Vincerx PHARMA

CORPORATE OVERVIEW

July 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

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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

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Management Team with Proven Track Record

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CEO

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Vincerx Pipeline

	PROGRAM	MECHANISM (*Potential*)	DISCOVERY | CMC | PHASE 1 | Phase 2	INDICATIONS	Upcoming Milestones
PTEFb	VIP152	CDK9 inhibitor (IV) *Best in Class*		Lymphomas (e.g., DHL, MCL, transformed FL) Solid tumors (e.g., Ovarian, TNBC, NEPC, tumor agnostic mt-MYC) Leukemias (e.g., CLL, RS)	Multiple Phase 2 studies 2H 2022
PTEFb	VIP217	CDK9 inhibitor (PO) *Best in Class*		TRANSCRIPTIONALLY ADDICTED TUMORS	IND 2024
Bioconjugation	VIP236	$\alpha_v\beta_3$-CPT SMDC *First in Class*		MULTIPLE SOLID TUMORS	IND 1H 2022
Bioconjugation	VIP943	Anti-CD123 + KSPi ADC *Best in Class*		LEUKEMIAS AND MDS	IND 2H 2023
Bioconjugation	VIP924	Anti-CXCR5 + KSPi ADC *Best in Class*		B-CELL MALIGNANCIES	IND 1H 2024
Bioconjugation	Vincerx Platform	TBD		TBD	TBD

ADC = antibody-drug conjugate; CLL = chronic lymphocytic leukemia; CRPC-NE = castration-resistant prostate cancer – neuroendocrine; DHL = double-hit lymphoma; FL = follicular lymphoma; IND = Investigational New Drug Application; IV = intravenous; MCL = mantle cell lymphoma; MDS = myelodysplastic syndromes; NHL = nonHodgkin lymphoma; PO = oral; PTEFb = positive transcription elongation factor b; RS = Richter syndrome, SMDC= small molecule drug conjugate; TBD = to be determined; TNBC = triple negative breast cancer

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PTEFb PROGRAM

VIP152 IV (Phase 1)
VIP217 Oral (Discovery)

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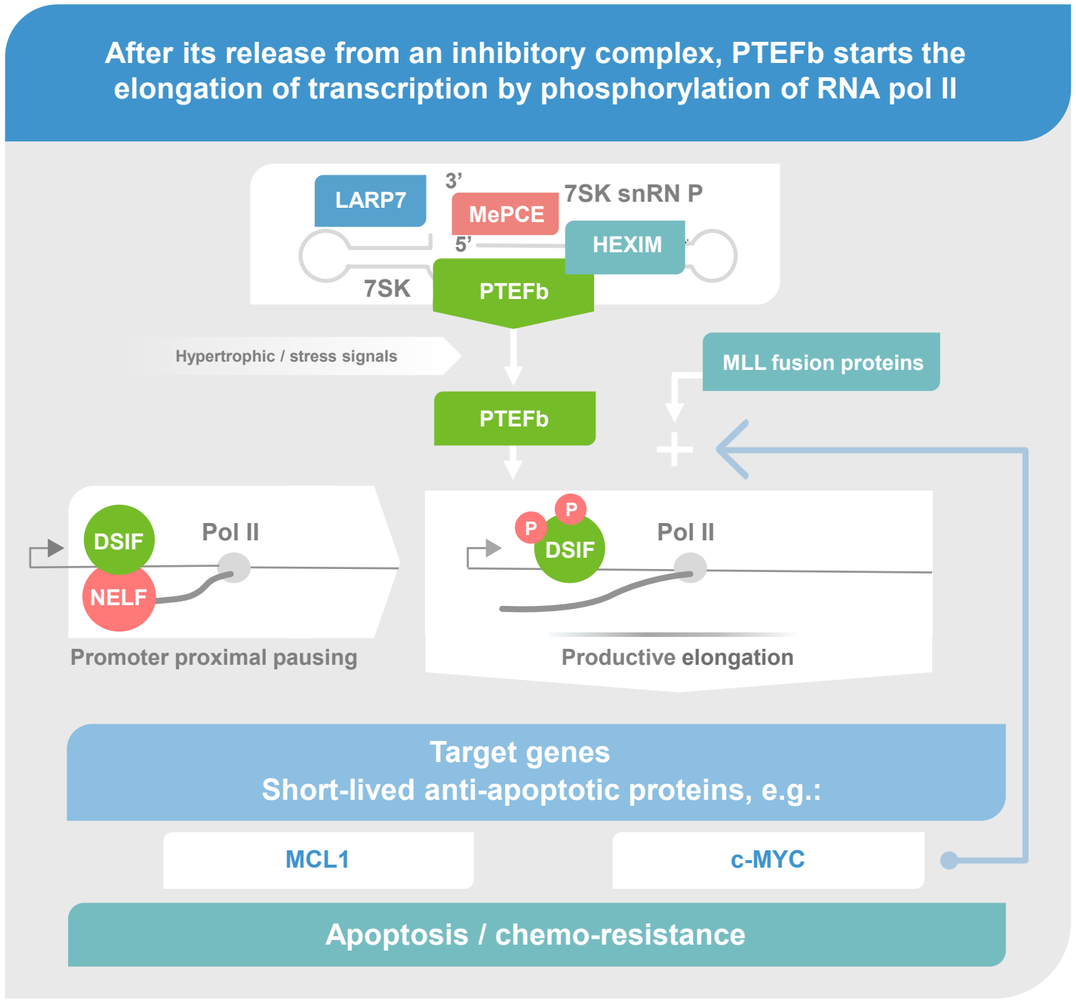
PTEFb: A Novel Target for Oncology



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

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VIP152 is the Most Selective CDK9 Inhibitor in the Clinic

Programs	**VIP152** Vincerx	**Atuveciclib** Vincerx	**AZD4573** AZ	**KB-0742** Kronos	**Dinaciclib** Merck	**Fadraciclib** Cyclacel	**Alvocidib (Flavopiridol)** Tolero	**Voruciclib** MEI Pharma
Selectivity	CDK9	CDK9	CDK1/9	CDK9	CDK1/2/5/9	CDK2/3/5/9	Pan CDK	Pan CDK
Development Stage	P1	-	P1	P1	P3 Mono P2 Combo	P1	P2	P1 mono and combo BCL2
Type of tumor	Hematologic & Solid tumors	-	Hematologic	Solid tumors	CLL stopped Solid combo with IO	AML, CLL, ALL Solid tumors	AML/MDS Combos	B-cell malignancies and AML
IC_{50} on CDK9	3 nM[1] [ATP]: 0.01 mM 4 nM [ATP]: 2 mM	13 nM[2]	14 nM[4]	6nM[6]	13 nM[3]	26 nM[5]	22nM[6]	1 nM[7]
Half life	4h	2-3h	<3h	-	3h	~1h	2-4h	30h
Route of Admin	IV	Oral	IV	Oral	IV	Oral & IV	IV	Oral

1. Lücking AACR 2017; 2. Lücking *Chem Med Chem* 2017; 3. Wells *Nat Commun* 2020; 4. Cidado *Clin Cancer Res* 2020; 5. Frame *PloS ONE* 2020 6. Day AACR 2021; 7. Dey *Sci Rep* 2017

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VIP152 Highly Selective and Potent CDK9 Inhibitor

Assay	VIP152
IC_{50} CDK9 [nM] low ATP	3
IC_{50} CDK9 [nM] high ATP	4

High potency is independent of [ATP]

Kinase	**Kd** [nM] @ DiscoverRx	**IC_{50}** [nM] @ Millipore
CDK9	1.3	13**
CDK1	n.a.	192
CDK2	710	158
CDK3	540	318
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.

Activity against all non-CDK kinases with <50x higher KDs

Kinase	Activity [nM]
CDK9	1.3
GSK3a	7.4
IRAK1	61

High selectivity over other CDKs, incl CDK2

Favorable non-CDK kinase selectivity profile

* No cyclin co-expression
** Probably lower limit of quantification

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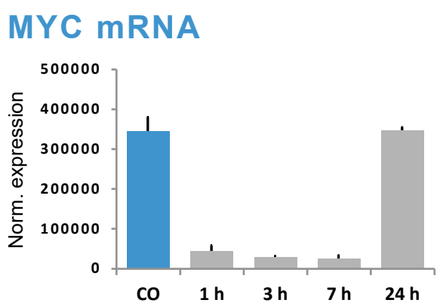
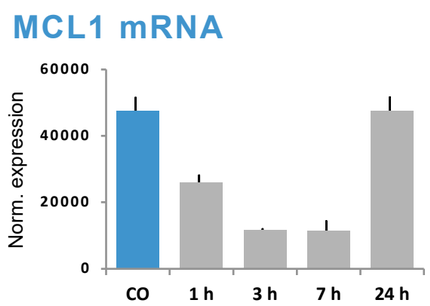
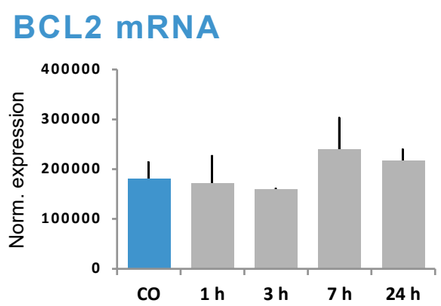
VIP152 MoA Transiently Inhibits the Transcription of MYC and MCL1

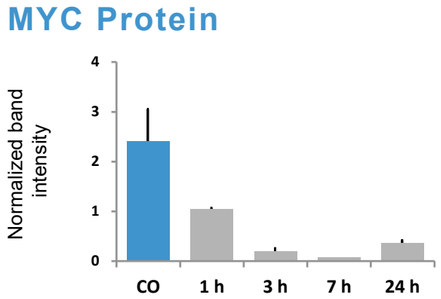
Reduction of MYC & MCL1 mRNA levels



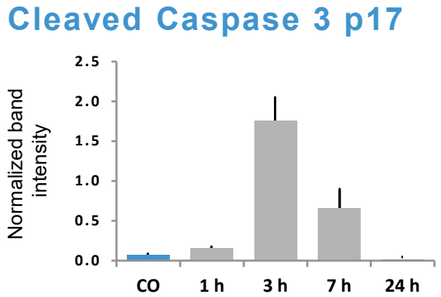


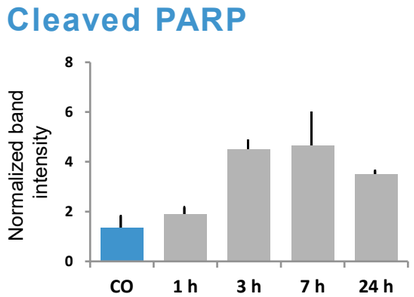


Durable reduction of MYC protein levels



Induction of apoptosis





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

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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

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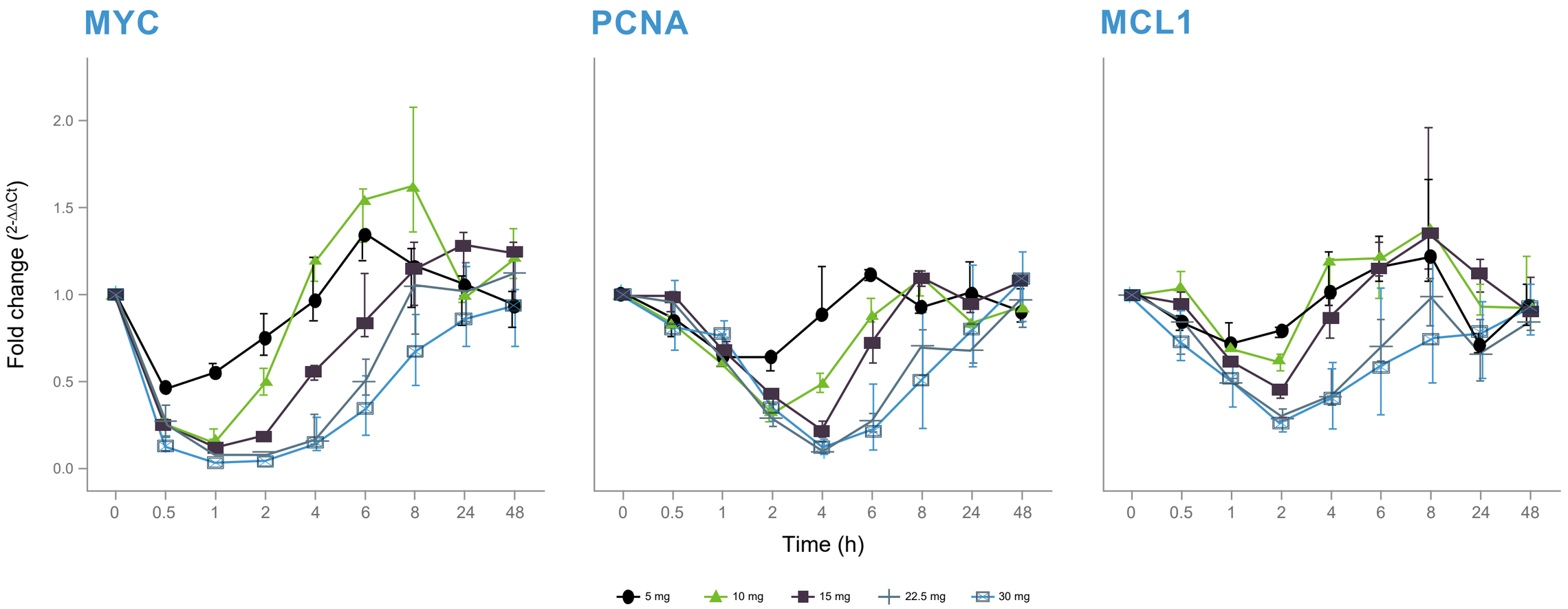
VIP152 Pharmacodynamic Activity in Patient Samples

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

Inhibition of MYC, MCL1 and cell proliferation (PCNA)



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Favorable 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

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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)

Patients evaluable for efficacy in Phase 1 (n=31) + expansion cohort (n=6)

DH-DLBCL n=7

2 CRs (29% CR rate)*

1 on treatment for 3.6 years

1 on treatment for 2.3 years

*Per investigator assessment

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Clinical Activity in Ph1 Dose Escalation with VIP152 – all comers

Background

- 30 subjects with various therapy-refractory solid tumors were treated as part of the dose-escalation. No biomarker was used for selection.
- The treatment was generally well-tolerated with neutropenia as the only Grade 4 toxicity.
- Seven subjects had stable disease, including ovarian, pancreatic, and salivary gland cancers.
- Stable disease was seen across all the dose cohorts.

Duration of Stable Disease by Malignancy Type

Type of malignancy	Dose (mg)	Last dose (cycle)	Months on Tx
OVARIAN	5	C3	1.9
APPENDIX CANCER	10	C5	2.8
NASOPHARYNGEAL	22.5	C3	1.7
PANCREATIC ADENOCARCINOMA	22.5	C3	1.9
CLIVAL CHORDOMA	22.5	C4	2.6
MALIGNANT NEOPLASM OF MAJOR SALIVARY GLAND	22.5	C24	16.8
PANCREATIC ADENOCARCINOMA	30	C14	9.5

Sources: 24Nov2020 Data, ADRS. Listing 14.2 /4; ADCE

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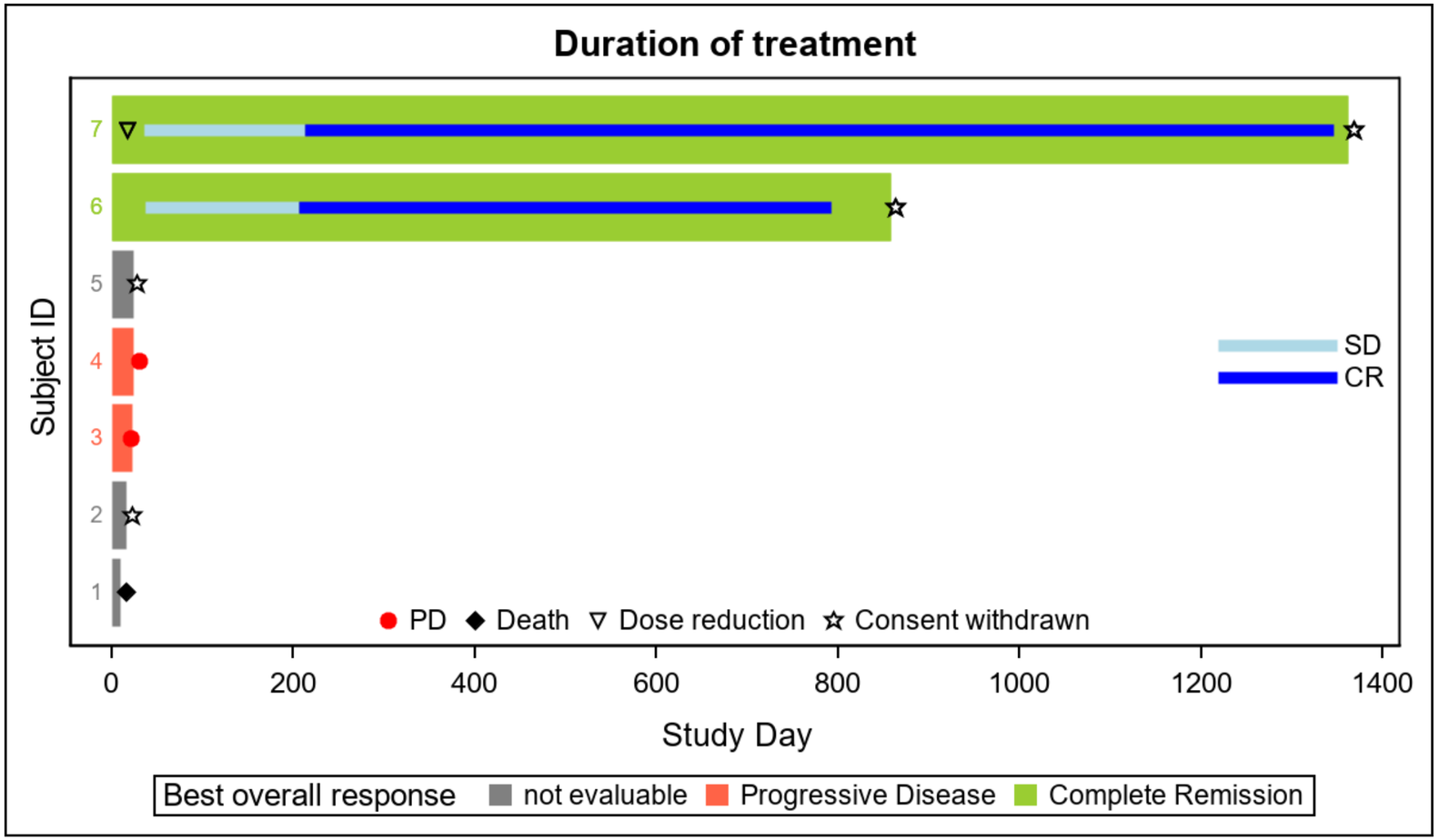
Clinical Efficacy and Long-term DoR in DHL (n=7)



2 CRs (29%) on treatment for:

- 3.7 years
- 2.3 years

Subject 1: Cause of death was clinical disease progression; however, scans were not performed for response criteria determination.
Subjects 2 and 5: Clinical progression and withdrawal by subject.
Subjects 6 and 7: Withdrawal by subject to eliminate hospital visits during COVID conditions

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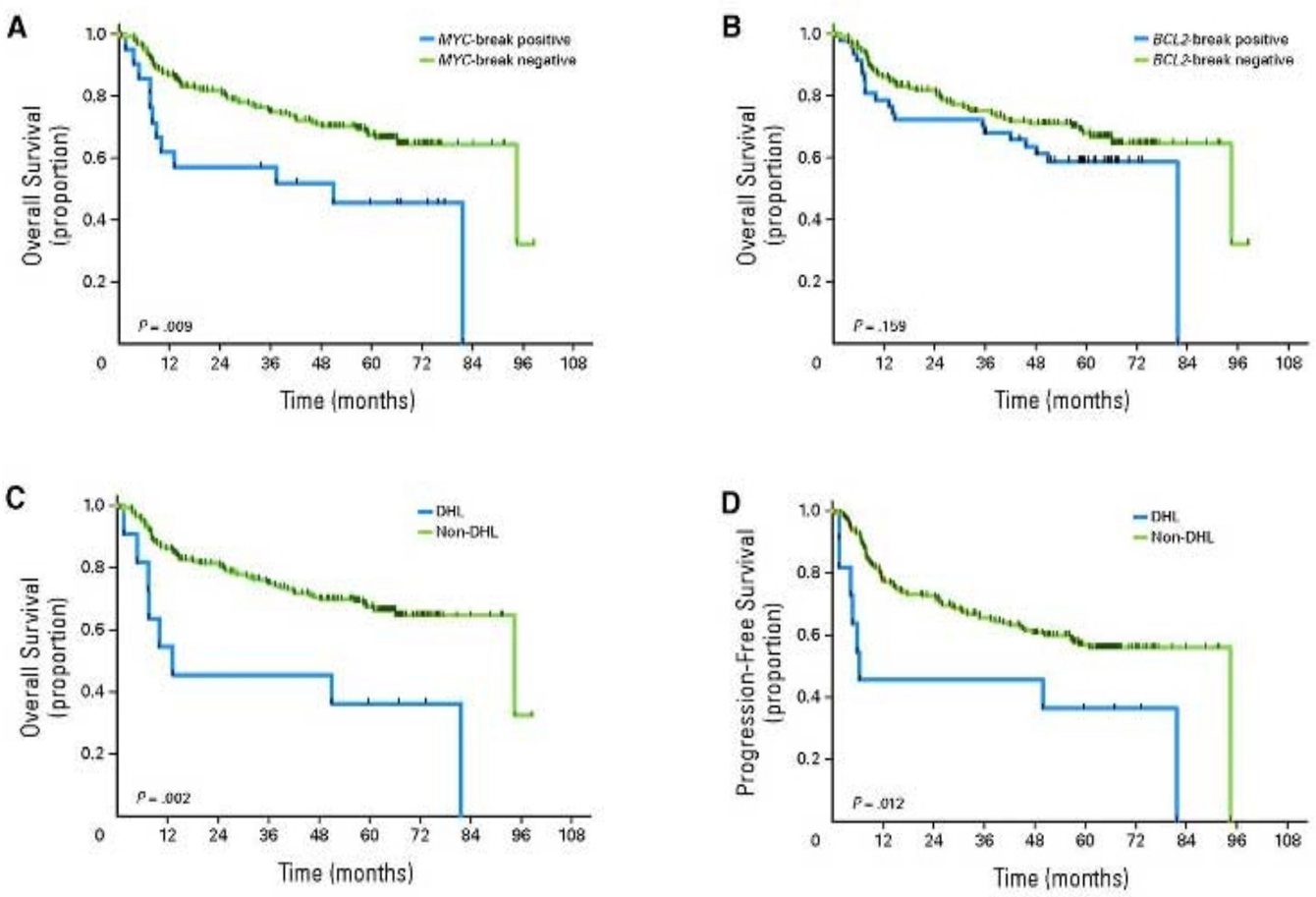
Poor Prognosis in Double-hit Lymphoma

Double-hit (DH)-DLBCL

- Activation of MYC and BCL2/BCL6 genes
  - Rearrangements
  - Overexpression
- 25% of r/r-DLBCL[1]
  - Median PFS 11 months[2]
  - Median OS 22 months[2]

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



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).

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

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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

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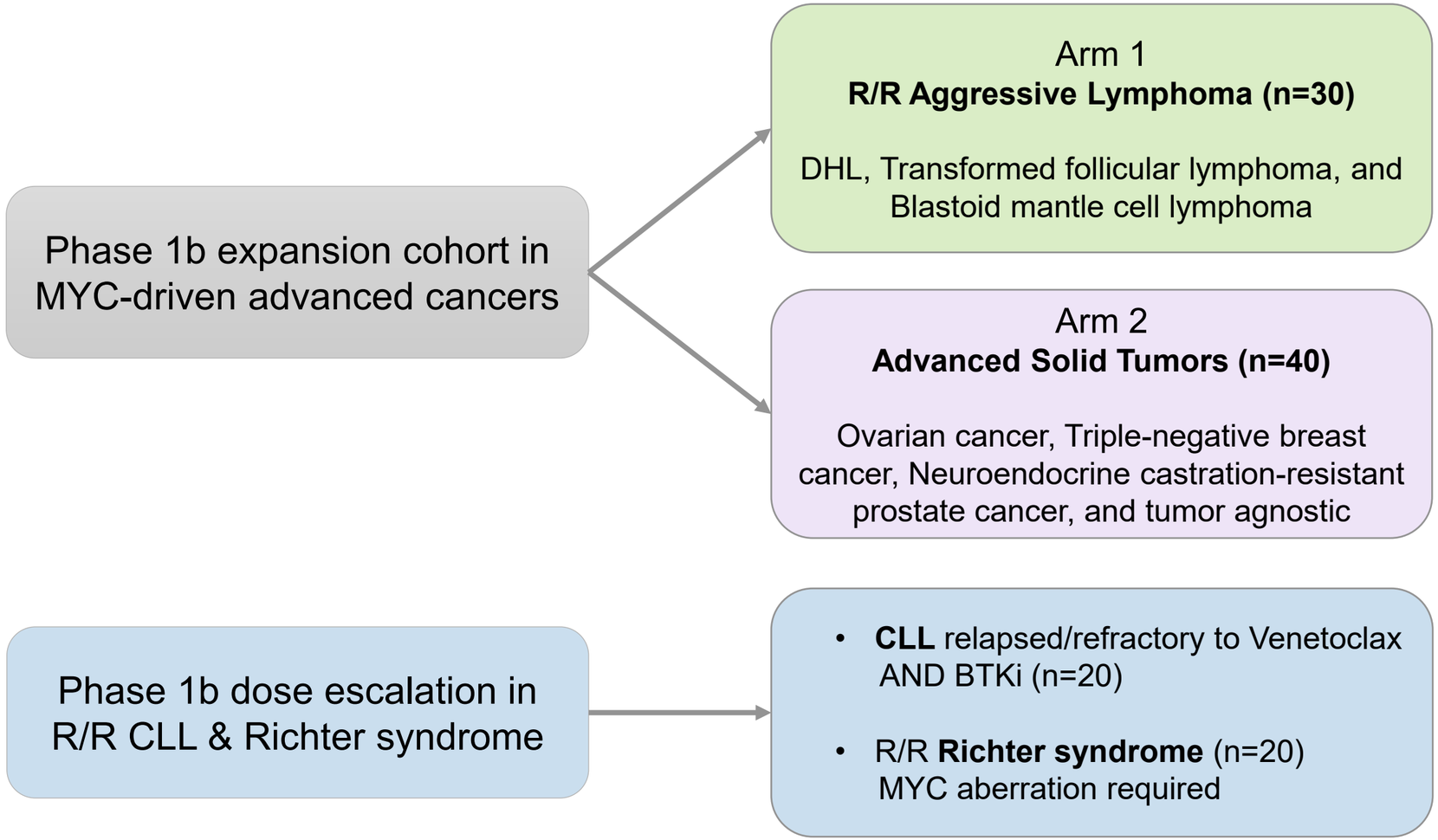
VIP152: Two Phase 1b Study Designs – Multiple Shots on Goal



- 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) is 20%-30% for a specific indication

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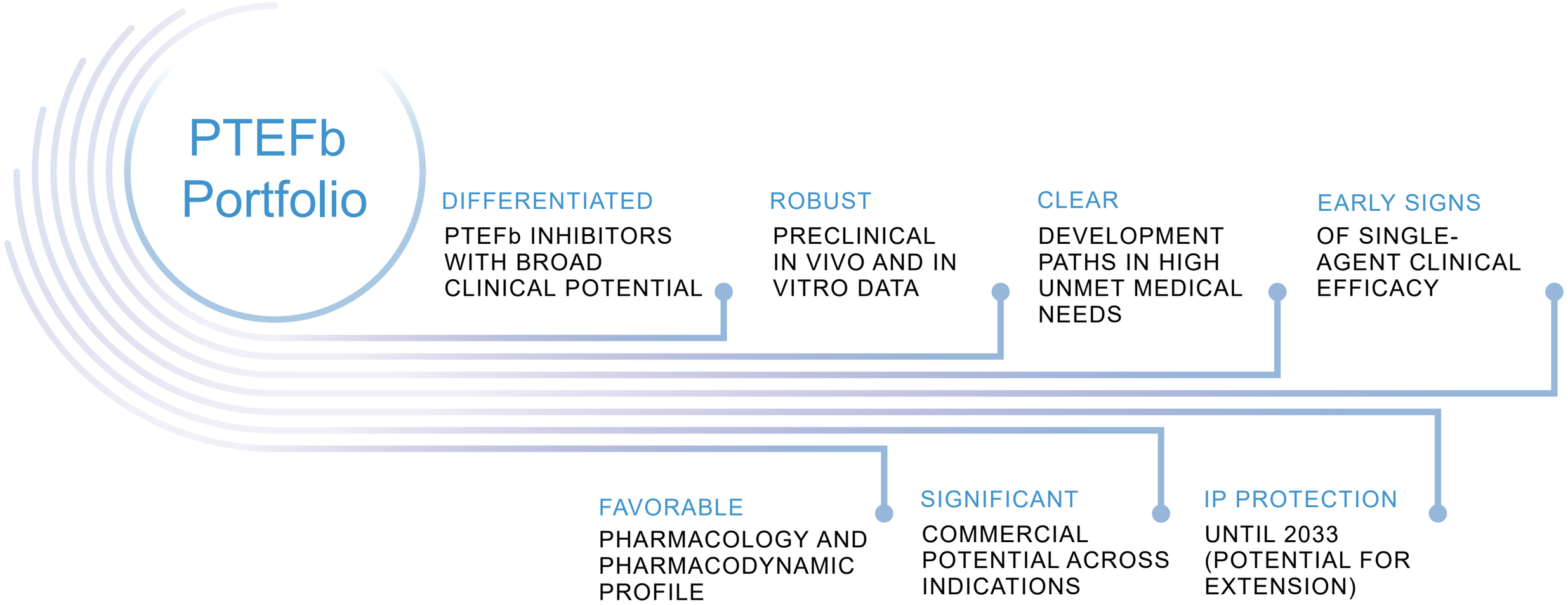
Summary: PTEFb Portfolio



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BIOCONJUGATION PLATFORM

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

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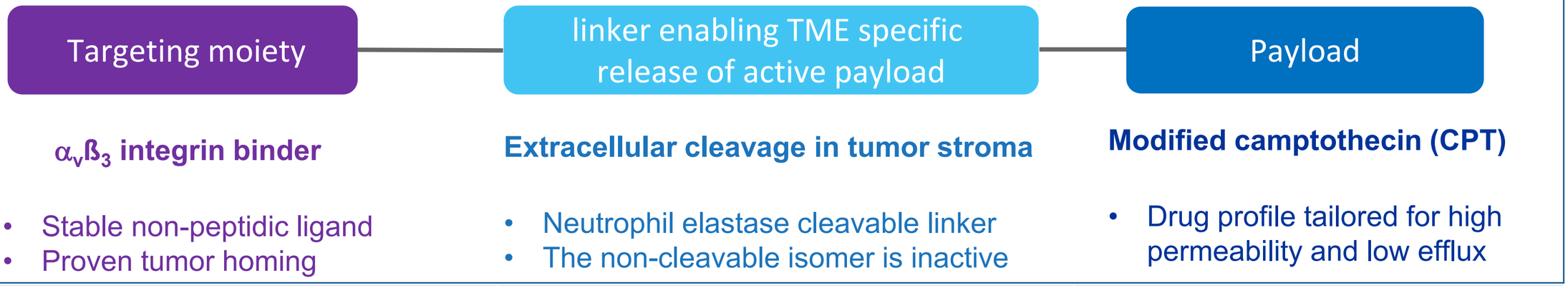
Targeted Small Molecule Drug Conjugate (SMDC) Technology

VIP236 is an SMDC that	• **targets the tumor microenvironment (TME)** • **is activated by the tumor stroma**



αvß3 integrins	**Neutrophil elastase**	**Modified CPT**
Play a critical role in the TME in: • Tumor progression & metastasis • Resistance to cytotoxic therapy • Recruitment of immune/inflammatory cells.	Belongs to a family of proteases that contribute to cancer progression. Its overexpression in the TME is associated with • Tumor evasion • Metastasis	Optimized to increase potency on mitotic cancer cells

The expression levels of $\alpha_v\beta_3$ integrins and neutrophil elastase are associated with aggressive disease in many cancers.

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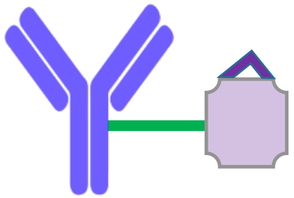
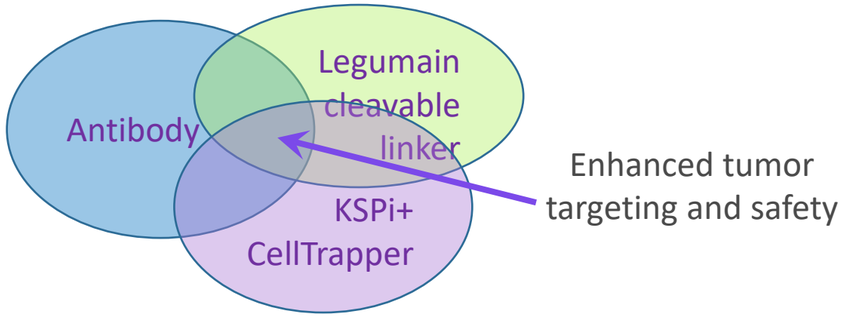
KSPi-Antibody Drug Conjugate Technology
Enhances Therapeutic Potential

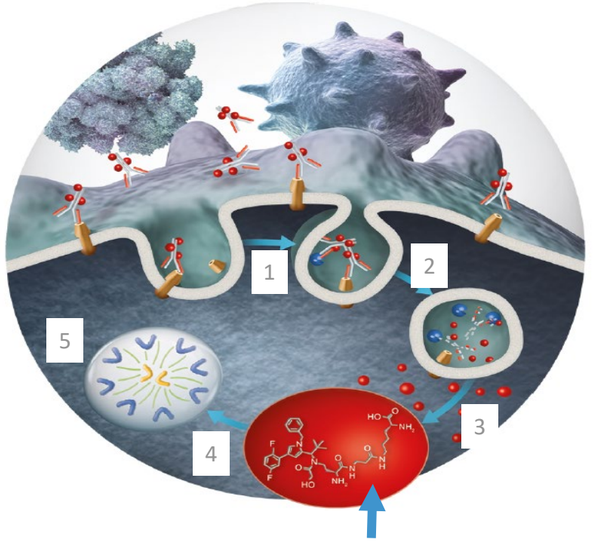


e.g. VIP943, VIP924



ADCs tuned for tumor specific payload release

Components	Features	Advantages
• **Antibody**	Abundant targets	• Tumor selectivity
• **Legumain-cleavable linker**	Cleaved by a very specific lysosomal (low pH) asparaginyl endopeptidase	• Tumor selectivity: Legumain is overexpressed in tumors vs normal and associated with poor prognosis • No non-specific cleavage: Unique cleavage sequence and low pH required • Flexibility to adapt linker to specific clinical applications
• **KSP inhibitor**	A novel, high potency MoA payload specific for dividing cells	• Low/no toxicity in non-dividing cells, no neurotoxicity • Potential to induce immunogenic cell death
• **CellTrapper™**	Reduces payload cell membrane permeability	• Chemical moiety that is part of the KSPi payload • Trapped KSPi payload concentrates in tumor cells • Released payload cannot enter healthy cells



cytotoxic payload with CellTrapper™ moiety enabling tumor accumulation

VIP943 Mechanism of action
anti-IL3Ra KSPi-ADC

1. VIP943 binds to IL3RA on surface of cell and gets internalized
2. Endosome fuses with lysosome: legumain digestion, release of cytotoxic payload containing a cell trapper moiety
3. Cytotoxic payload (KSPi) enters cytoplasm
4. KSPi inhibits spindle apparatus (KSP, Eg5)
5. Mitotic catastrophe

Vincerx PHARMA

Expected Upcoming Milestones

Program	2021	2022	2023	2024
Clinical Programs				
VIP152 CDK9 inhibitor (IV)	Begin Ph1b in Myc Hematology Begin Ph1b in Myc Solid Tumors Begin Ph1b in R/R D CLL	Initial Clinical Data in Ph1b studies Multiple Ph2 DHL MCL post BTK CLL post VEN, BTK Richter's Syndrome Ovarian NEPC MYC dep. solid tumors		
VIP236 SMDC		IND in Solid Tumors		
VIP943 IL3RA			IND in hematologic malignancies	
VIP924 CXCR5			IND in hematologic malignancies	

Vincerx PHARMA

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
- Clinical data 1H2022 or earlier and Ph2s by end of 2022

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 1H2022, ADCs 1H2023