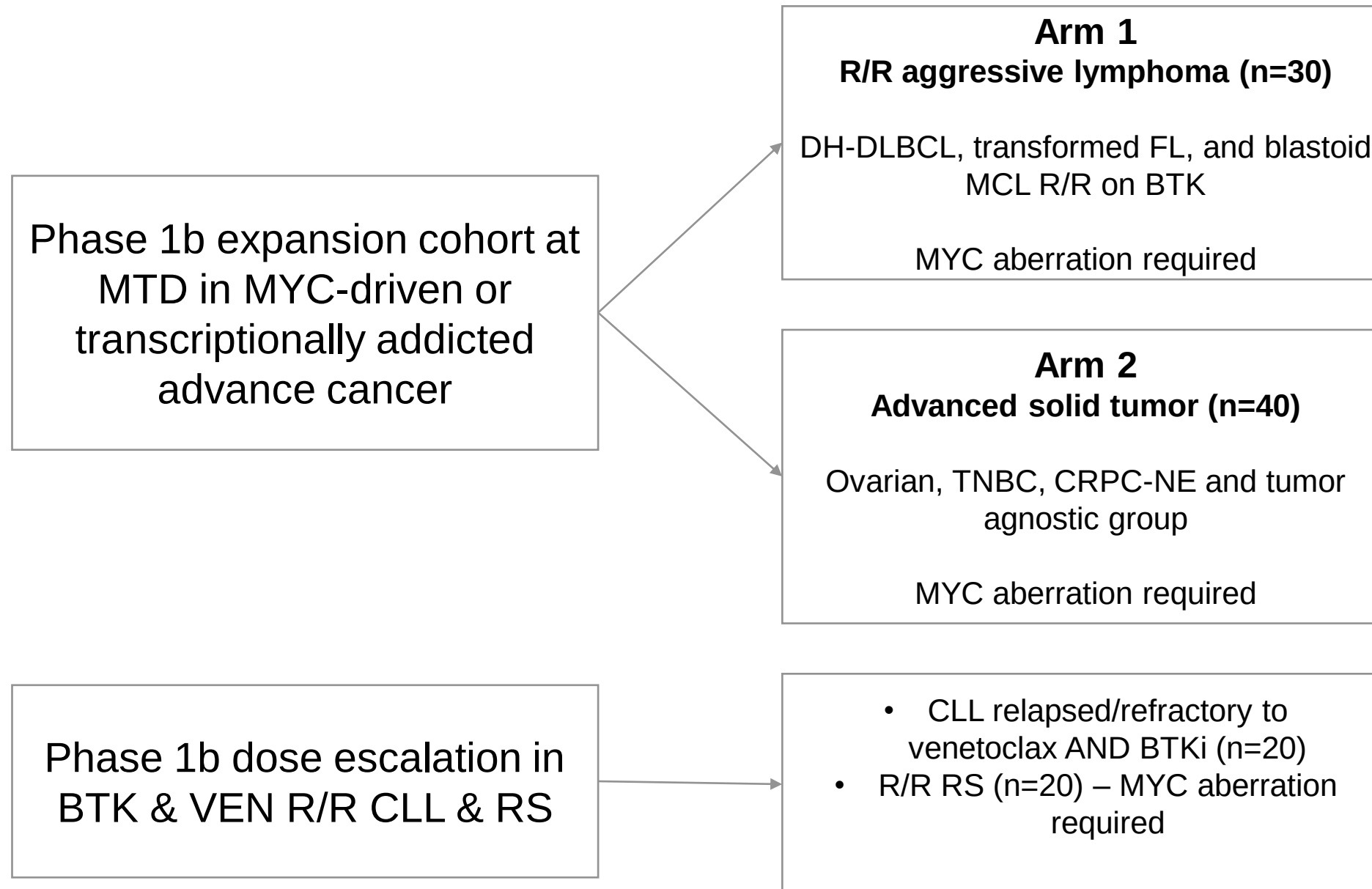
- MYC and MCL1 driven solid tumors
- Opportunity for addressing drug-resistance by combining with SOC

VIP152: Clinical Development Plan

Multiple Accelerated Approval Opportunities

Phase	Design	Population	Countries
1b	Myc driven heme tumors	DH-DLBCL, Transformed FL, RS, blastoid MCL	US
1b	Myc driven solid tumors	Ovarian, TNBC, CRPC [incl NE]	US
1b	Double refractory/relapse (BTK & VEN)	R/R CLL	US

VIP152: Two Phase 1b study designs



- ❖ Arms 1 and 2: FoundationOne or locally confirmed MYC overexpression/translocation to enroll
- ❖ Each group within each arm will be evaluated separately for safety and efficacy
- ❖ May move forward to Phase 2 if ORR (investigator –assessed) 20%-30% for specific disease

Summary: PTEFb Portfolio

PTEFb Portfolio

DIFFERENTIATED
PTEFb INHIBITORS
WITH BROAD
CLINICAL POTENTIAL

ROBUST
PRECLINICAL
IN VIVO AND IN
VITRO DATA

CLEAR
DEVELOPMENT
PATHS IN HIGH
UNMET MEDICAL
NEEDS

EARLY SIGNS
OF SINGLE-
AGENT CLINICAL
EFFICACY

FAVORABLE
PHARMACOLOGY AND
PHARMACODYNAMIC
PROFILE

SIGNIFICANT
COMMERCIAL
POTENTIAL ACROSS
INDICATIONS

IP PROTECTION
UNTIL 2033
(POTENTIAL FOR
EXTENSION)

BIOCONJUGATION PLATFORM

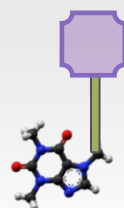
VIP236 (SMDC)
VIP943 (CD123)
VIP924 (CXCR5)

Vincerx's Proprietary Bioconjugate Platforms – Shaping the Future

Turning 10 years of Bayer discovery know-how into break-through treatment modalities

SMALL MOLECULE DRUG CONJUGATE

SMDC

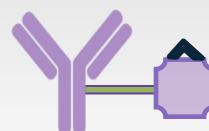


- Conjugate design for highly aggressive advanced diseases
- Targets cell surface cancer markers
- Drug release in tumor stroma
- Tailored warhead design

Preclinical IND in ~1 year

NEXT GEN ADC PLATFORM

ADC



- 2 candidates for heme malignancies
- Proprietary linker-payload technology
- Preferential activation in tumors increase therapeutic window
- Extension to solid tumors for best-in-class treatment modalities

IL3RA ADC: Preclinical
CXCR5 ADC: Preclinical

DISCOVERY PROGRAMS

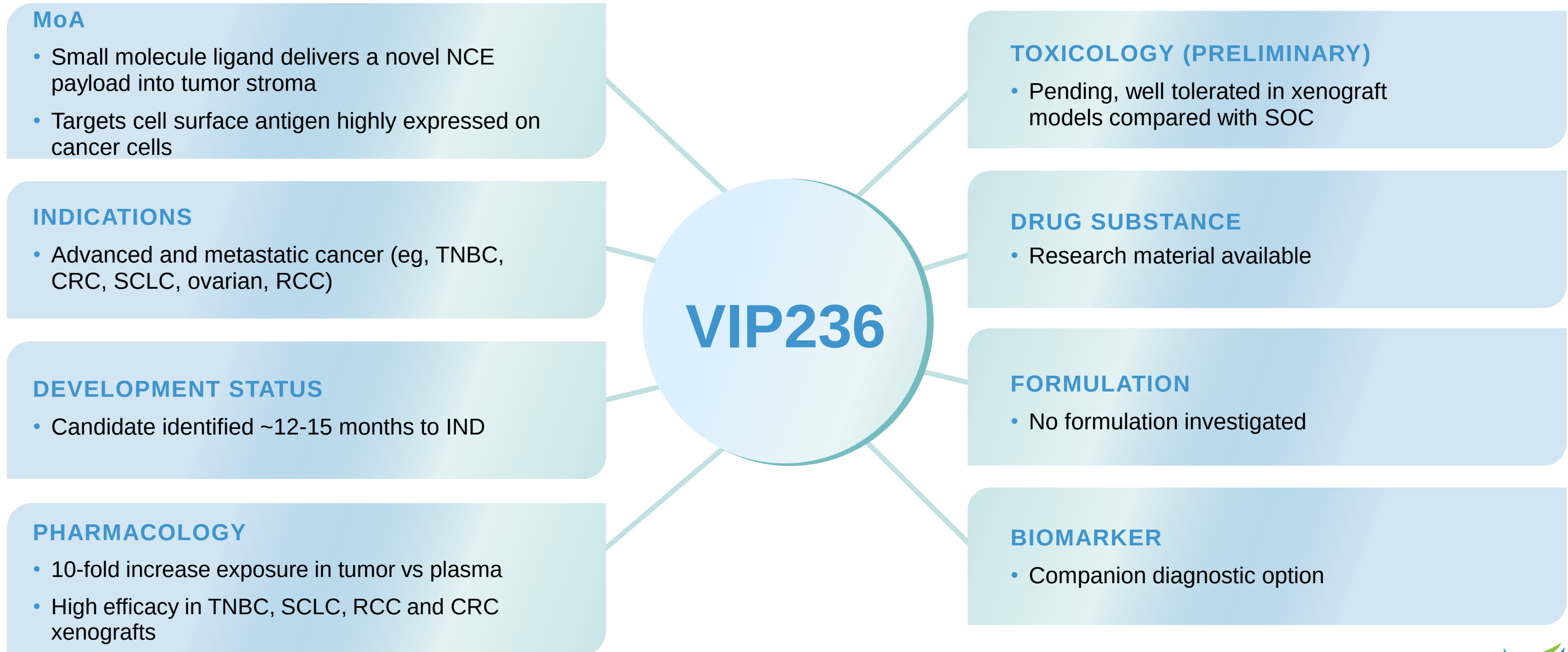
- New antibodies with KSPi linker-payload technologies
- Multiple combinations of modular platform components

Discovery stage

SMALL MOLECULE DRUG CONJUGATE (SMDC)

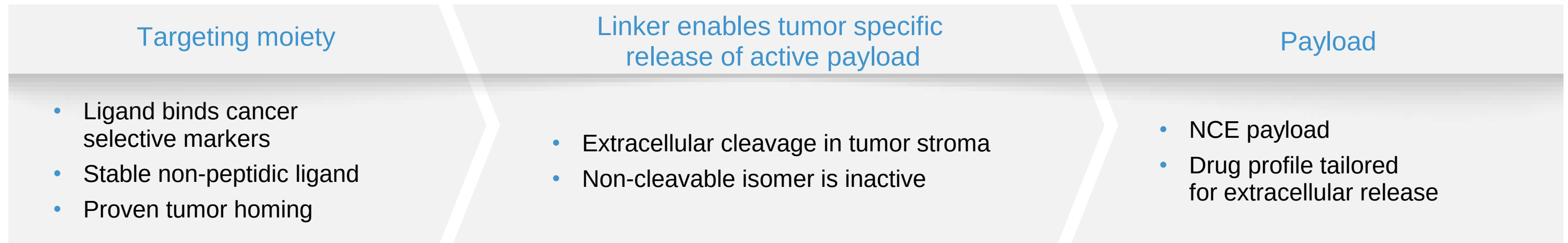
VIP236

Key Features of VIP236, Cancer Cell Surface Targeting SMDC



SMDC Dual Targeting Rationale

Tumor Stroma Activated Conjugate



Differentiated profile

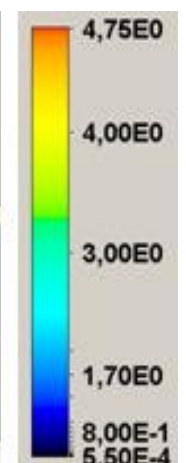
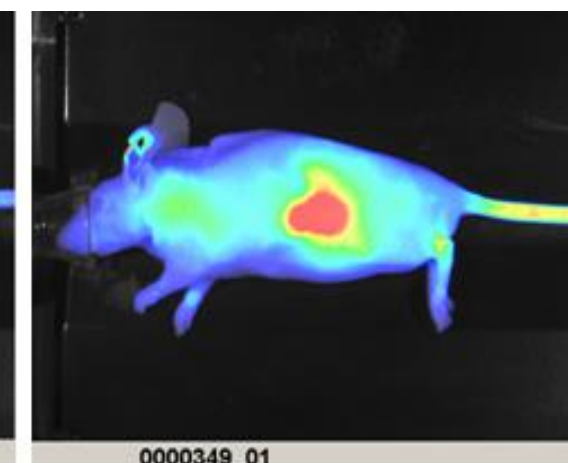
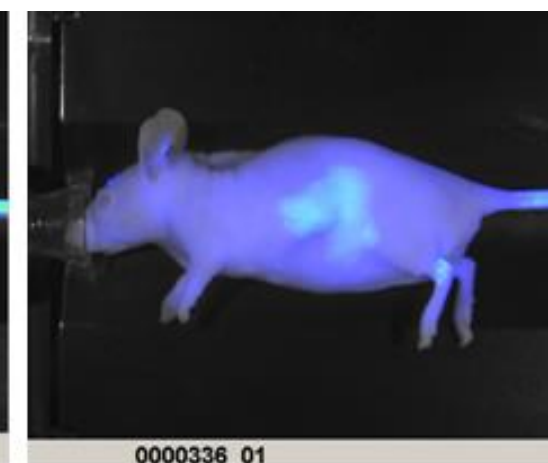
- Target: Cancer cell-surface marker
- Linker: Cleaved by protease in tumor stroma
- Payload: NCE with an improved profile
- In vivo proof of concept in multiple solid tumor models (colon, breast, SCLC, RC)
- Well tolerated after repeated dosing

Imaging shows efficient targeting of tumor ligands

Unconjugated dye IR800

Neg ctrl ligand-IR800

Cancer specific ligand-IR800



NEXT GEN ADC PLATFORM

VIP943
VIP924

Vincerx's Next Generation ADC Technology Solutions

Problems of ADCs

NextGen Design Features ¹

Impact/Benefits

High-potency payloads have narrow therapeutic index

KSP inhibitor is a novel payload class in ADCs

Low/no toxicity in non-dividing cells, no neurotoxicity
High potency and **novel MoA**
Flexibility, compatible with different linker designs

Off-target toxicities due to leaking and unspecific cleavage of highly toxic, cell-permeable toxophores

Stable linker specifically cleaved by legumain, a tumor associated protease
Impermeable payload — Cell Trapper™ attached to KSPi to reduce membrane permeability

Unique cleavage sequence post Asn (no unspecific cleavage)
Second level of tumor targeting via specific ADC activation
Safety: No unspecific uptake of released payload in healthy cells detected
Efficacy: High and long-lasting tumor accumulation

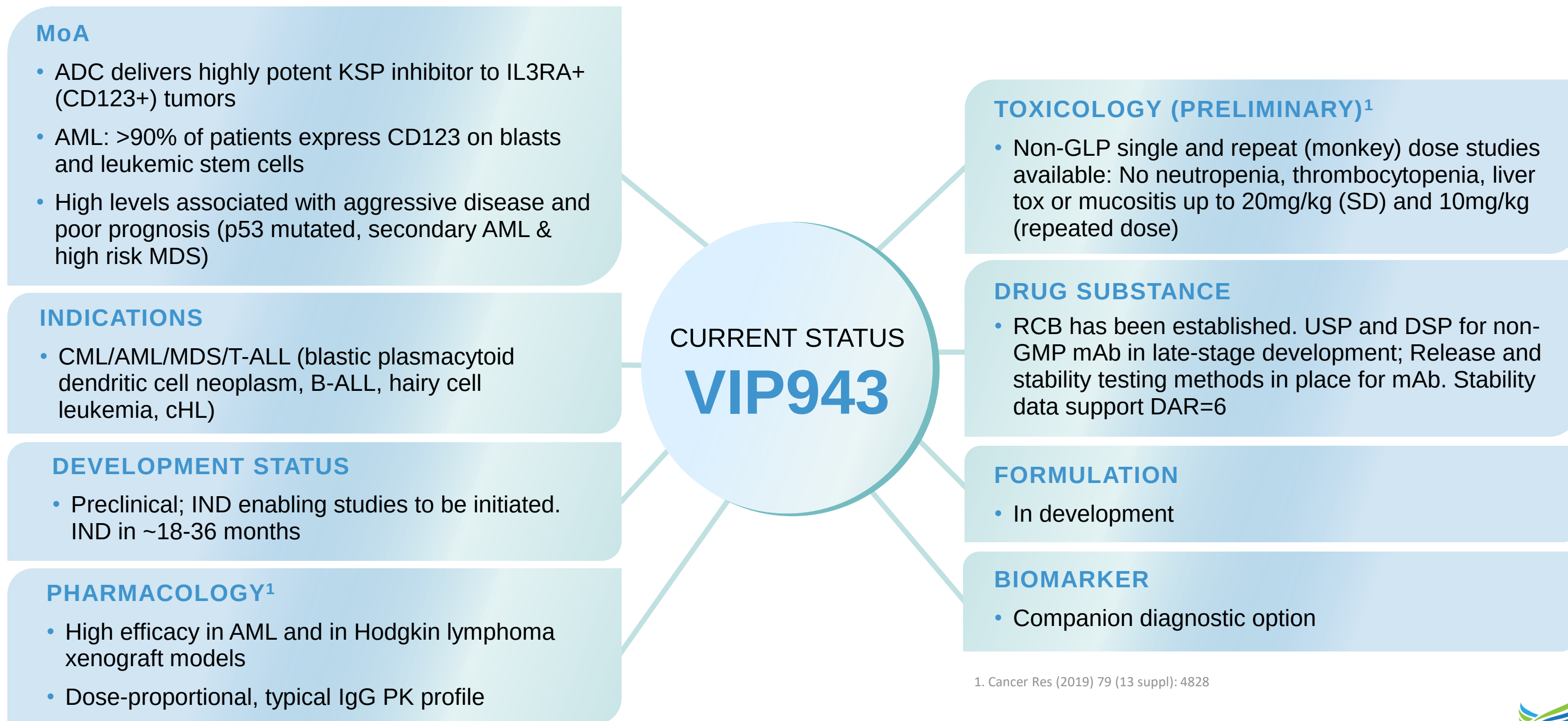
Highly lipophilic payloads cause aggregation and unspecific pinocytosis of ADCs

KSPi payload with Cell Trapper™ is hydrophilic and does not cause aggregation

Safety: No side effects associated with aggregation detected
Efficacy: Allows for DAR of 6 without affecting PK
CMC: Less risk for reduced shelf live & particle formation

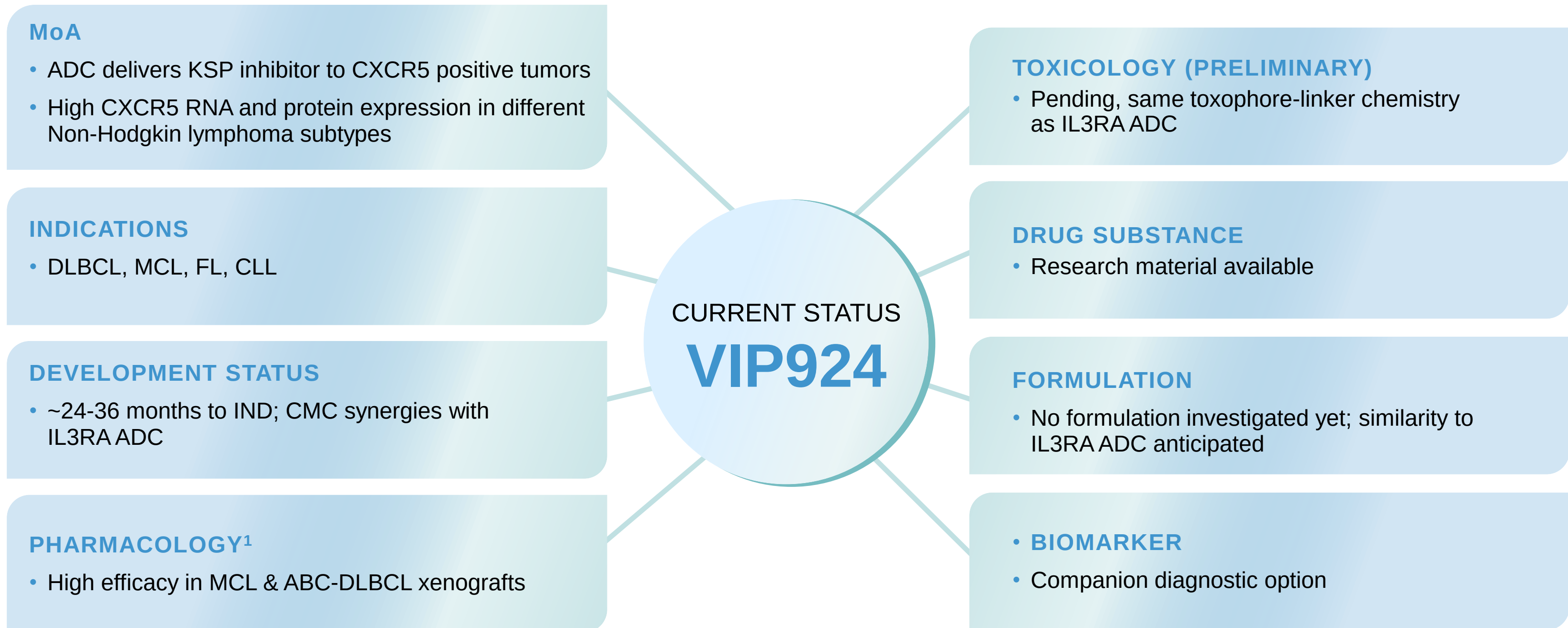
1. <https://dx.doi.org/10.1021/asc.bioconjchem.0c00357>

Key Features of VIP943, an IL3RA-KSPi ADC



1. Cancer Res (2019) 79 (13 suppl): 4828

Key Features of VIP924, a CXCR5-KSPi ADC



1. Cancer Res (2019) 79 (13 suppl): 4825

Expected Upcoming Milestones

VIP152

- Q1 2021 – Begin Phase 1b study for Myc driven hematologic malignancies
- Q1 2021 – Begin Phase 1b study for Myc driven solid tumors
- Q1 2021 – Begin Phase 1b study for R/R D CLL
- H1 2022– Initial clinical data from Phase 1b studies

VIP236

- H1 2022 – Begin FIH study for solid tumors

VIP943

- H2 2022 to H1 2024 – Begin FIH study for CD123+ hematologic malignancies

VIP924

- H2 2023 to H2 2024 – Begin FIH study for CXCR5+ hematologic malignancies

Vincerx Summary



A strong management team with a proven track record of successes

- Publicly traded company (PCYC): Co-development w JNJ, \$1B; Sale to Abbvie, \$21B
- Private company (Acerta) founded company on preclinical asset and took it to approval and sale of company: M&A \$7B, AZN
- >20 years of experience in CDK9 space
- >10 years of ADC development experience from discovery to clinical development

De-risked clinical pipeline, multiple shots on goal

- Clinical stage asset with clinical POC – single agent remissions (>2y) in a very aggressive disease (DH-DLBCL)
- Accelerated Approval opportunities as a potential best-in-class monotherapy – strong commercial potential in oncology
- Safety profile will support future combination studies

Innovative, next-generation bioconjugation platform

- Modular technology designed to address specific challenges of current ADCs in the clinic
- KSPi-ADC safety profile has been de-risked in cyno tox studies with potential first-in-class & best-in-class opportunity
- SMDC is ready for IND after GLP tox