VincerX PHARMA

CORPORATE OVERVIEW

MAY 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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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

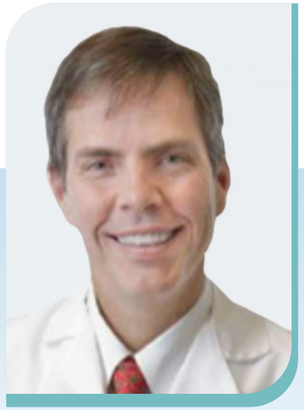
- Cofounder of Acerta Pharma
- Former Sr Director of Clinical Development of Pharmacyclics
- Extensive drug development experience from preclinical stages through NDA submission

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

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Management Team Experience - Proven Track Record

CALQUENCE® (acalabrutinib) 100 mg capsules

$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

2013	2014	2016	2017	2019
Acerta founded	1st patient dosed	AZ acquisition	Accelerated approval in MCL	Approved in CLL

imbruvica® (ibrutinib) 140mg capsules

$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

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Vincerx Pipeline

PTEFb

Bioconjugation

PROGRAM	DISCOVERY	PRECLINICAL	PHASE 1
VIP152	IV administration: 2 Ph 1 trials: solid tumors/NHL; AML		Ph 1 ongoing Promising efficacy signals at tolerated doses in solid tumor/NHL trial
VIP217	Oral follow-on		
VIP236 SMDC	Multiple Solid Tumors		• Targeting cancer surface markers with a novel payload • PoC in multiple murine cancer models
VIP943 ADC	Leukemias and MDS		• ADC delivers KSP inhibitor to IL3RA+ (CD123+) tumors • AML: >90% of patients express CD123 on blasts and leukemic stem cells
VIP924 ADC	B-cell Malignancies		• ADC delivers KSP inhibitor to CXCR5 positive tumors • High CXCR5 RNA and protein expression in various NHL subtypes

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

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PTEFb PROGRAM

VIP152 IV (Phase 1)
VIP217 Oral (Discovery)

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Summary of PTEFb (CDK9) Portfolio – Clinical & Discovery

PTEFb
(CDK9)
Program

MODE OF ACTION

- Highly selective PTEFb inhibitor, rapid depletion of short-lived mRNAs of known oncogenes eg, MYC and MCL1

INDICATIONS

- Solid and hematologic tumors
- Combinations with standard therapies and investigational agents

CLINICAL STATUS[1,2]

- Trial in solid and heme cancers, early signs of efficacy in DH-DLBCL and disease control in solid tumors
- Safety and PK findings support further development

CMC

- Drug substance and drug product are available

IP

- Broad intellectual property protection
- Exclusivity for composition of matter until at least 2033, plus potential extensions

ORAL FOLLOW-ON

- Oral follow-on opportunity in discovery phase

1. Blood (2018) 132 (Supplement 1): 4055.
2. JCO (2018) 36 (15): 2507.

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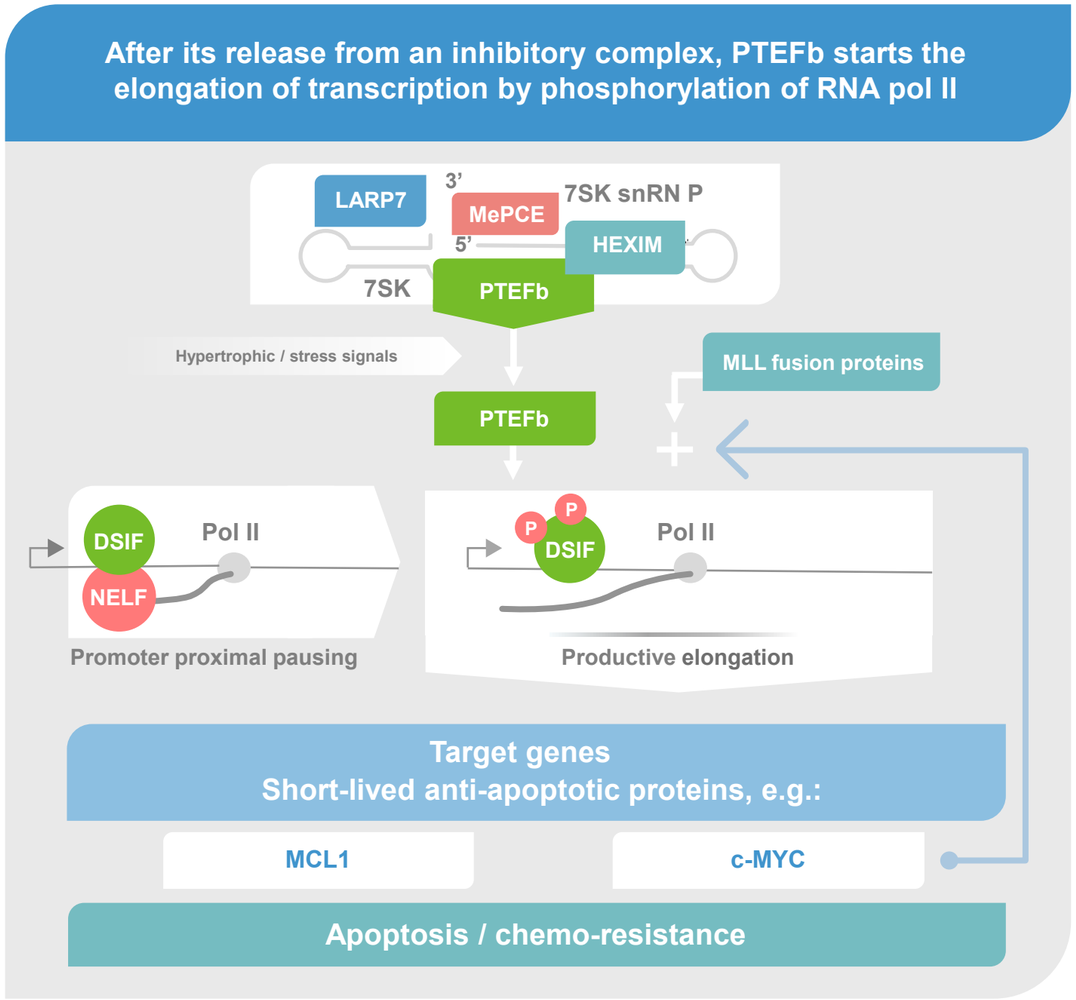
PTEFb: A Novel Target for Oncology



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	Alvocidib (Flavopiridol) Tolero	AZD4573 AZ	Dinaciclib Merck	Fadraciclib Cyclacel	KB-0742 Kronos	Voruciclib MEI Pharma
Selectivity	CDK9	CDK9	Pan CDK	CDK1/9	CDK1/2/5/9	CDK2/3/5/9	CDK9	Pan CDK
Development Stage	P1	-	P2	P1	P3 Mono P2 Combo	P1	P1	P1 mono and combo BCL2
Type of tumor	Hematologic & Solid tumors	-	AML/MDS Combos	Hematologic	CLL stopped Solid combo with IO	AML, CLL, ALL Solid tumors	Solid tumors	B-cell malignancies and AML
IC_{50} on CDK9	3 nM[1] ATP[0.01 mM] 4 nM ATP[2 mM]	13 nM[2]	22 nM[3]	14 nM[4]	13 nM[3]	26 nM[5]	6 nM[6]	1 nM[7]
Half life	4h	2-3h	2-4h	<3h	3h	~1h	-	30h
Route of Admin	IV	Oral	IV	IV	IV	Oral & IV	Oral	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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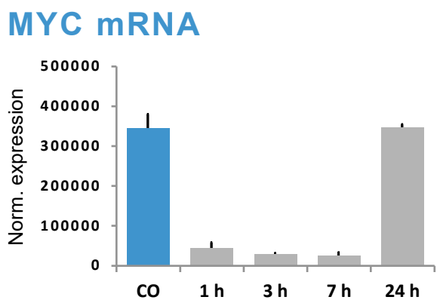
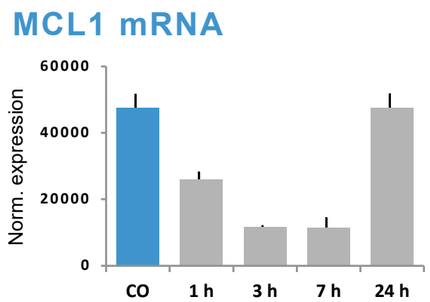
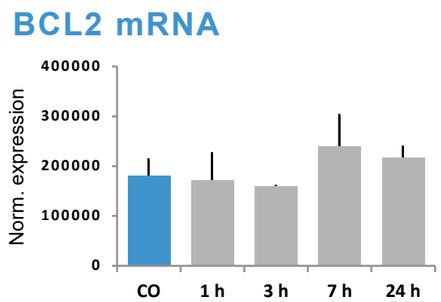
MoA – VIP152 Inhibits the Transcription of MYC and MCL1

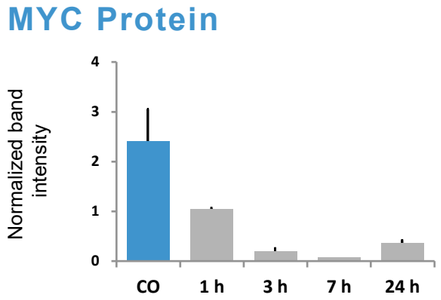
Reduction of MYC & MCL1 mRNA levels



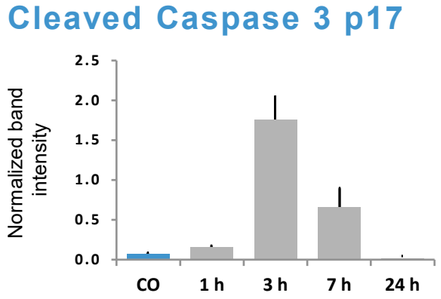


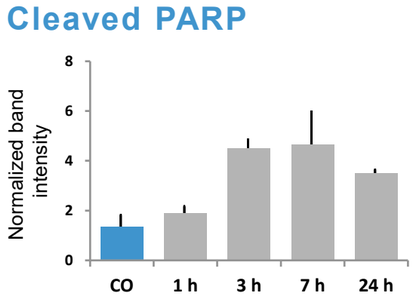


Durable reduction of MYC protein levels



Induction of apoptosis





In vivo MoA in JJN3 multiple myeloma xenografts in mice upon a singe 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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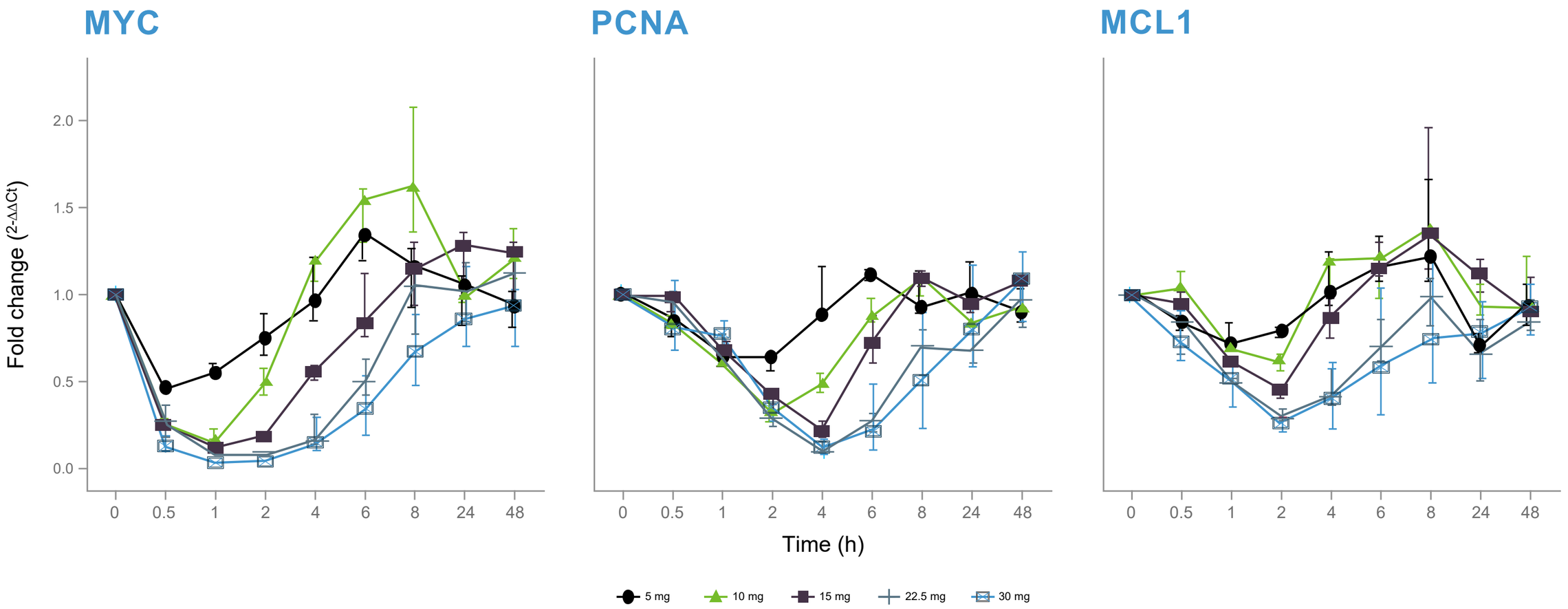
VIP152 Pharmacodynamic Activity in Patient Samples

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

Inhibition of MYC, MCL1 and cell proliferation (PCNA)



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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

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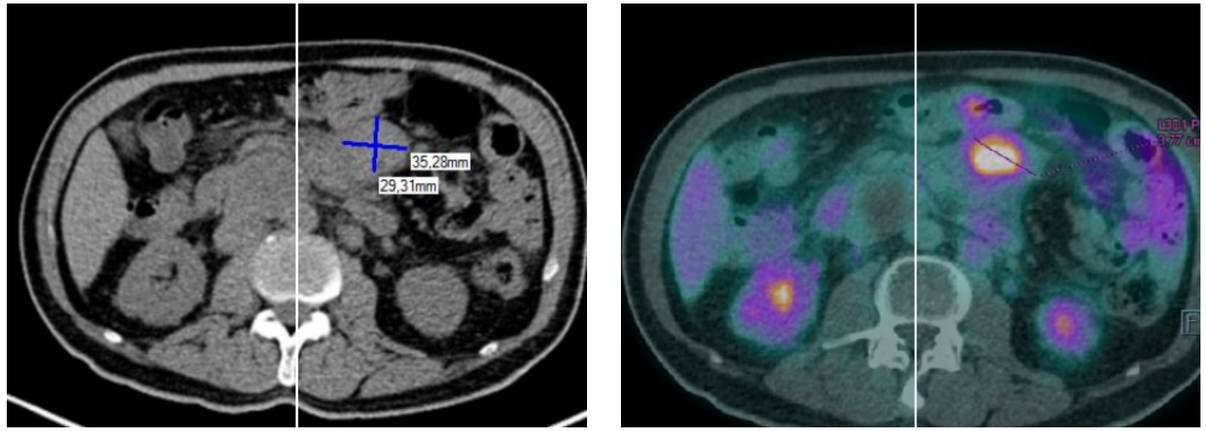
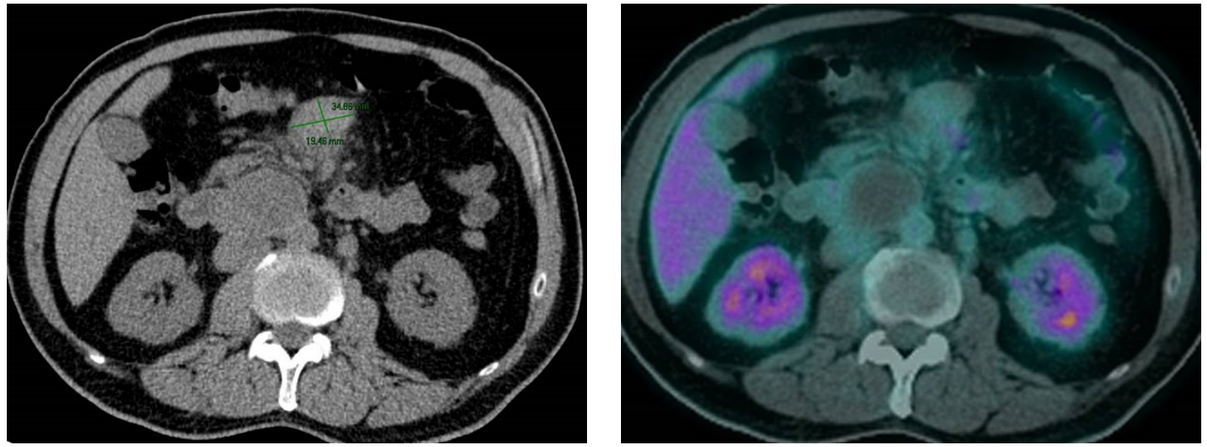
Complete Metabolic Response Observed in Patient with Treatment-refractory Double-hit DLBCL

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

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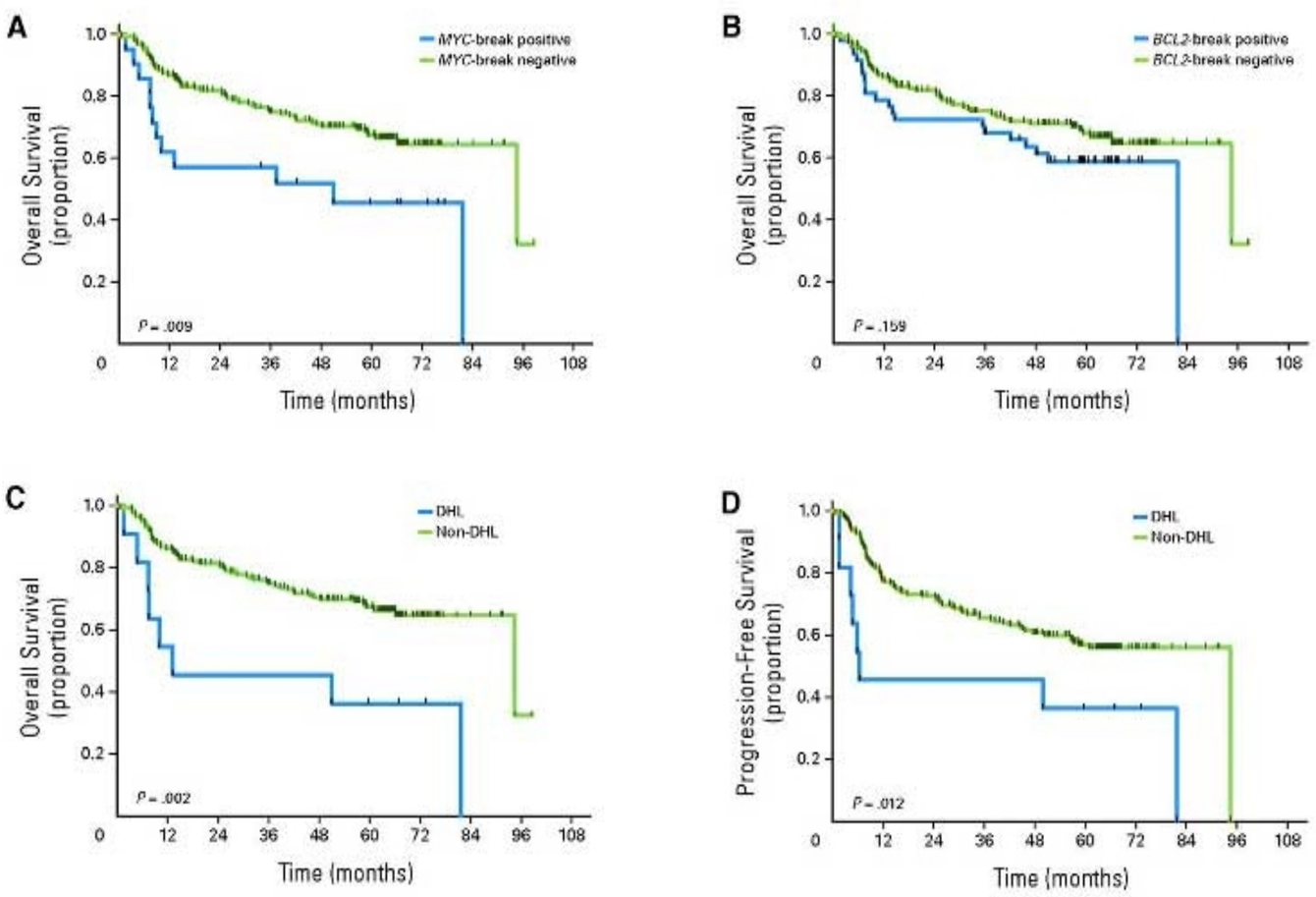
Poor Prognosis in Double-hit Lymphoma

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: 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

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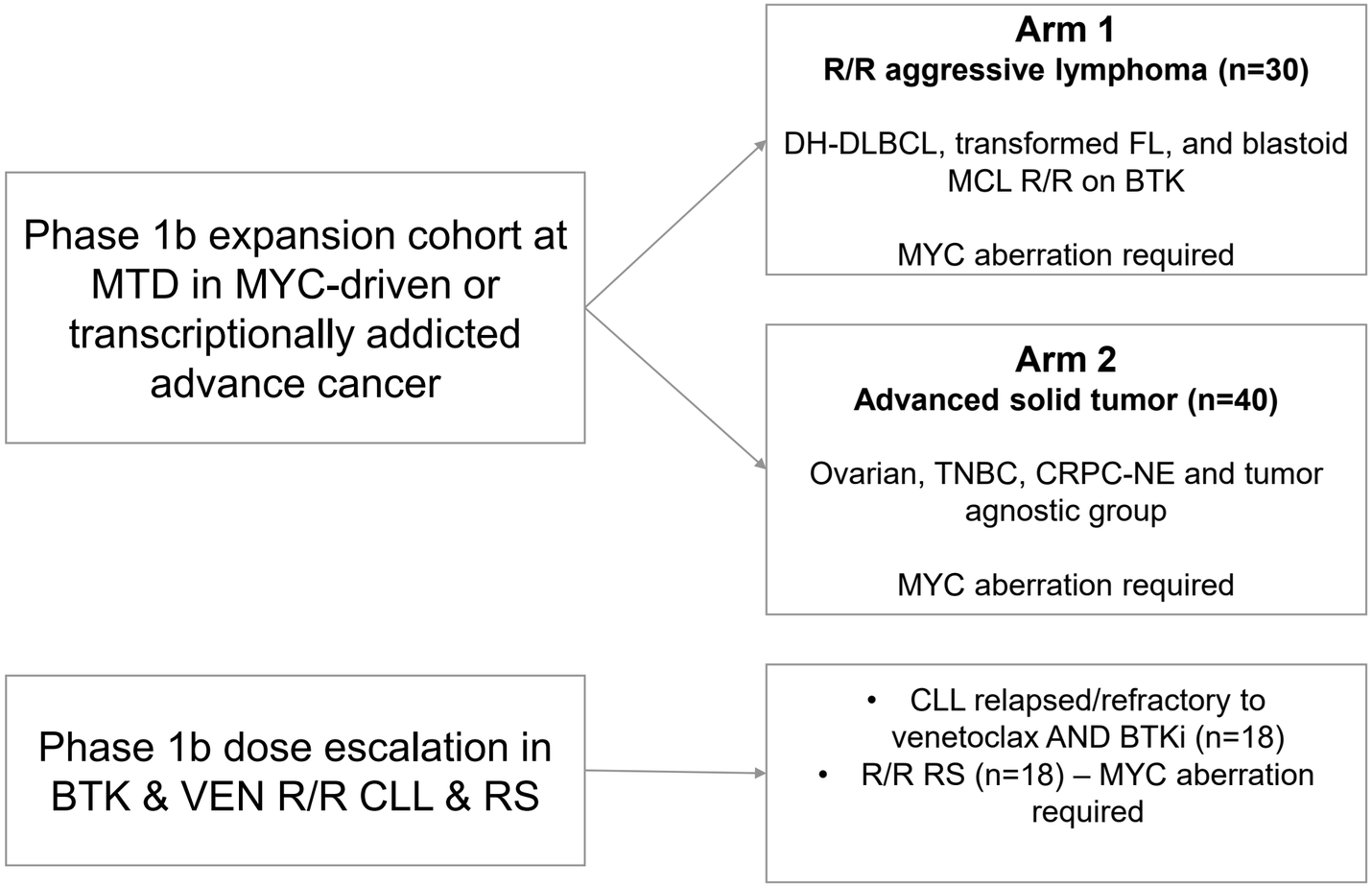
VIP152: Two Phase 1b study designs



- 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

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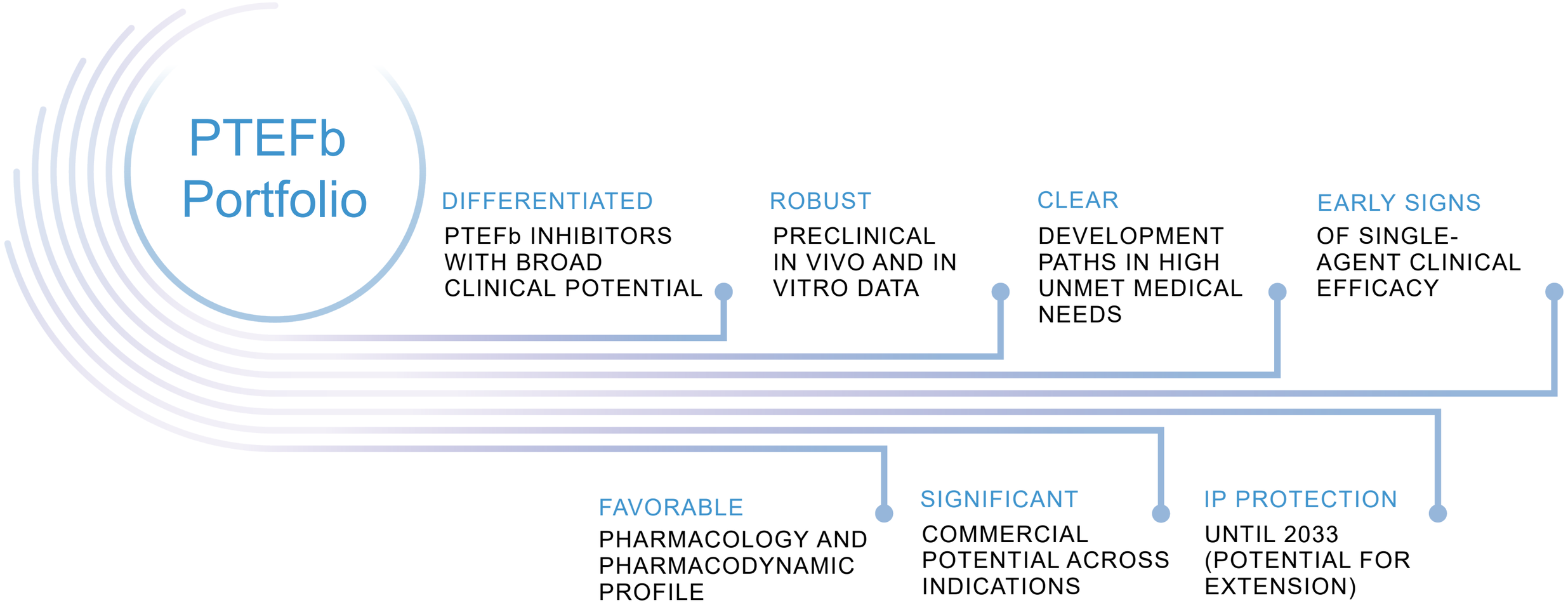
Summary: PTEFb Portfolio



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BIOCONJUGATION PLATFORM

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

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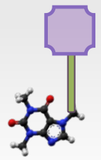
Vincerx's Proprietary Bioconjugate Platforms – Shaping the Future

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

SMALL MOLECULE DRUG CONJUGATE

SMDC

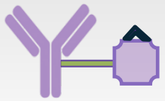


- Conjugate design for highly aggressive advanced diseases
- Targets cell surface cancer markers
- Drug release in tumor stroma
- Tailored warhead design

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

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SMALL MOLECULE DRUG CONJUGATE (SMDC)

VIP236

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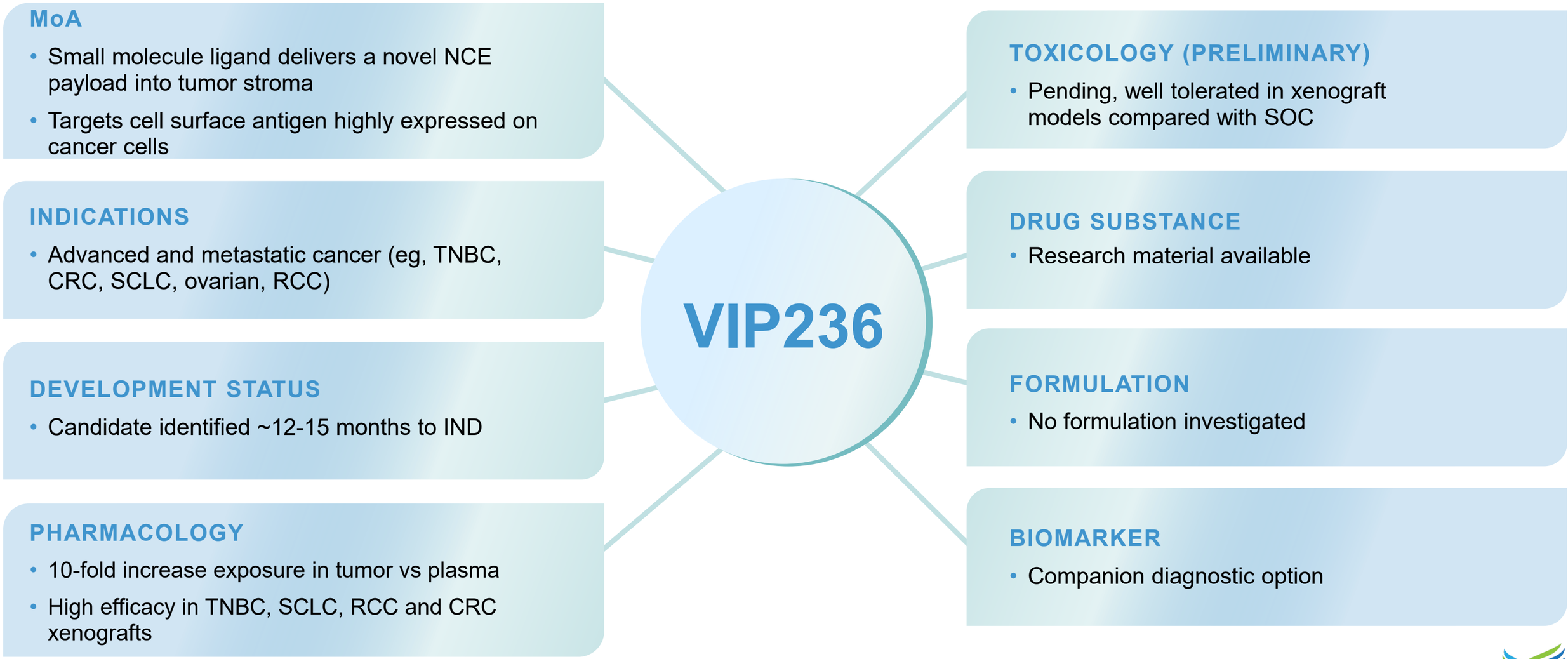
Key Features of VIP236, Cancer Cell Surface Targeting SMDC



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SMDC Dual Targeting Rationale

Tumor Stroma Activated Conjugate

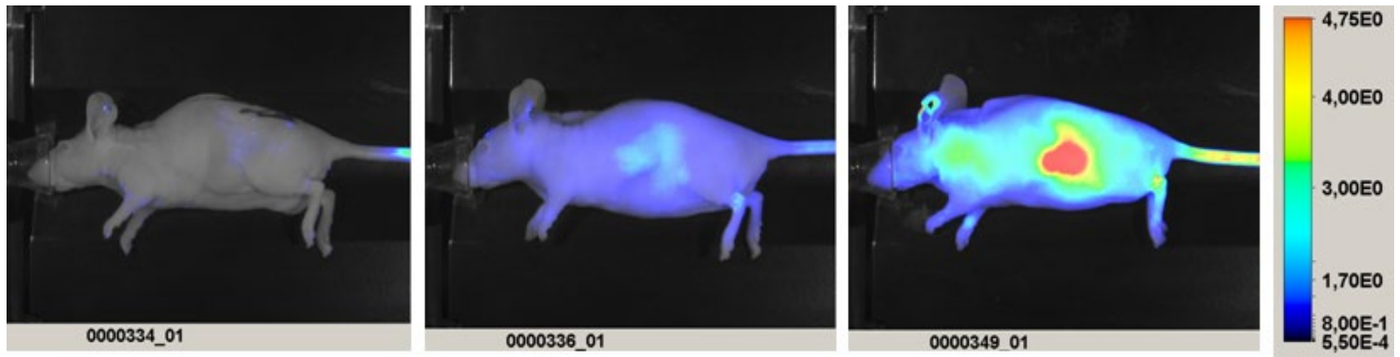
Targeting moiety	Linker enables tumor specific release of active payload	Payload
• Ligand binds cancer selective markers • Stable non-peptidic ligand • Proven tumor homing	• Extracellular cleavage in tumor stroma • Non-cleavable isomer is inactive	• NCE payload • Drug profile tailored for extracellular release

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



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NEXT GEN ADC PLATFORM

VIP943
VIP924

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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

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Key Features of VIP943, an IL3RA-KSPi ADC

MoA

- ADC delivers highly potent KSP inhibitor to IL3RA+ (CD123+) tumors
- AML: >90% of patients express CD123 on blasts and leukemic stem cells
- High levels associated with aggressive disease and poor prognosis (p53 mutated, secondary AML & high risk MDS)

INDICATIONS

- CML/AML/MDS/T-ALL (blastic plasmacytoid dendritic cell neoplasm, B-ALL, hairy cell leukemia, cHL)

DEVELOPMENT STATUS

- Preclinical; IND enabling studies to be initiated. IND in ~18-36 months

PHARMACOLOGY[1]

- High efficacy in AML and in Hodgkin lymphoma xenograft models
- Dose-proportional, typical IgG PK profile

CURRENT STATUS

VIP943

TOXICOLOGY (PRELIMINARY)[1]

- Non-GLP single and repeat (monkey) dose studies available: No neutropenia, thrombocytopenia, liver tox or mucositis up to 20mg/kg (SD) and 10mg/kg (repeated dose)

DRUG SUBSTANCE

- RCB has been established. USP and DSP for non-GMP mAb in late-stage development; Release and stability testing methods in place for mAb. Stability data support DAR=6

FORMULATION

- In development

BIOMARKER

- Companion diagnostic option

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

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Key Features of VIP924, a CXCR5-KSPi ADC

CURRENT STATUS

VIP924

MoA
- ADC delivers KSP inhibitor to CXCR5 positive tumors
- High CXCR5 RNA and protein expression in different Non-Hodgkin lymphoma subtypes

INDICATIONS
- DLBCL, MCL, FL, CLL

DEVELOPMENT STATUS
- ~24-36 months to IND; CMC synergies with IL3RA ADC

PHARMACOLOGY[1]
- High efficacy in MCL & ABC-DLBCL xenografts

TOXICOLOGY (PRELIMINARY)
- Pending, same toxophore-linker chemistry as IL3RA ADC

DRUG SUBSTANCE
- Research material available

FORMULATION
- No formulation investigated yet; similarity to IL3RA ADC anticipated

BIOMARKER
- Companion diagnostic option

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

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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

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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