CORPORATE OVERVIEW

AUGUST 2022

Vincerx PHARMA®

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Vincerx PHARMA®

OUR VISION

WE ASPIRE TO CONQUER CANCER

by addressing the unmet medical needs of our patients with paradigm-shifting therapeutics

Building a Next-Generation Oncology Pipeline

STRONG MANAGEMENT TEAM WITH A PROVEN TRACK RECORD OF CLINICAL AND REGULATORY SUCCESS

DIVERSE PIPELINE WITH MULTIPLE BEST IN CLASS OPPORTUNITIES

INNOVATIVE, NEXT-GENERATION BIOCONJUGATION PLATFORM

Vincerx PHARMA

Renowned Management Team

120 YEARS OF DRUG DEVELOPMENT EXPERIENCE

AHMED HAMDY

RAQUEL IZUMI

TOM THOMAS

ALEX SEELENBERGER

BEATRIX STELTE-LUDWIG

HANS-GEORG LERCHEN

PROVEN TRACK RECORD OF SUCCESSFUL DRUG DEVELOPMENT, APPROVALS, AND VALUE CREATION

Acerta Pharma · AMGEN · AstraZeneca · BAYER

CSL Behring Biotherapies for Life® · Genentech · Janssen Pharmaceutical Companies of Johnson & Johnson · pharmacyclics An AbbVie Company · PDL BioPharma®

Vincerx PHARMA

Extensive Company Expertise

51 DRUG APPROVALS/LAUNCHES

37 ONCOLOGY DRUG APPROVALS/LAUNCHES

105 PRIOR BIOTECH/PHARMA COMPANIES

>40 DECADES OF BIOTECH/PHARMA EXPERIENCE

Blockbuster Exits

CALQUENCE®
(acalabrutinib) 100 mg capsules

$7B acquisition by AstraZeneca (AZ) in 2016 for acalabrutinib in phase 3

MANAGEMENT TEAM'S CONTRIBUTION

Founded Acerta with acalabrutinib at preclinical stage

Accelerated approval in 4 years

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

imbruvica®
(ibrutinib)
560, 420, 280, 140 mg tablets | 140, 70 mg capsules

$975M partnership with Janssen in 2011 ($150 up front, $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 3 phase 2 studies garnered break through therapy designation and accelerated approvals

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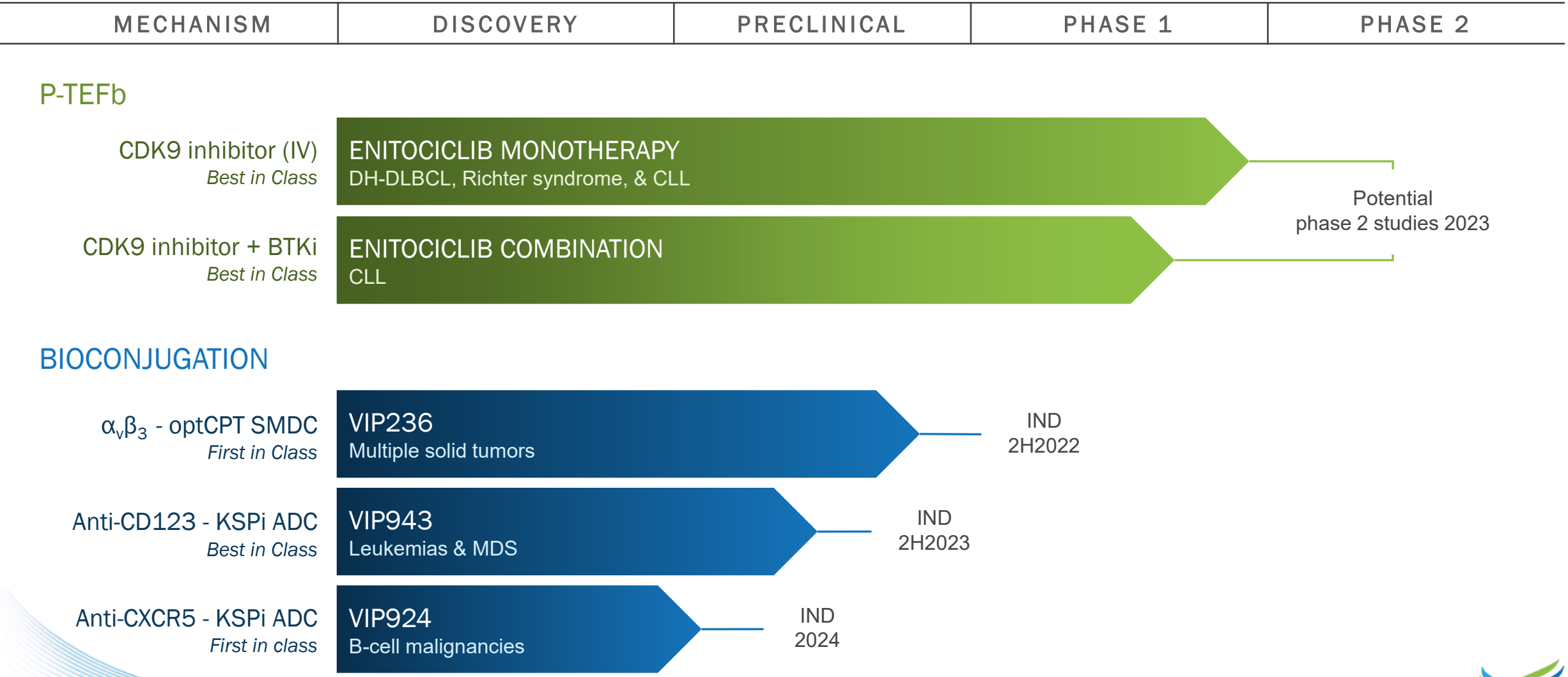
CLL, chronic lymphocytic leukemia; MCL, mantle cell lymphoma.

Pipeline



ADC, antibody-drug conjugate; BTKi, Bruton tyrosine kinase inhibitor; CDK, cyclin-dependent kinase; DH-DLBCL, double-hit diffuse large B-cell lymphoma; IND, indication; IV, intravenous; KSPi, kinesin spindle protein inhibitor; MDS, myelodysplastic syndrome; optCPT, optimized camptothecin; P-TEFb, positive transcription elongation factor B; SMDC, small molecule drug conjugate.

Developing Solutions to the ADC Problems

CHALLENGES

Side effects caused by cell-permeable DNA-damaging payloads or microtubule inhibitors	Premature release of cytotoxic payloads	ADC aggregation and unspecific cellular uptake driven by hydrophobic payloads

VINCERX DESIGN SOLUTIONS	BENEFITS
Internalizing highly selective antibodies	Direct tumor targeting
Linker specifically cleaved intracellularly by legumain, a tumor-associated protease	Unique cleavage sequence (no unspecific cleavage) Second level of tumor targeting via specific ADC activation
KSPi, a novel payload class in ADCs that is hydrophilic	High potency and novel MoA Efficacy: Allows for high DAR without affecting PK Safety: No side effects associated with aggregation
Impermeable payload — CellTrapper™ attached to KSPi to reduce membrane permeability KSPi payload with CellTrapper is hydrophilic	Efficacy: High and long-lasting tumor accumulation Safety: No unspecific uptake of released payload into healthy cells Low/no toxicity in nondividing cells, no neurotoxicity Flexibility, compatible with different linker designs

DAR, drug antibody ratio; MoA, mechanism of action; PK, pharmacokinetics.

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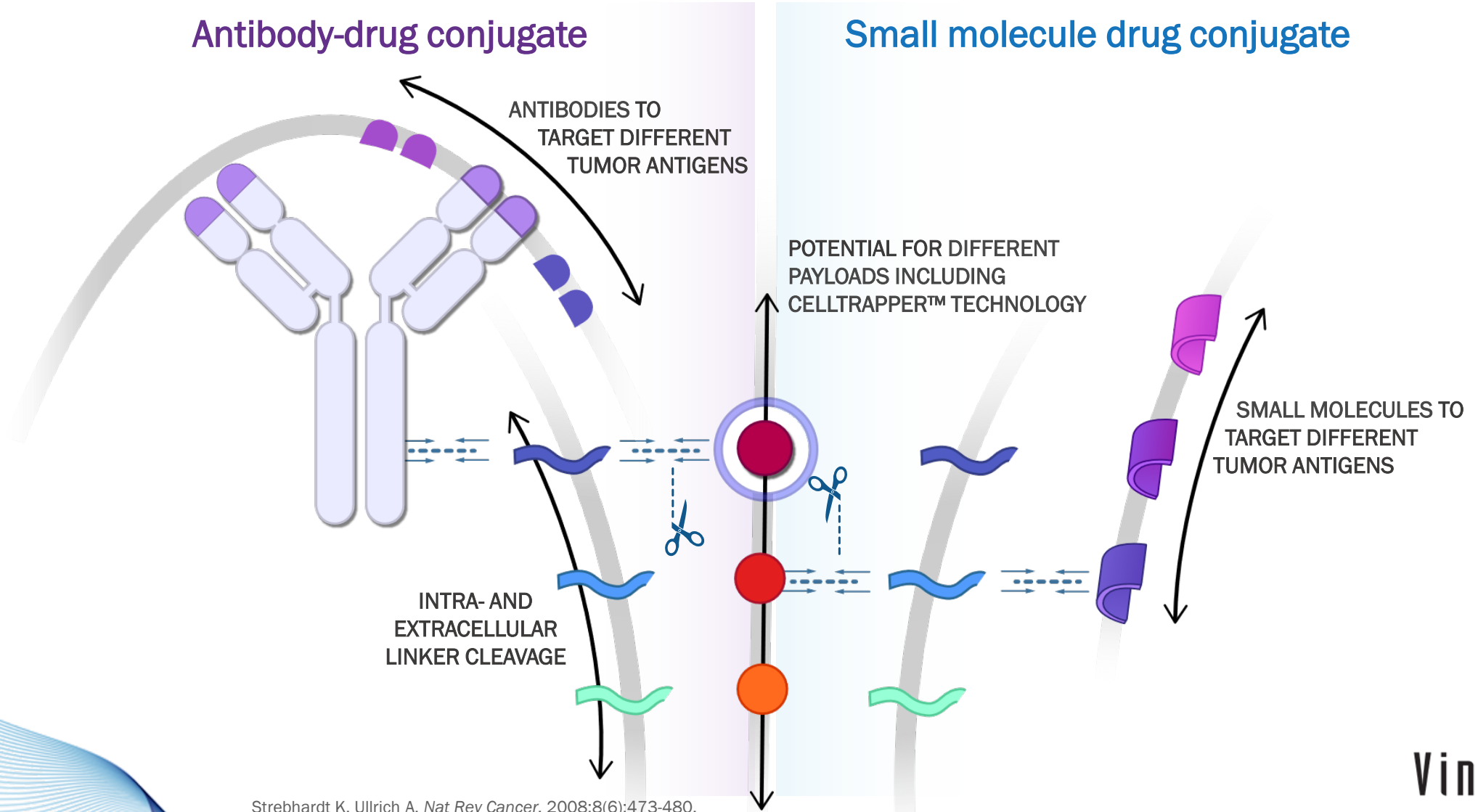
Delivering on the Promise of the “Magic Bullet”

PROPRIETARY TUNABLE AND MODULAR PLATFORM: DESIGNING BESPOKE THERAPIES



Strebhardt K, Ullrich A. *Nat Rev Cancer.* 2008;8(6):473-480.

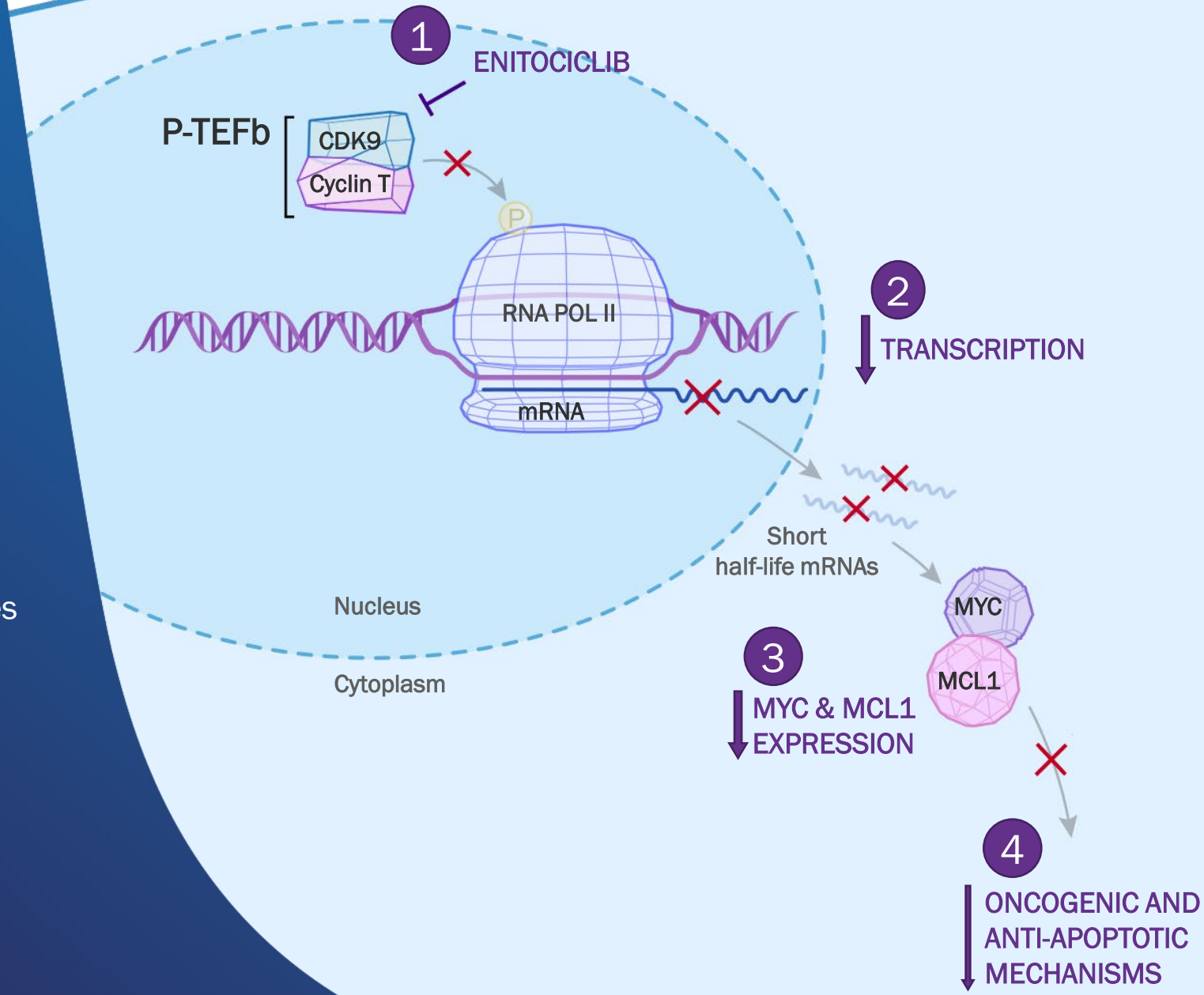
Enitociclib

P-TEFb PROGRAM

Positive Transcription Elongation Factor B (P-TEFb)

A NOVEL TARGET FOR ONCOLOGY

1. Enitociclib inhibits CDK9 preventing activation of RNA polymerase II
2. Inactivation of RNA polymerase II causes rapid depletion of short-lived mRNAs
3. Expression of known oncogenes, MYC and MCL1, is reduced
4. Control of MYC and MCL1 delivers “oncogenic shock”



mRNA, messenger RNA.

Enitociclib Demonstrates Highest CDK9 Selectivity

Enitociclib Is the Most Selective CDK9 Inhibitor

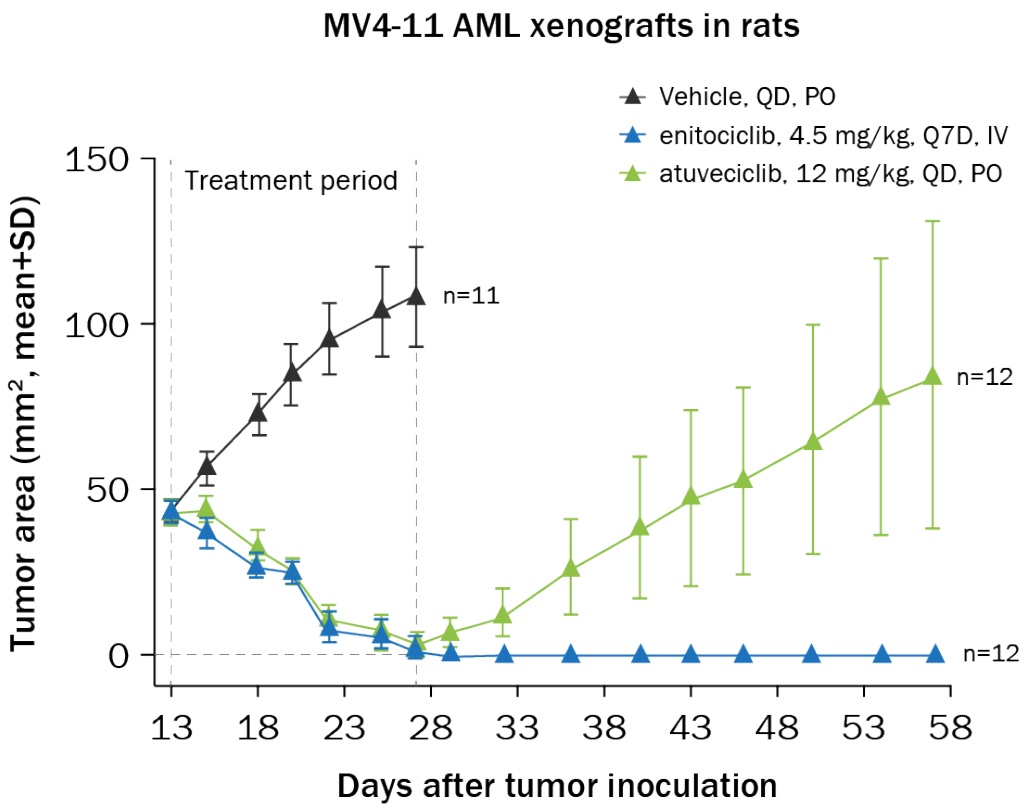
Target	Enitociclib Kd [nM]	Fadraciclib Kd [nM]	Flavopiridol Kd [nM]	KB-0742 Kd [nM]	AZD4573 Kd [nM]
CDK9	0.57	63	2.9	19	0.73
CDK1	>1000-fold	>10-fold	>50-fold	>10-fold	<10-fold
CDK2	>1000-fold	<10-fold	>250-fold	>10-fold	<10-fold
CDK3	>1000-fold	<10-fold	>100-fold	>10-fold	<10-fold
CDK4-cyclinD1	>250-fold	<10-fold	<10-fold	>10-fold	<10-fold
CDK4-cyclinD3	>100-fold	<10-fold	<10-fold	>10-fold	>10-fold
CDK5	>1000-fold	<10-fold	>10-fold	>10-fold	>50-fold
CDK6	>1000-fold	>10-fold	>250-fold	>10-fold	<10-fold
CDK7	>50-fold	<10-fold	>10-fold	<10-fold	<10-fold
GSK3A	>10-fold	>10-fold	>100-fold	>10-fold	<10-fold
IRAK1	>100-fold	>10-fold	>250-fold	>10-fold	>10-fold

Fold difference relative to Kd values determined for CDK9.

Enitociclib Retains Potency at Low and High ATP Concentrations

Compound	Enitociclib	Fadraciclib	Flavopiridol	KB-0742	AZD4573
IC50 (nM) at 10 μM ATP	4.52	28.20	5.96	29.40	3.20
IC50 (nM) at 2 mM ATP	11.80	1.670	32.80	1.130	4.22

Once-Weekly IV Dosing With Enitociclib (VIP152) Is More Effective Than Daily Oral Dosing With Atuveciclib

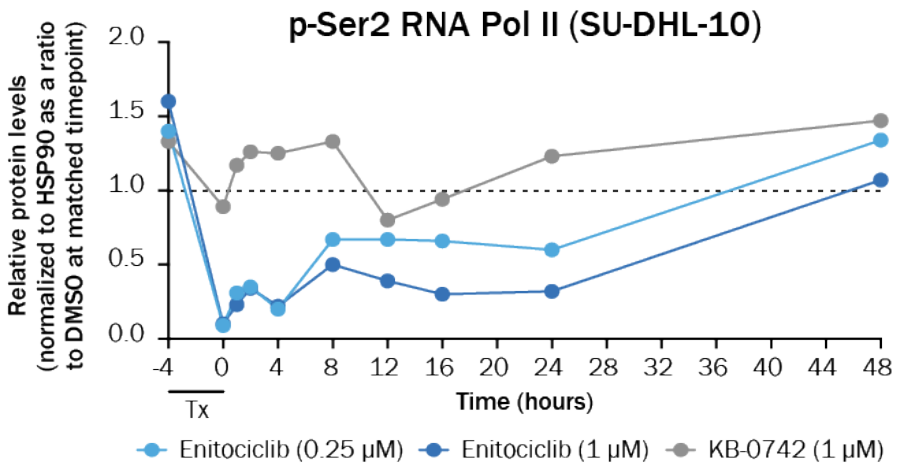


AML, acute myeloid leukemia; ATP, adenosine triphosphate; IC, inhibitory concentration; IV, intravenous; PO, by mouth; Q7D, every 7 days; QD, once daily; SD, standard deviation.

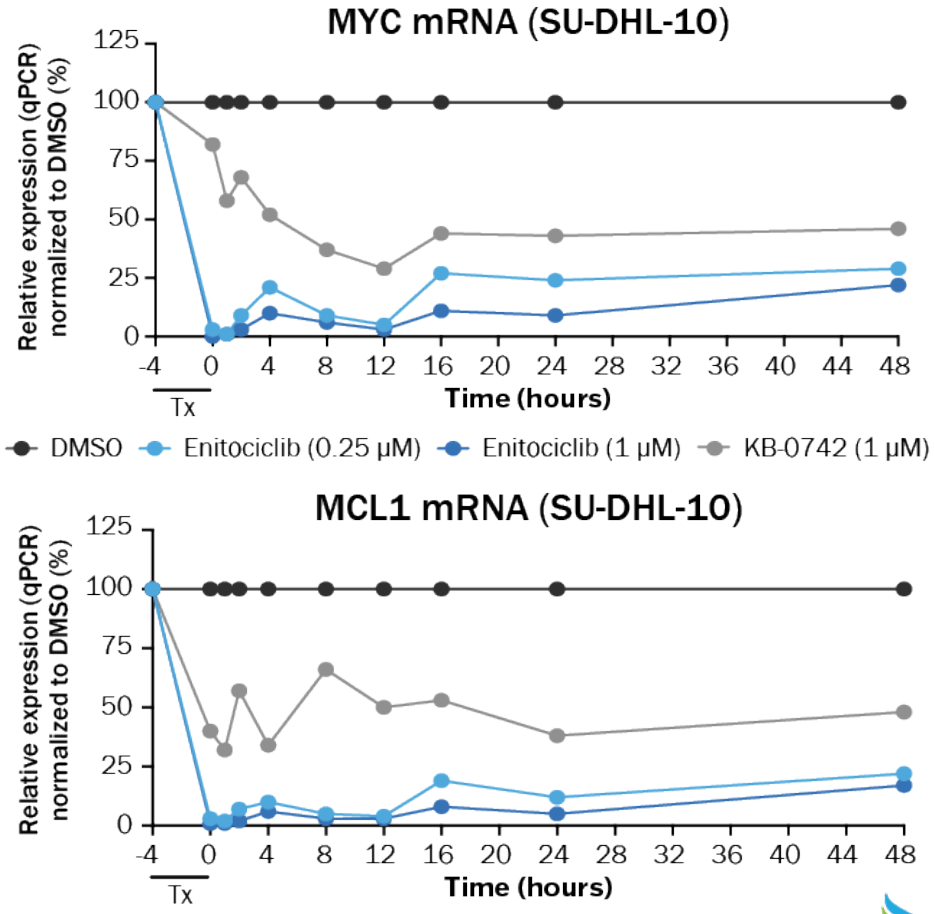
Frigault, et al. EHA 2022.
Diamond, et al. CCR 2022.

Enitociclib MoA Drives Sustained Inhibition of MYC and MCL1 Transcription

Enitociclib Provides Most Robust and Durable Reduction of p-RNA Polymerase II



Enitociclib Depletes and Maintains Deeper Inhibition of MYC and MCL1 mRNA

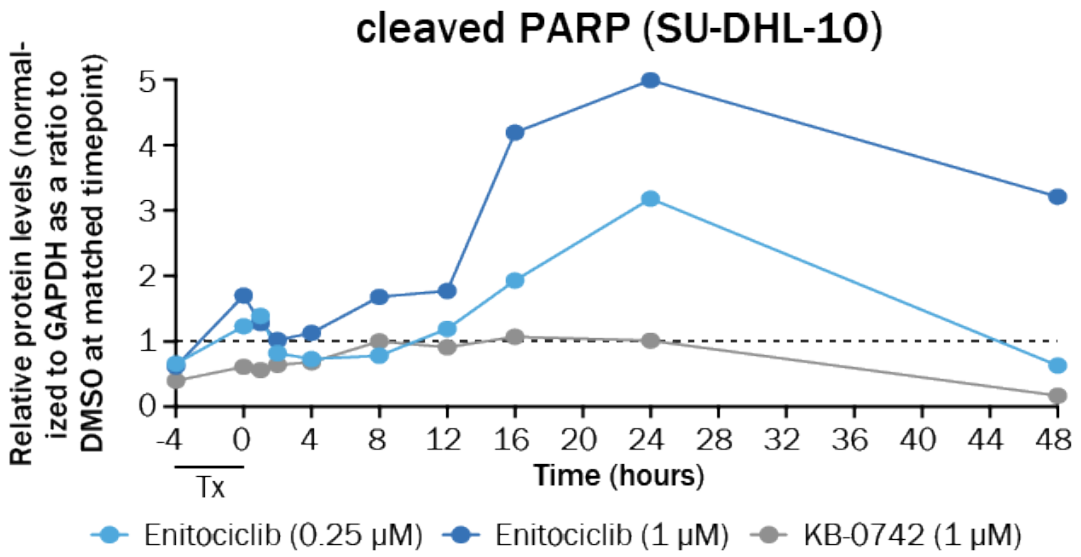


4-hour in vitro pretreatment with enitociclib compared with KB-0742 in the MYC-mutant and overexpressed SU-DHL-10 cell line

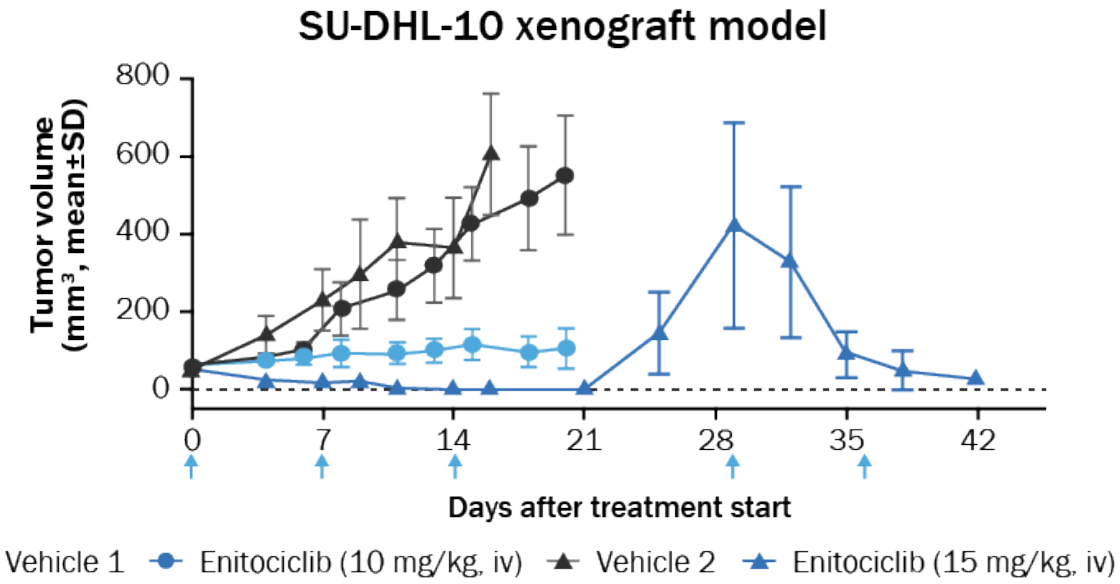
DMSO, dimethyl sulfoxide; qPCR, quantitative polymerase chain reaction; Tx, treatment.
Frigault, et al. ASH 2021.

Enitociclib Leads to Apoptosis Induction and In Vivo Tumor Regression

Enitociclib Leads to 3- to 5-fold Induction of Apoptosis In Vitro



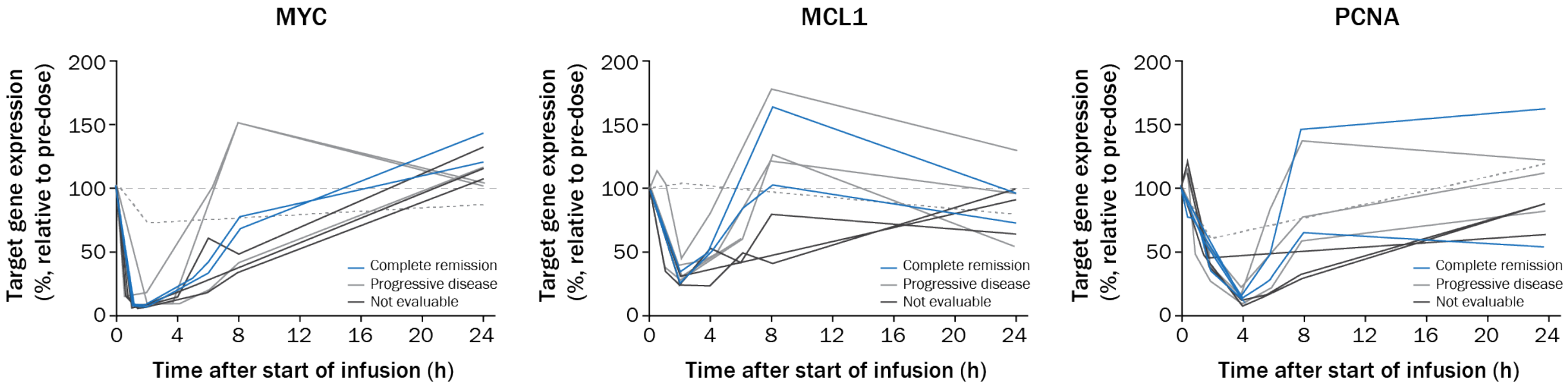
Dose-Dependent In Vivo Tumor Growth Inhibition



GAPDH, glyceraldehyde 3-phosphate dehydrogenase; PARP, poly(ADP-ribose) polymerase.
Frigault, et al. ASH 2021.

Enitociclib MoA Confirmed in the Clinic

Enitociclib Robust Down Modulation of MYC, MCL1, and PCNA mRNAs in Whole Blood of Patients With DLBCL and CLL



DLBCL, diffuse large B-cell lymphoma; PCNA, proliferating cell nuclear antigen.
Frigault, et al. EHA 2022.

Potential Indications

MYC AND MCL1 OVEREXPRESSION IS A HALLMARK OF MULTIPLE AGGRESSIVE, RESISTANT TUMORS REPRESENTING A WIDE-RANGING UNMET MEDICAL NEED

MYC-Dependent B-Cell Lymphoma
DH-DLBCL, Transformed FL,
Richter Syndrome, MCL

MCL1-Dependent Leukemias
CLL, AML, MDS

MCL1-Dependent Multiple Myeloma

MCL1- or MYC-Dependent Solid Tumors
Ovarian, TNBC, CRPC including NEPC

Lossos IS, et al. *PNAS*. 2002;99(13):8886-8891; Wang N, et al. *Blood.* 2020;135(1):17-27; Jain, et al. ASH. 2021; Yue XY, et al. *Cancer Cell Int*. 2020;524(20); Wallington-Beddoe CT, Mynott J. *Hematol Oncol*. 2021;14(1):151; Lourenco C, et al. *Nat Rev Cancer*. 2021;21(9):579-591.

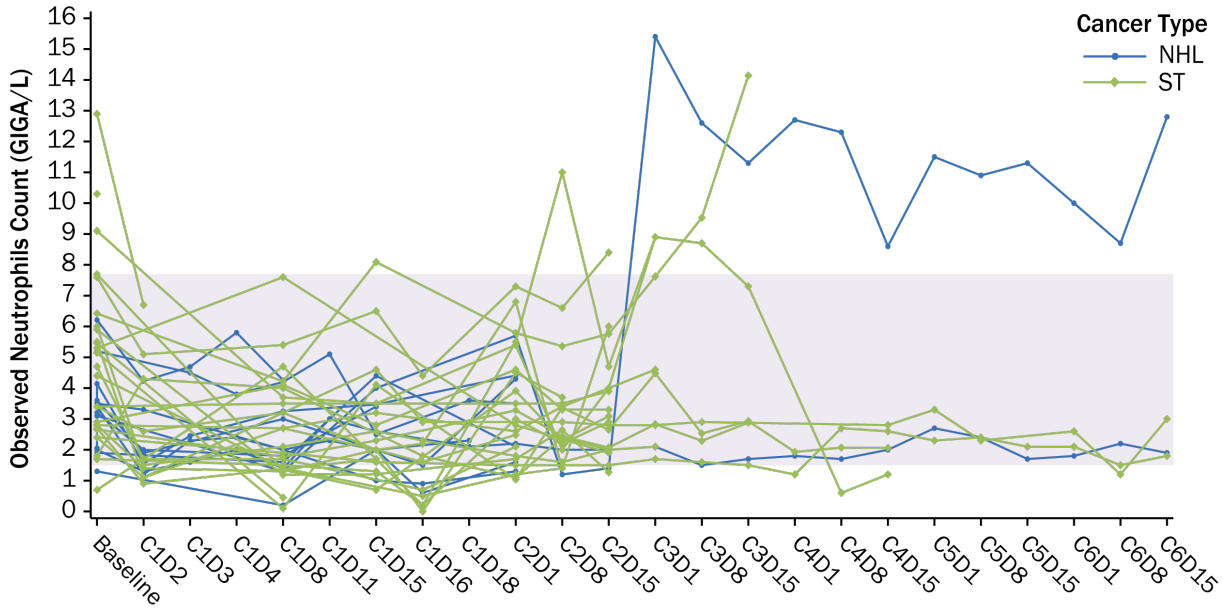
CRPC, castration-resistant prostate cancer; FL, follicular lymphoma; NEPC, neuroendocrine prostate cancer; TNBC, triple-negative breast cancer.

Enitociclib Has a Favorable Safety Profile in Patients With Solid Tumors and Lymphoma

Treatment-Emergent Adverse Events (n=56)

Adverse Events (>15%)	Any Gr	Gr 1	Gr 2	Gr 3	Gr 4	Gr 5
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Nausea	37 (66.1)	22 (39.3)	15 (26.8)	0	0	0
Vomiting	30 (53.6)	20 (35.7)	10 (17.9)	0	0	0
Fatigue	19 (33.9)	8 (14.3)	10 (17.9)	1 (1.8)	0	0
Anemia	17 (30.4)	6 (10.7)	7 (12.5)	4 (7.1)	0	0
Diarrhea	17 (30.4)	13 (23.2)	4 (7.1)	0	0	0
Neutropenia	12 (21.4)	0	4 (7.1)	5 (8.9)	3 (5.4)	0
Constipation	9 (16.1)	7 (12.5)	2 (3.6)	0	0	0

Neutropenia Is an On-Target (CDK9) Toxicity and Is Monitorable and Manageable With Supportive Care (n=37 patients at 30 mg dose)



Cardiac safety analysis (n=57)

In an analysis of triplicate electrocardiogram and matched PK data from 57 patients with solid or hematologic cancer, enitociclib did not prolong (<10 ms) the QTc interval (QTc/F) after a single or multiple 5 to 30 mg doses once weekly, indicating a favorable cardiac safety profile

Gr, grade; NHL, non-Hodgkin lymphoma; ST, solid tumor.
Frigault, et al. EHA 2022.

Enitociclib Is Well Tolerated and Induces Durable Complete Responses (n=54)

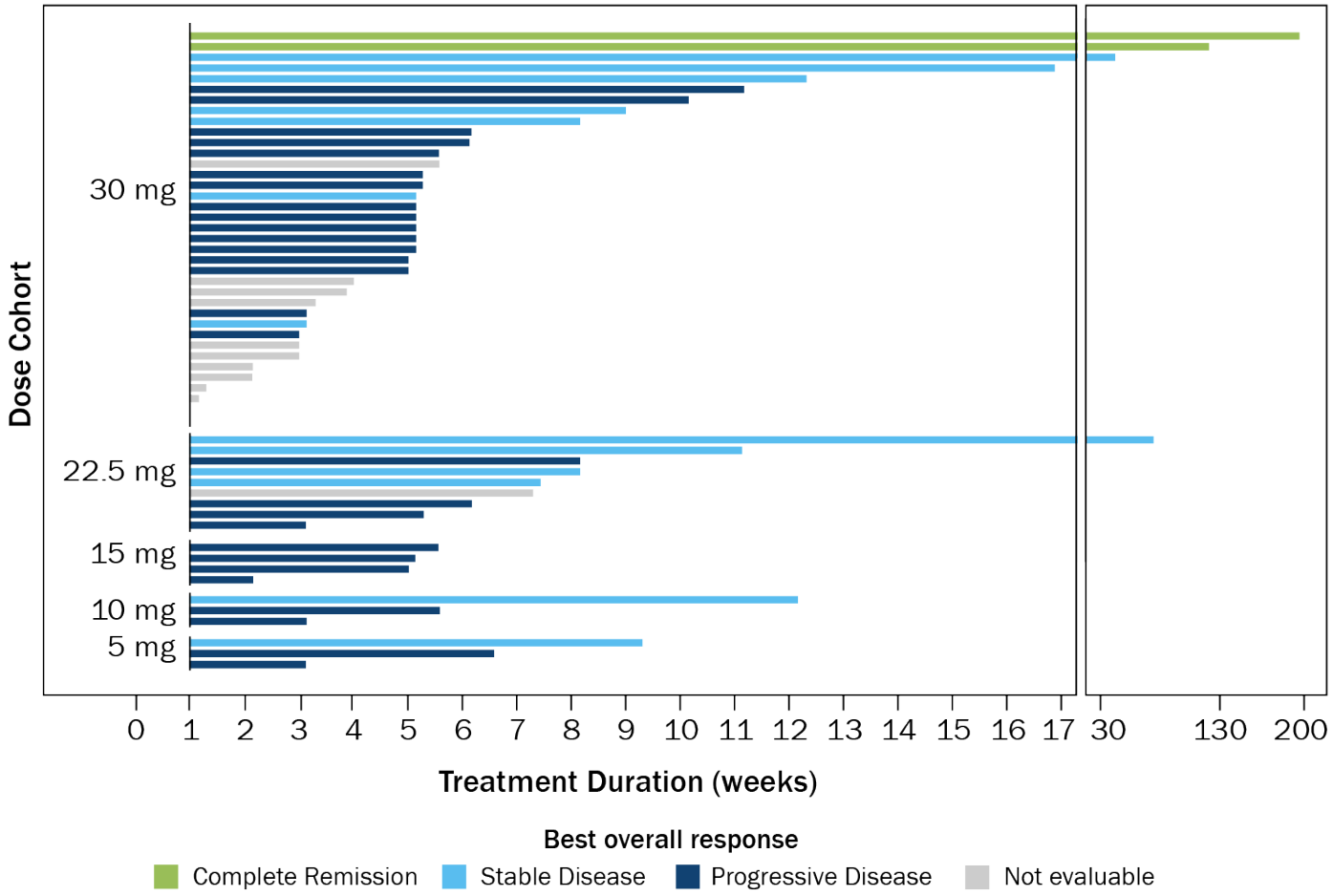
Monotherapy Activity

2 CRs of 7 DH-DLBCL (29% CR rate)

- 1 on treatment for 3.7 years
- 1 on treatment for 2.3 years
- Both patients continue in full remission ~2 years after stopping treatment

13 patients had stable disease as best response:

- 5 ovarian cancer, 2 to 6 cycles
- 2 pancreatic cancer, 3 and 14 cycles
- 2 esophageal/nasopharyngeal, 2 and 3 cycles
- 1 salivary gland cancer, 24 cycles
- 1 breast cancer, 3 cycles
- 1 clival chordoma, 4 cycles
- 1 appendix cancer, 5 cycles



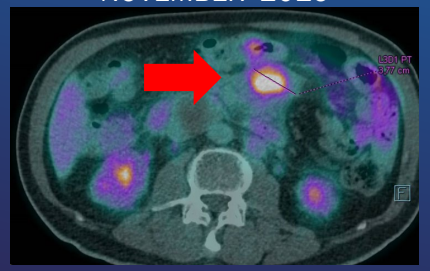
CR, complete response.

Enitociclib Induces Durable Complete Remissions in Refractory DH-DLBCL

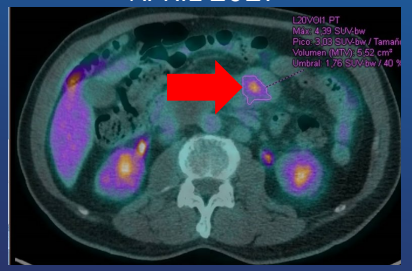
PATIENT 1: Diagnosis DH-DLBCL With MYC and BCL2 Translocations

Age: 58 years
Cell of origin: GCB
Prior therapy (response):
- R-CHOP (PR)
- Radiotherapy (PR)
- R-GemOx (PD)

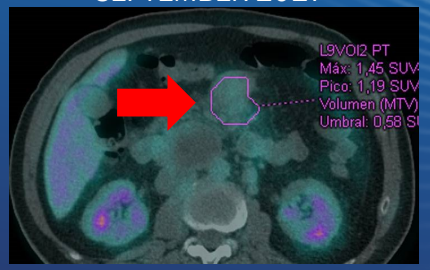
NOVEMBER 2016



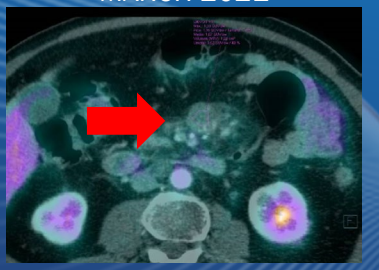
APRIL 2017



SEPTEMBER 2017



MARCH 2021



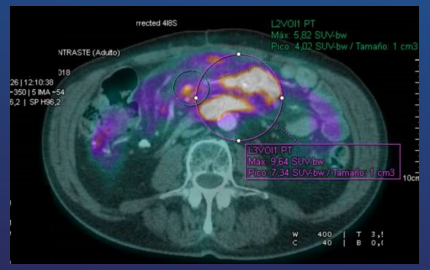
Date of 1st dose: 08 NOV 2016

Date of last dose: 16 JUL 2020

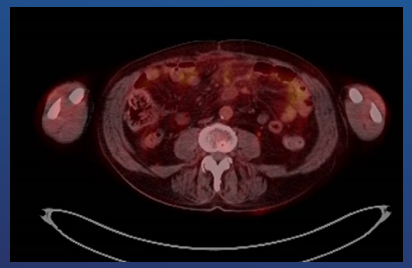
PATIENT 2: Diagnosis DH-DLBCL With MYC and BCL2 Translocations

Age: 78 years
Cell of origin: GCB
Prior therapy (response):
- R-EPOCH (PR)
- R-DHAP (PD)
- Palliative radiotherapy

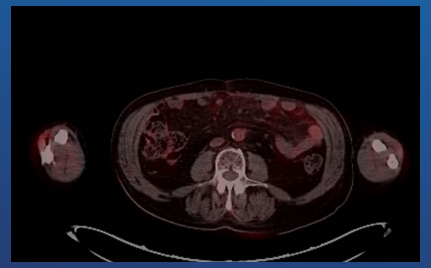
MARCH 2018



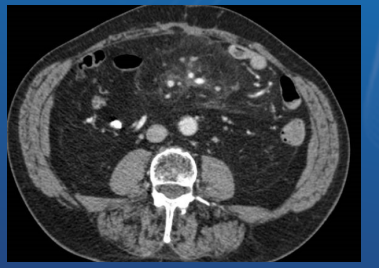
OCTOBER 2018



JUNE 2020



FEBRUARY 2021

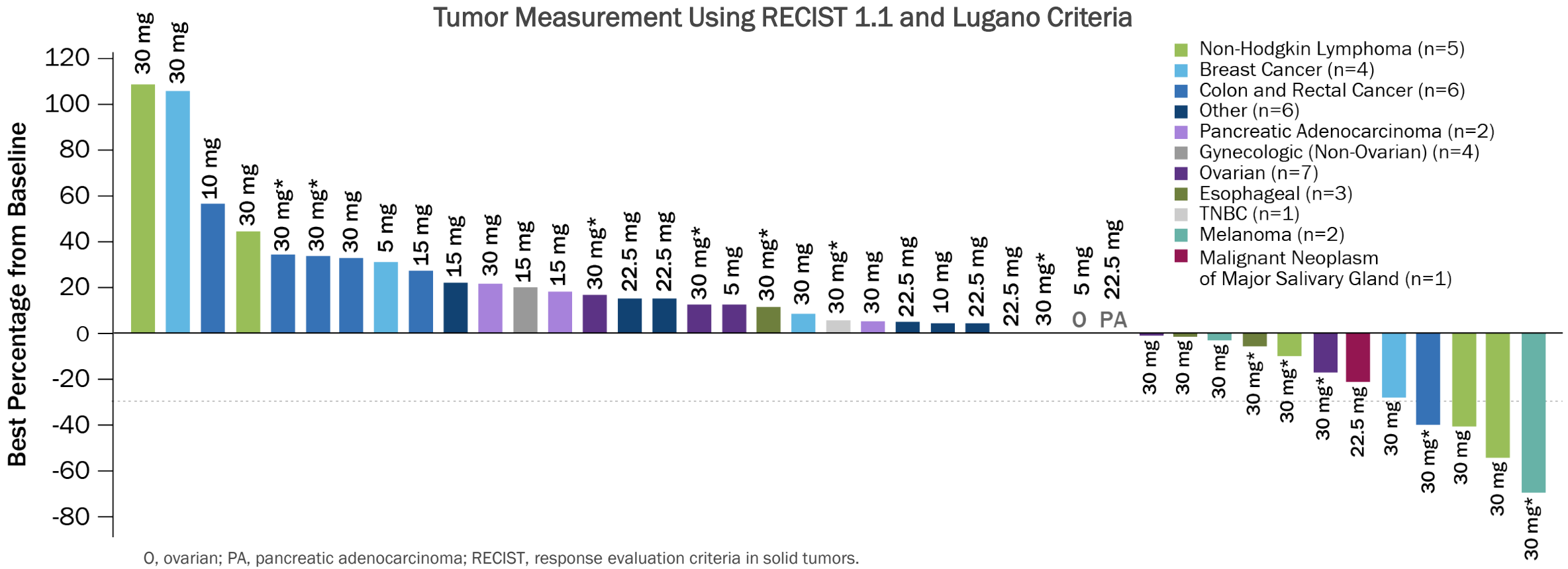


Date of 1st dose: 03 APR 2018

Date of last dose: 23 JUL 2020

CHOP, cyclophosphamide, doxorubicin, vincristine, prednisone; DHAP, dexamethasone, cytarabine, cisplatin; EPOCH, etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin; GCB, germinal center B-cell; GemOx, gemcitabine, oxaliplatin; PD, progressive disease; PR, partial response; R, rituximab.

Enitociclib Demonstrates Reduction in Tumor Volume in Various Indications (n=41)



O, ovarian; PA, pancreatic adenocarcinoma; RECIST, response evaluation criteria in solid tumors.
Note: Other tumor types include appendix cancer, clival chordoma, nasopharyngeal, prostate cancer, supraglottic carcinoma, and thymoma.
*Vincerx Pharma, Inc. enrolled patients.

VNC-152-101 Lymphoma and Solid Tumors Study Design

AMENDMENT 7

PART 1: Dose Escalation
Completed

- Study was conducted by Bayer (BAY 1251152)
- 37 patients treated
- DLT was neutropenia
- MTD at 30mg once-weekly IV

PART 2: Expansion Cohort Arm 1
R/R Aggressive Lymphoma (n=40) MYC aberration required*

- *DH-DLBCL (n=10) Enrolling*
- *Transformed FL (n=10), MCL (n=10), other lymphoma group + MYC aberration (eg, DLBCL, Burkitt lymphoma) (n=10) Paused*

PART 2: Expansion Cohort Arm 2
Advanced Solid Tumor (n=40) MYC aberration required*

- *Ovarian (n=10), TNBC (n=10), CRPC/NEPC (n=10), tumor agnostic group (n=10) Paused*

PART 3/4: Combo in MYC+ Advanced Tumor (n=30)

- Pembrolizumab + VIP152 in MYC+ advanced tumor (dose escalation + expansion) *Paused*

PART 5: Monotherapy Infusion Optimization
R/R Aggressive Lymphoma (n=10)

- *DH-DLBCL (n=10) Enrolling*

*FoundationOne® (or similar commercial panel) or locally confirmed MYC overexpression/amplification/translocation required to enroll; n=10 for each group within each arm; total N=80. Each group within each arm will be evaluated separately for safety and efficacy.

DLT, dose-limiting toxicity; MTD, maximum tolerated dose; R/R, relapsed/refractory.

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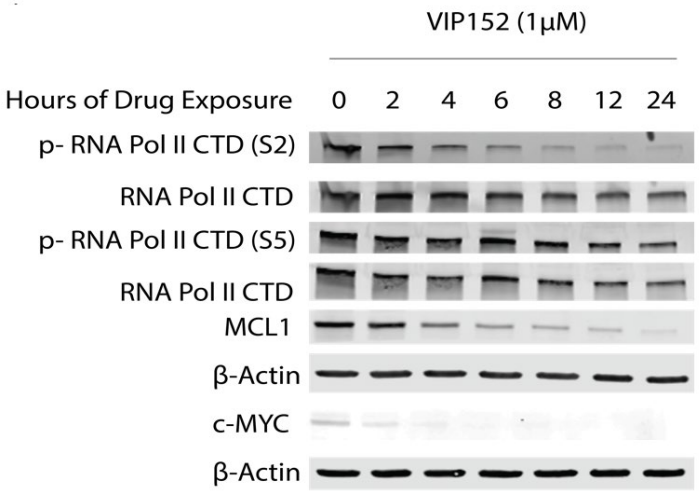
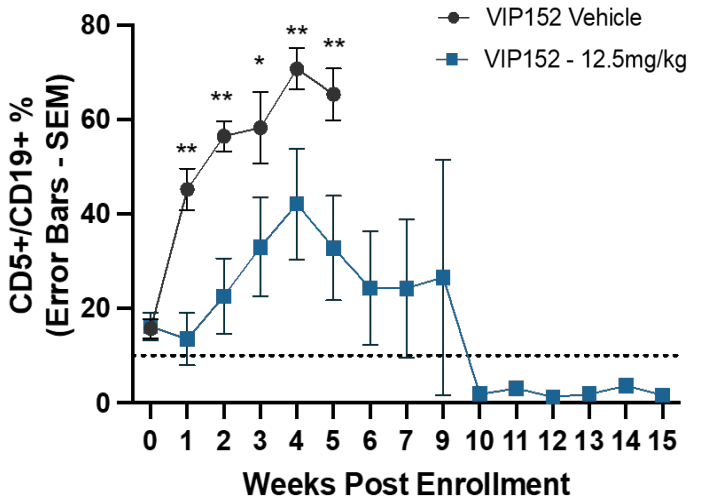
Targeting P-TEFb With Enitociclib (VIP152) in CLL

Depletion of MCL1 and MYC Protein Through RNA Polymerase II Phosphorylation Reduction in HG-3 Cell Line

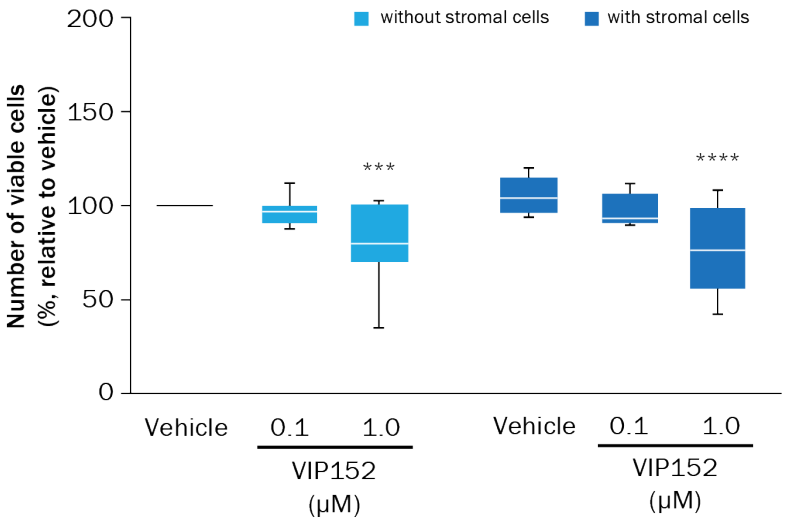


Weekly Dosing Decreases Peripheral Disease in a Circulating CLL Mouse Model



n=10 per arm, * = p <0.05, ** = p <0.005

Cytotoxicity in Cells Derived From CLL Patients Who Are R/R to Ibrutinib and Venetoclax With TP53 Mutations



CDK9 is a proven R/R CLL target and may deliver residual disease eradication in BTKi combination

- Dinaciclib and flavopiridol monotherapy efficacy (ORR 25%-40%, PFS 7.5-13.7 months) reported in phase 2/3 CLL trials
- Like venetoclax, enitociclib also targets the anti-apoptotic pathway by downregulation of MCL1

CTD, C-terminal domain; ORR, overall response rate; PFS, progression-free survival; SEM, standard error of the mean.

Sher, et al. ASH 2021.
Frigault, et al. EHA 2022.

Vincerx PHARMA

VNC-152-102 CLL Study Design

AMENDMENT 2

PART 1: Dose Escalation *Enrolling*
CLL refractory/intolerant to BTKi and Venetoclax

- Dose Level 1: 10mg > 15mg intra-patient dose escalation (n=3)
- Dose Level 2: 15mg > 30mg intra-patient dose escalation (n=3)
- Once-weekly 30-min IV

Enitociclib Monotherapy CLL (n=8)

- Patients with R/R high-risk (del17p or TP53 mutation) CLL who have received ≥ 1 prior therapy that includes venetoclax and have not achieved CR

OR

- Received ≥ 2 prior therapies and intolerant to BTKi and/or venetoclax

PART 2: Enitociclib Combination With Approved BTKi (n=8)

- Dose Level 1: 20mg once-weekly (n=3)
- Dose Level 2: 30mg once-weekly (n=5)
- CLL patients currently on approved BTKi monotherapy >12 months who have only achieved SD, PR, or PR with lymphocytosis

Enitociclib Monotherapy Richter Syndrome (n=8)

- CLL transformed to DLBCL R/R after at least 1 prior therapy

Enrollment to Part 2 will begin after the safety of monotherapy 30mg enitociclib once-weekly has been evaluated in 6 subjects with CLL or Richter Syndrome by the safety review committee.

SD, stable disease.

Vincerx PHARMA

Key Features of Enitociclib

SAFETY

FAVORABLE SAFETY PROFILE IN THE CLINIC WITH MAINLY GR 1-2 TOXICITIES REPORTED

ON-TARGET TOXICITY OF NEUTROPENIA IS MANAGED BY ONCE-WEEKLY DOSING AND SUPPORTIVE CARE

EFFICACY

A POTENT AND THE MOST SELECTIVE CDK9 INHIBITOR CURRENTLY IN CLINICAL DEVELOPMENT

REPRODUCIBLE DOWNMODULATION OF MYC, MCL1, AND PCNA mRNA IN THE WHOLE BLOOD

DURABLE CRs OBSERVED IN 2 PTS WITH DH-DLBCL FOR ~5.5 AND ~4.0 YEARS, OF WHICH 3.7 AND 2.3 YEARS WERE ON TREATMENT

COMMERCIAL POTENTIAL

COMMERCIAL POTENTIAL ACROSS MULTIPLE INDICATIONS, BOTH HEMES AND SOLIDS

IP PROTECTION UNTIL 2033 (POTENTIAL FOR EXTENSION)

IP, intellectual property.

BIOCONJUGATION PLATFORM

Vincerx PHARMA®

Solutions to the ADC Problems

CHALLENGES

Side effects caused by cell-permeable DNA-damaging payloads or microtubule inhibitors	Premature release of cytotoxic payloads	ADC aggregation and unspecific cellular uptake driven by hydrophobic payloads

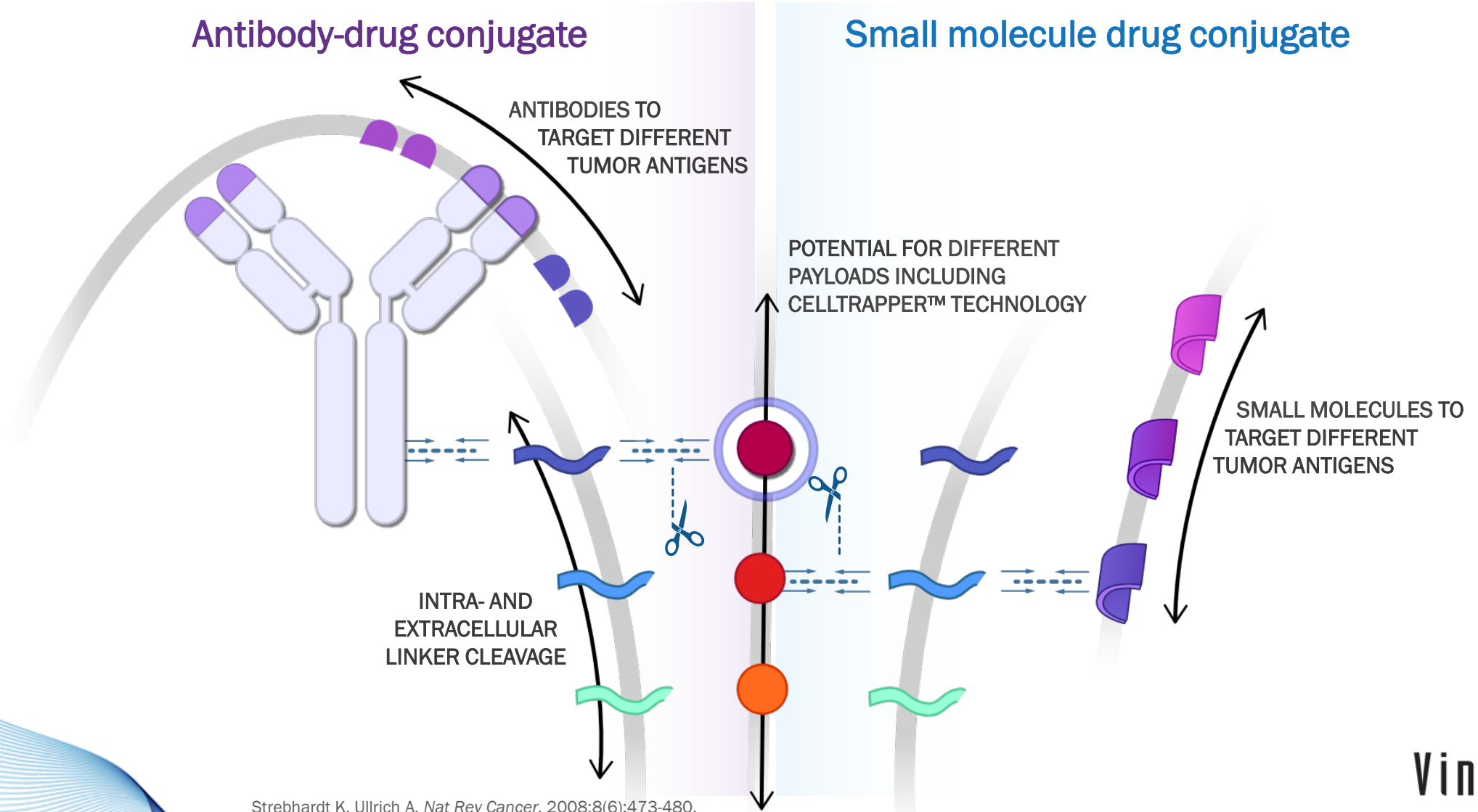
VINCERX DESIGN SOLUTIONS	BENEFITS
Internalizing highly selective antibodies	Direct tumor targeting
Linker specifically cleaved intracellularly by legumain, a tumor-associated protease	Unique cleavage sequence (no unspecific cleavage) Second level of tumor targeting via specific ADC activation
KSPi, a novel payload class in ADCs that is hydrophilic	High potency and novel MoA Efficacy: Allows for high DAR without affecting PK Safety: No side effects associated with aggregation
Impermeable payload — CellTrapper™ attached to KSPi to reduce membrane permeability KSPi payload with CellTrapper is hydrophilic	Efficacy: High and long-lasting tumor accumulation Safety: No unspecific uptake of released payload into healthy cells Low/no toxicity in nondividing cells, no neurotoxicity Flexibility, compatible with different linker designs

DAR, drug antibody ratio; MoA, mechanism of action; PK, pharmacokinetics.

Vincerx PHARMA

Delivering on the Promise of the “Magic Bullet”

OUR PROPRIETARY TUNABLE AND MODULAR PLATFORM: DESIGNING BESPOKE THERAPIES



Strebhardt K, Ullrich A. *Nat Rev Cancer.* 2008;8(6):473-480.

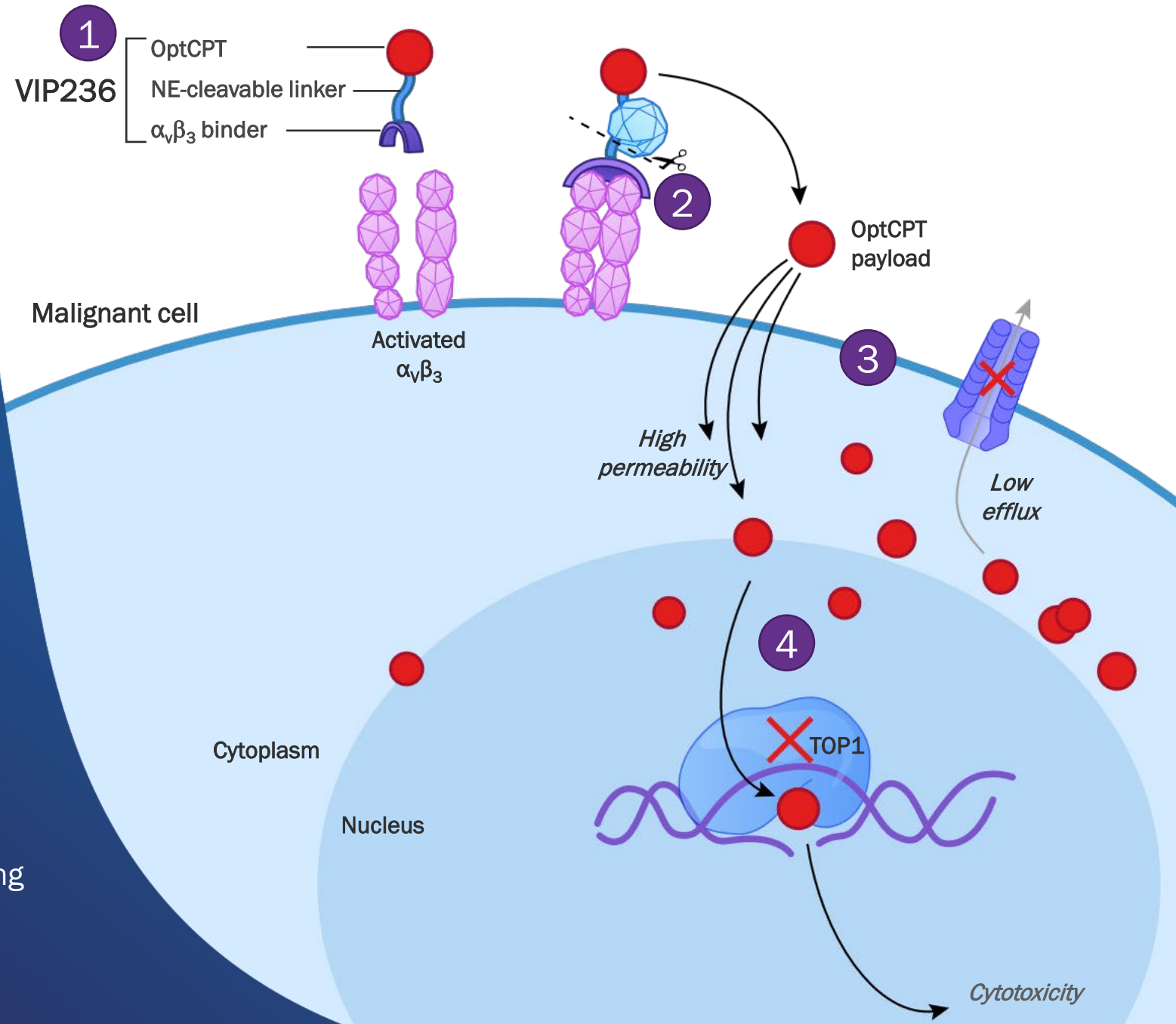
VIP236
$\alpha_v\beta_3$-optCPT SMDC

Vincerx PHARMA

VIP236 $\alpha_V\beta_3$ Small Molecule Drug Conjugated to an optCPT

ENHANCED SAFETY AND PRECISION PROFILE

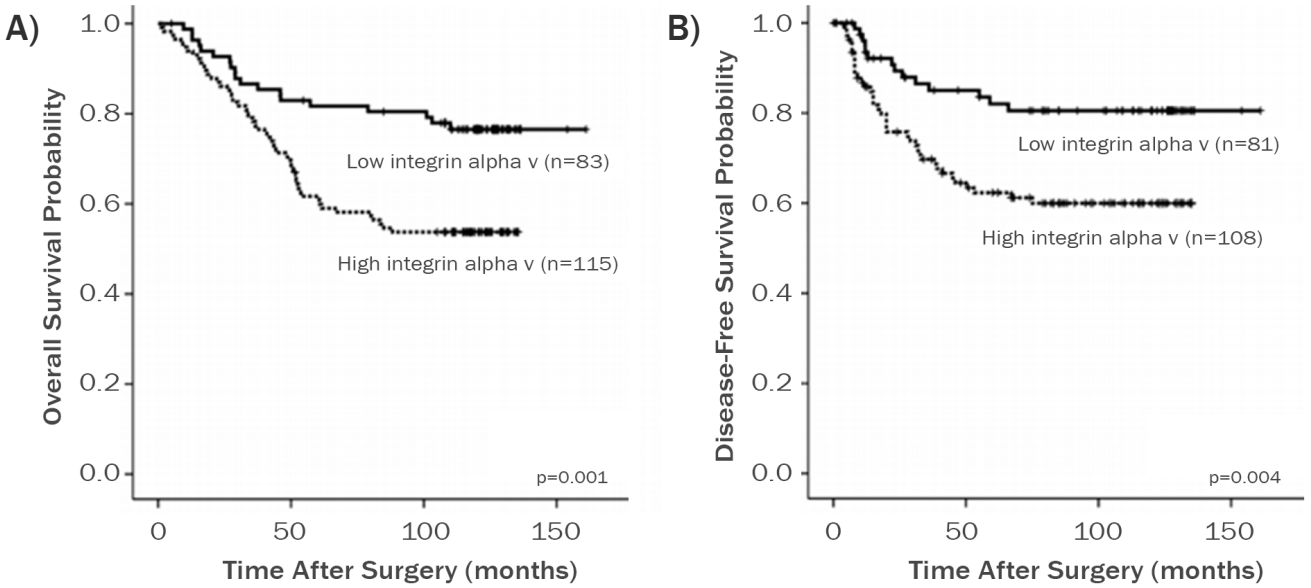
1. VIP236 is an $\alpha_V\beta_3$ integrin binder linked to an optCPT payload
2. Payload is released by the enzyme NE in the tumor microenvironment
3. The payload accumulates in the tumor cell due to high permeability and resistance to drug transporters
4. The payload inhibits topoisomerase 1 causing DNA damage and leading to cytotoxicity



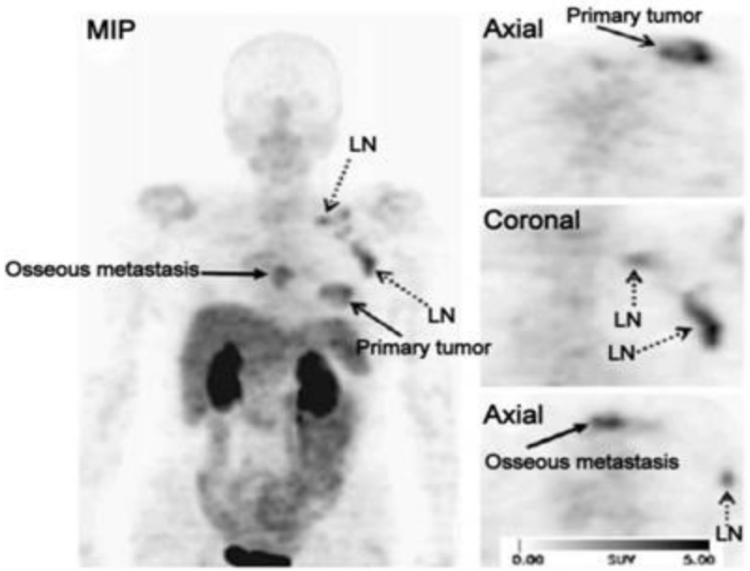
NE, neutrophil elastase.

$\alpha_v\beta_3$ Is a Target Across Solid Tumor Indications

Kaplan-Meier Survival Curves of Overall Survival (A) and Disease-Free Survival (B) According to Integrin a_v Expression Status in CRC



Imaging αvβ3 With Radiolabeled [^{18}F]Galacto-RGD Peptide in a Patient With Invasive Ductal Breast Cancer

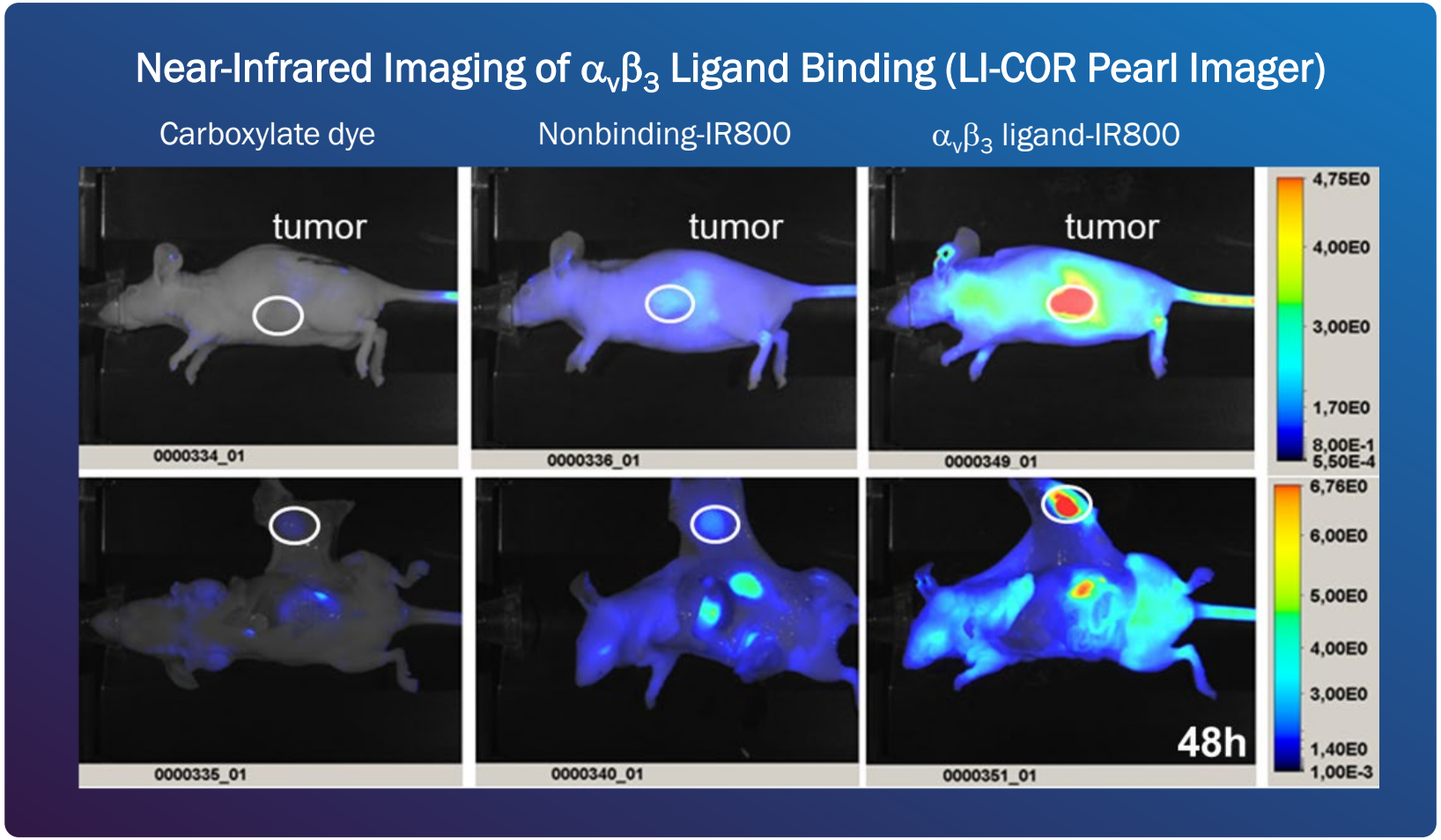


- $\alpha_v\beta_3$ is absent on resting endothelial cells and healthy organs
- High expression on activated endothelial cells and in advanced and metastatic tumors
- Expression correlates with poor prognosis in CRC and in other indications
- Anti-angiogenic therapies targeting $\alpha_v\beta_3$ showed good safety profile with optimal homing to the tumor and metastasis but with limited efficacy

CRC, colorectal cancer; LN, lymph node; MIP, maximum intensity projection; RGD, arginine-glycine-aspartate.

Ha SY, et al. *J Clin Pathol.* 2014;67(7):576-587.
Chen H, et al. *Theranostics.* 2016;6(1):78-92.

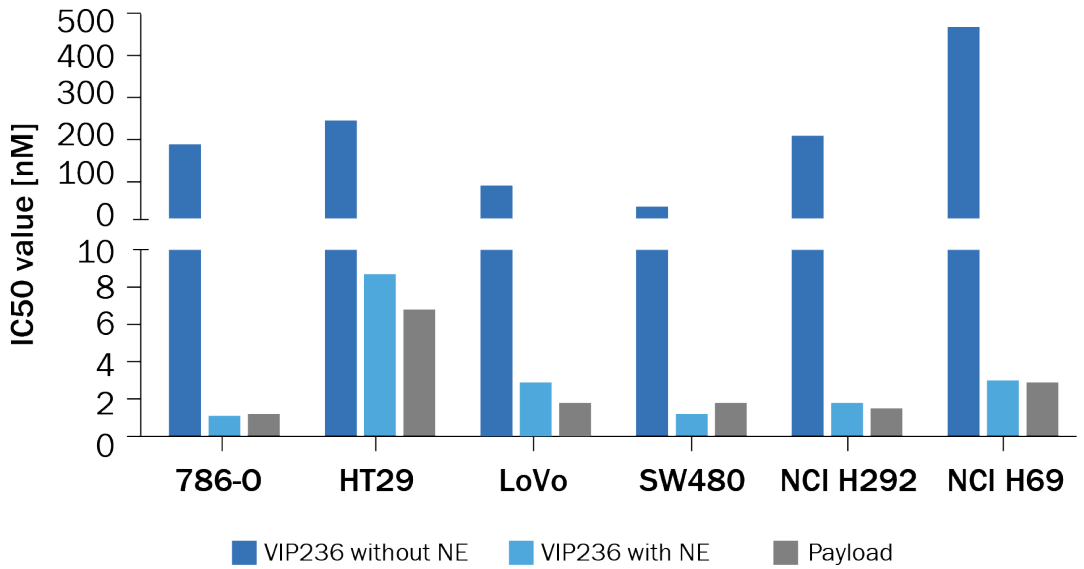
VIP236 Specifically Binds to Activated $\alpha_v\beta_3$ in the Tumor



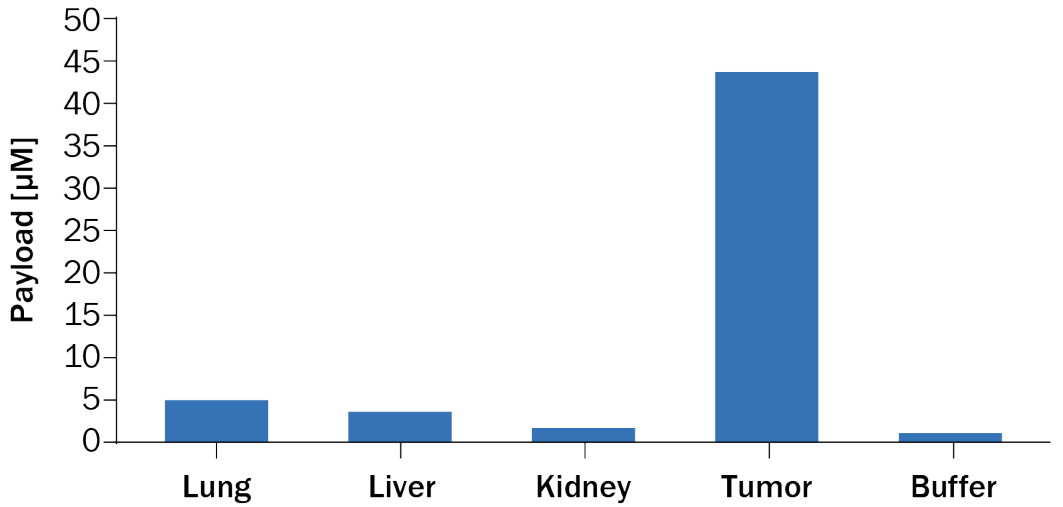
- Highly specific binding of VIP236 to activated $\alpha_v\beta_3$ in the tumor (>40-fold increase in signal as compared with other organs)
- In normal tissue, $\alpha_v\beta_3$ present in an inactive form (bend structure)

VIP236 Cytotoxicity Requires Tumor NE to Release Payload

VIP236 Cytotoxicity in Cell Lines Is NE Dependent

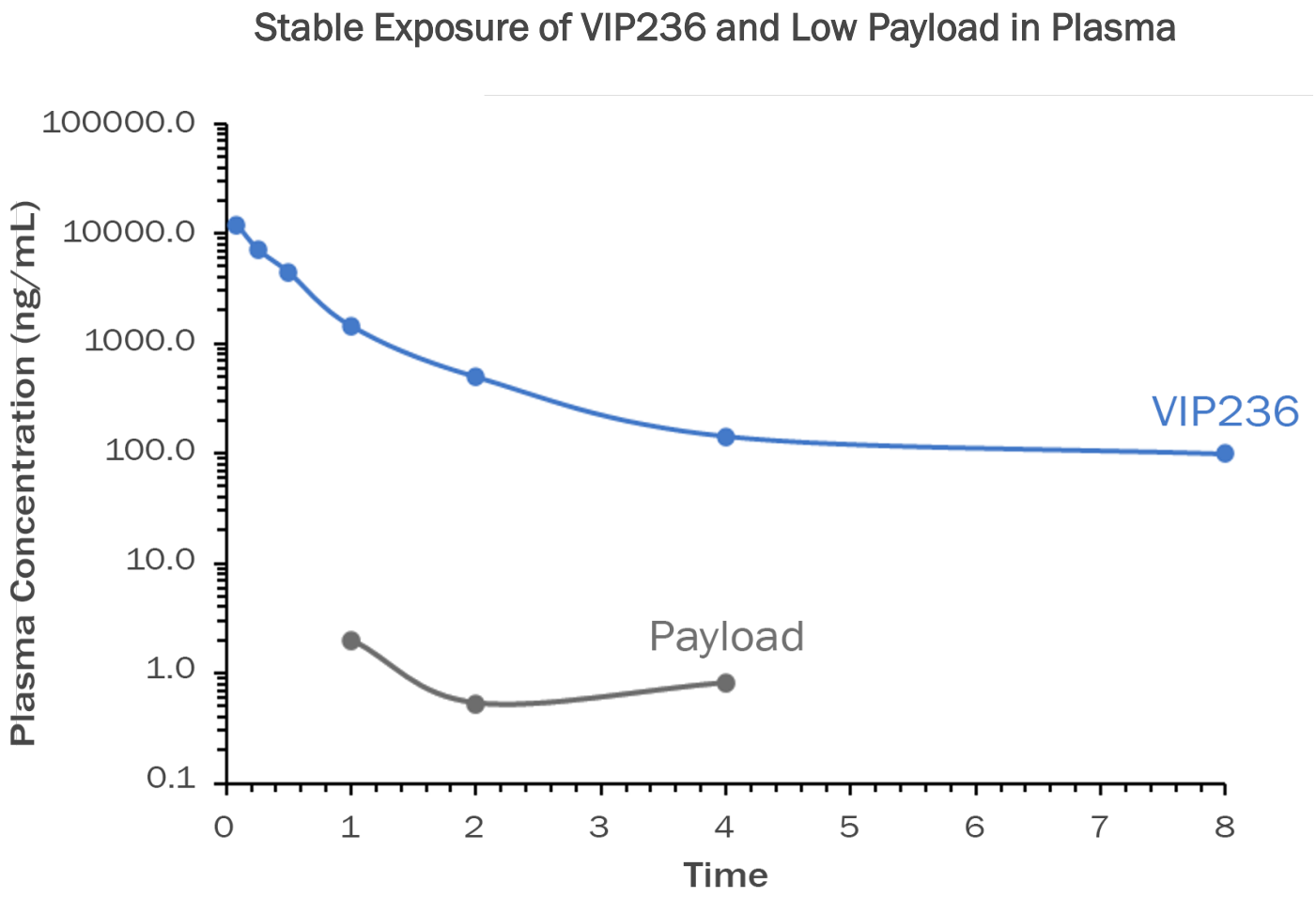


Payload Liberated by NE in Tumor Lysates



- Specific cleavage by NE is required for activation of the payload
- NE activation of payload does not interfere with achieving optimal cytotoxic activity of the payload
- Lysates derived from HCT 116 xenograft mouse model confirm the highly selective release of the payload in the tumor

VIP236 Is Highly Stable in Plasma With Low Exposure of Payload

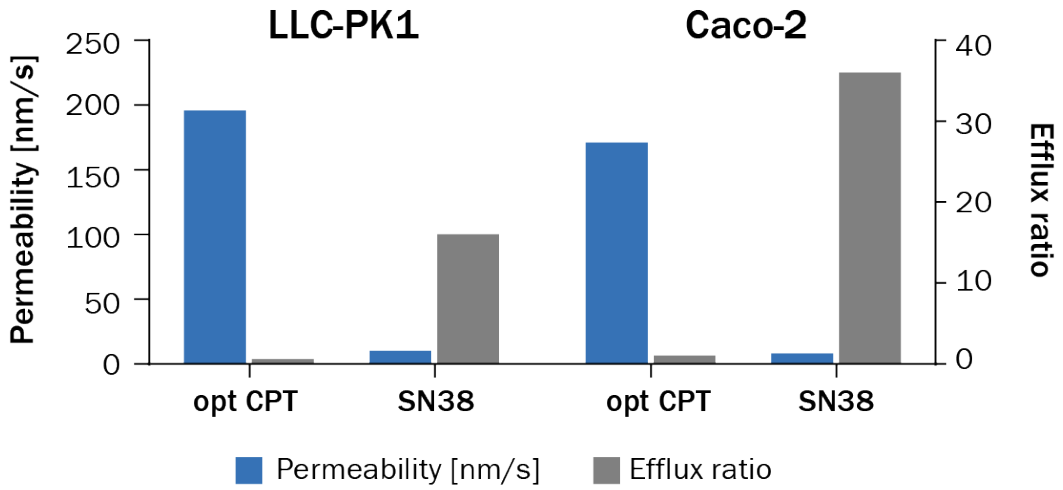


VIP236 (2mg/kg) single dose in mice

- Low exposure of the payload in plasma measured in PK analysis
- High stability in rat plasma of VIP236 with no degradation within 24h (data not shown)

OptCPT Payload Overcomes SN38 Transporter Efflux Liabilities

Permeability and Efflux Ratio With P-gp–Expressing LLC-PK1 and Caco-2 Cells



Cytotoxicity of optCPT and SN38 in NCI-H1975 Parental and P-gp or BCRP Transporter Overexpressing Cells

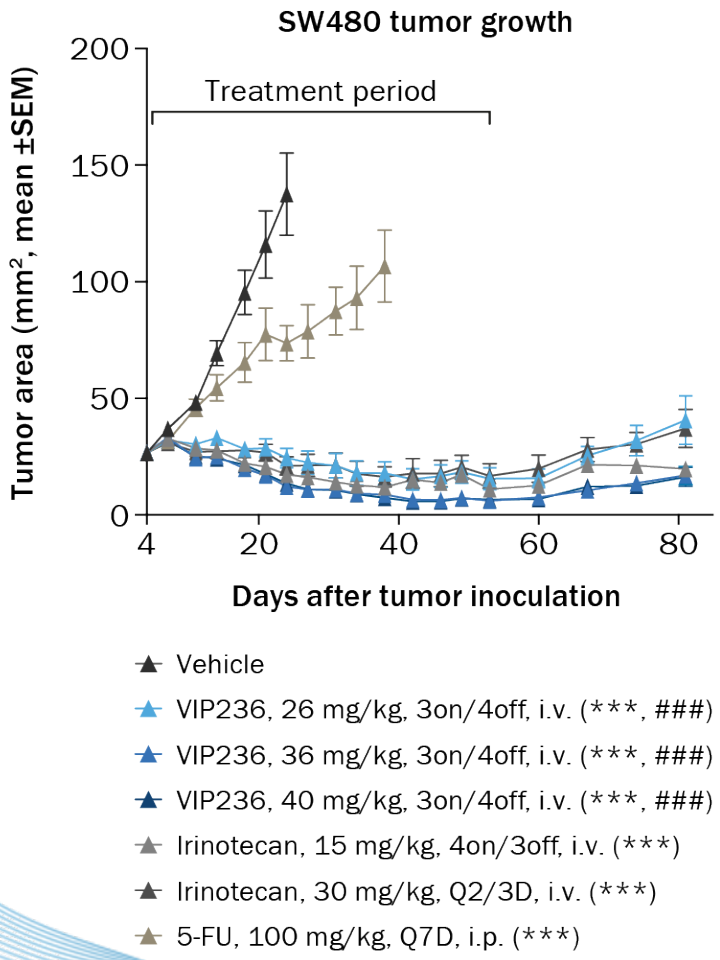
	IC50 (nM)		
Compound	NCI-H1975	NCI-H1975 — P-gp	NCI-H1975 — BCRP
SN38	45	141	512
OptCPT	19	34	27

- Payload: structurally related to the active metabolite of irinotecan known as SN38
- The payload of VIP236 is optimized for high permeability with low efflux potential to overcome transporter-mediated resistance observed with SN38
- The optCPT payload of VIP236 is not a P-gp or BCRP (ABCG2) transporter substrate showing no decreased cytotoxicity in transporter-expressing cell lines
  - In contrast, SN38 cytotoxicity decreases in transporter-expressing cell lines

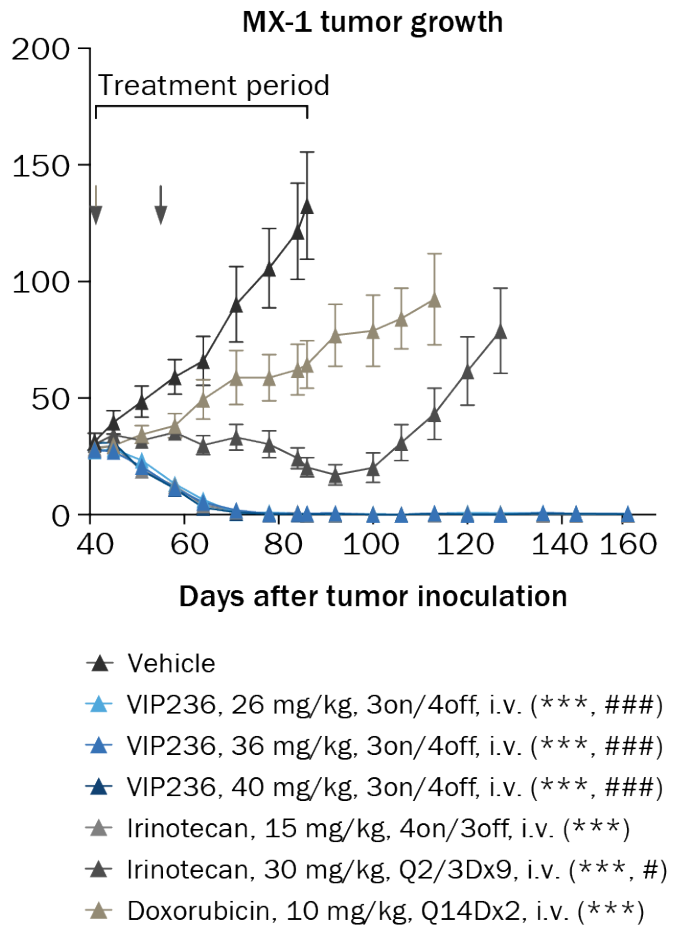
BCRP, breast cancer resistance protein; P-gp, P-glycoprotein

Significant Tumor Regression Observed Across Various Xenograft Models With VIP236 Treatment

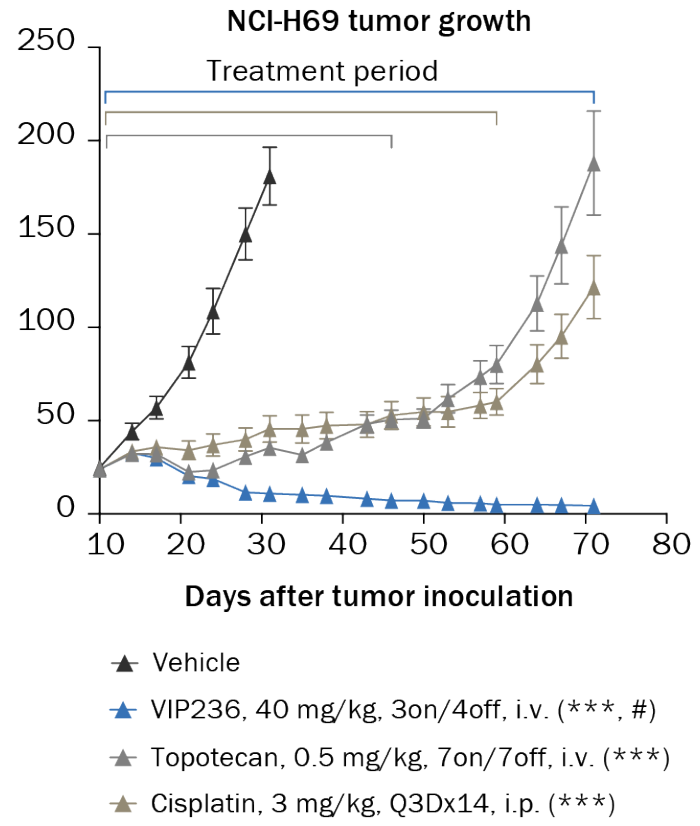
MSS CRC



TNBC



SCLC



5-FU, fluorouracil; MSS, microsatellite stable; SCLC, small cell lung cancer.
P value <0.05 flagged (*), *P*-value <0.01 (**), *P*-value <0.001 (***).
* compared with vehicle, # compared with 5-FU in SW480, doxorubicin in MX-1, and cisplatin in NCI H69 model.

VIP236 Potential Indications

HIGH EXPRESSION OF $\alpha_V\beta_3$ IS OBSERVED IN ADVANCED AND METASTATIC TUMORS AND CORRELATES WITH POOR PROGNOSIS

- Gastric and Colorectal Cancer
- Breast Cancer
- Lung Cancer Metastases
- Ovarian and Endometrial Cancer
- Sarcoma
- Renal Cell Carcinoma

Key Features of VIP236

SAFETY

TAILORED SMDC DESIGNED TO SELECTIVELY BIND TO TUMOR CELLS OF METASTATIC CANCERS

TARGET AND LINKER TECHNOLOGY DRIVES TUMOR SELECTIVITY AND PAYLOAD ACTIVATION

optCPT OVERCOMES SN38 TRANSPORTER EFFLUX LIABILITIES

EFFICACY

ACTIVATED $\alpha_v\beta_3$ IS HIGHLY EXPRESSED IN INVASIVE TUMORS

EXTRACELLULAR LINKER CLEAVAGE BY NE PROVIDES SELECTIVE ACTIVATION

10x GREATER DELIVERY OF PAYLOAD TO TUMOR

DURABLE TUMOR REGRESSIONS IN VIVO

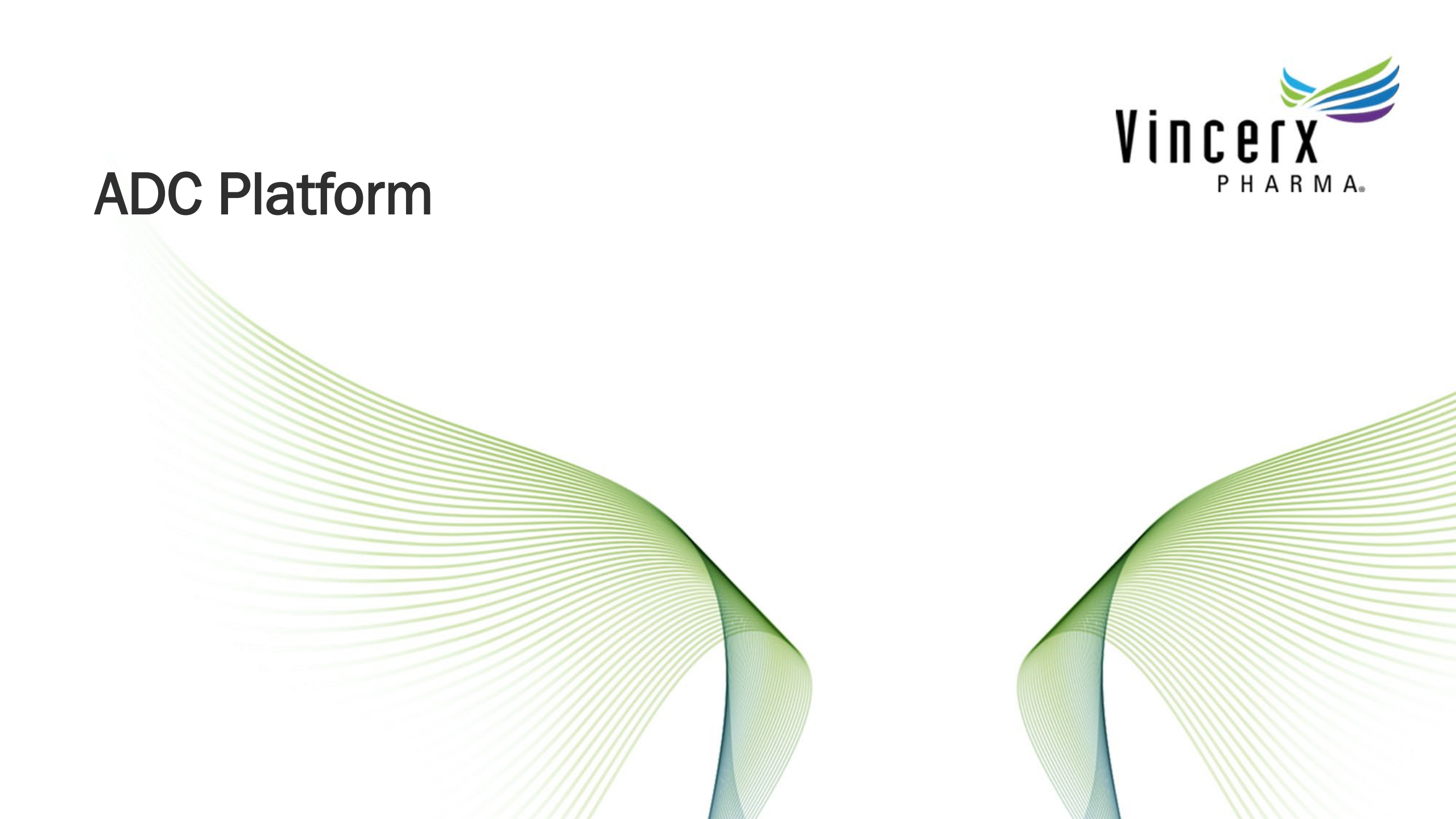
SIGNIFICANT REDUCTION OF METASTASES IN VIVO

COMMERCIAL POTENTIAL

PROVEN PAYLOAD CLASS IN CLINIC

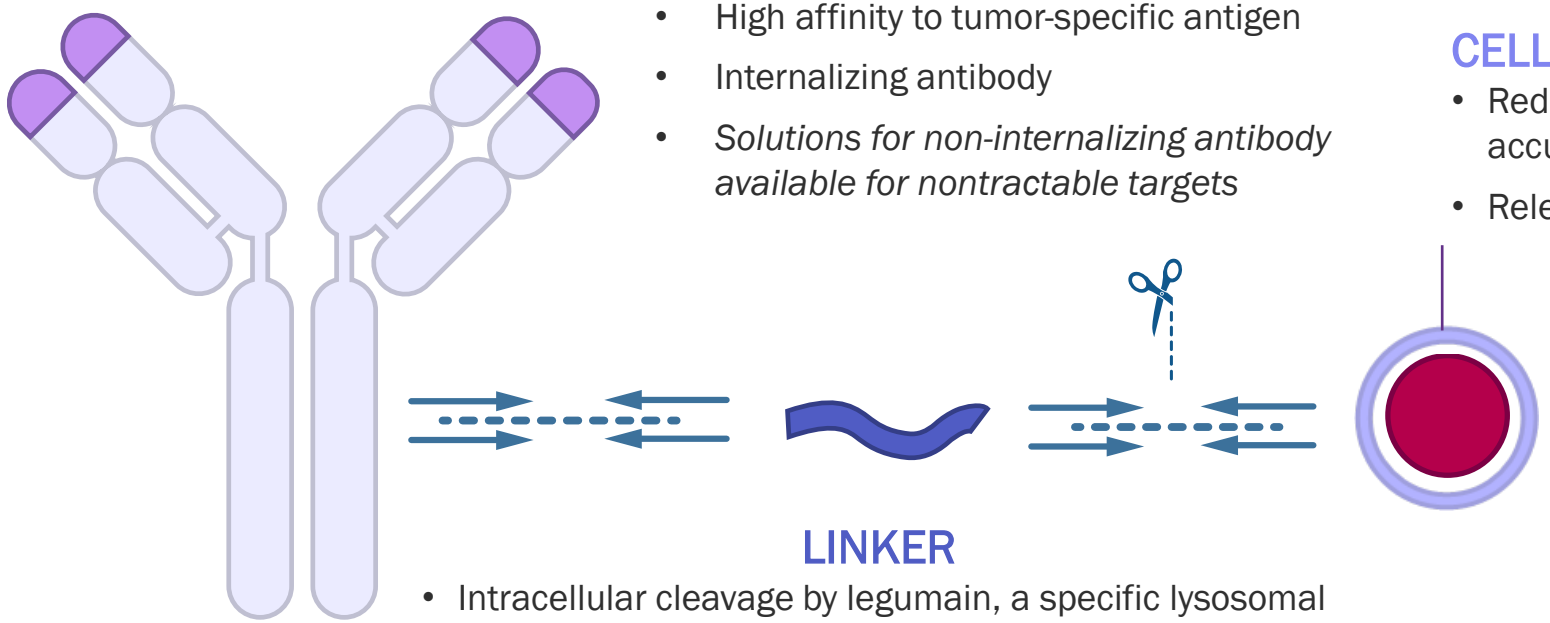
BROAD PATIENT POPULATION OPPORTUNITY IN MULTIPLE SOLID TUMOR INDICATIONS

ADC Platform



Solving ADC Problems With Innovative Technology

INCREASING THE THERAPEUTIC WINDOW BY IMPLEMETING ADDITIONAL SAFETY FEATURES



ANTIBODY

- High affinity to tumor-specific antigen
- Internalizing antibody
- *Solutions for non-internalizing antibody available for nontractable targets*

CELLTRAPPER™

- Reduced payload cell membrane permeability allows accumulation of payload in the tumor cell
- Released payload cannot enter healthy cells

LINKER

- Intracellular cleavage by legumain, a specific lysosomal protease avoiding premature cleavage
- Legumain is overexpressed in tumors vs normal cells and associated with poor prognosis
- *Modular technology with extracellular cleavage possible*
- *Site-specific or non–site-specific linker payload conjugation available for tunable DAR*

PAYLOAD

- KSPi, a novel, high-potency MoA payload specific for dividing cells
- Low/no toxicity in nondividing cells, no neurotoxicity
- Potential to induce immunogenic cell death
- *Alternative payloads/potency available*

Vincerx PHARMA

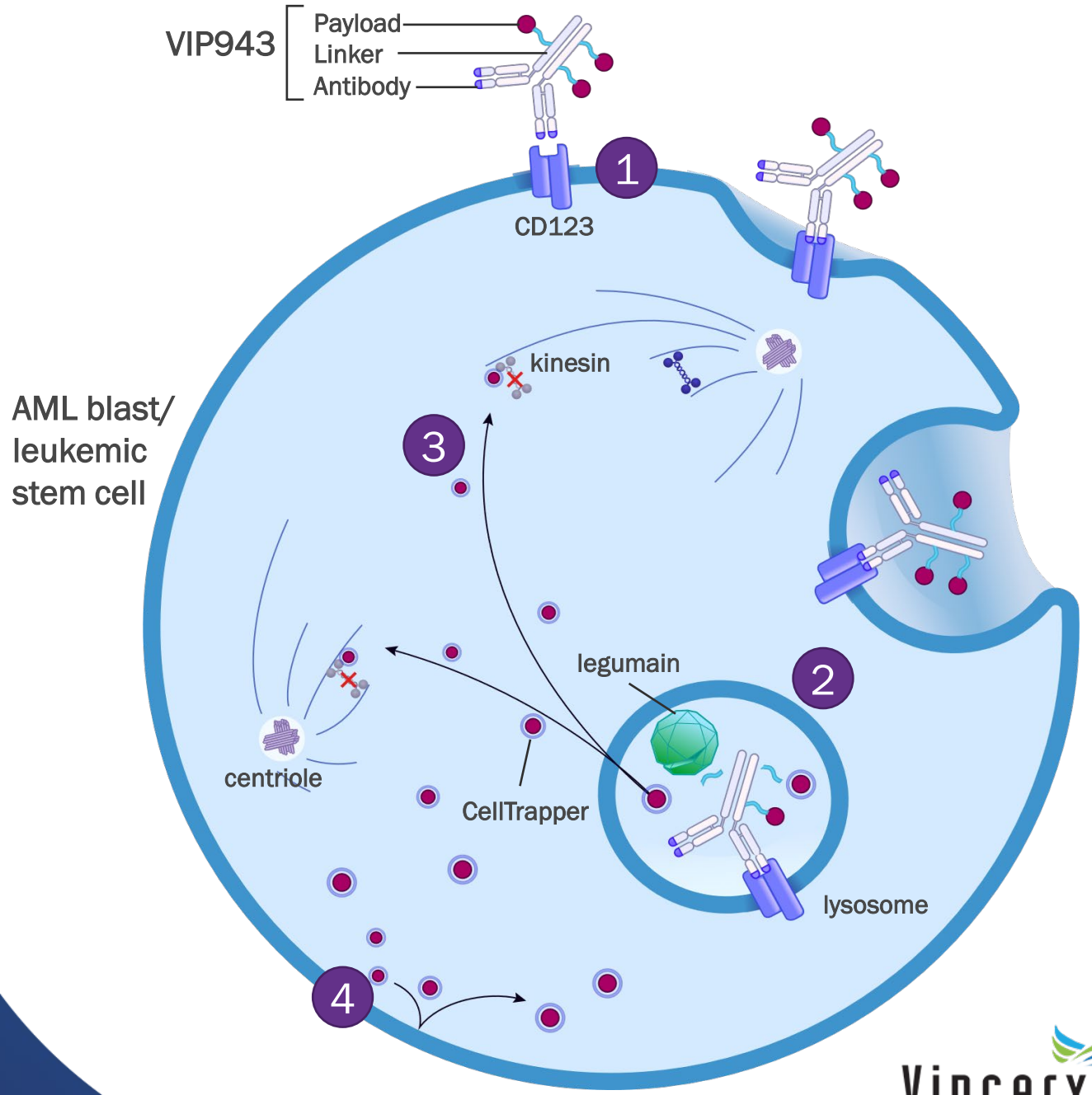
VIP943
CD123-KSPi ADC

Vincerx PHARMA

VIP943
CD123-KSPi

ANTIBODY-DRUG CONJUGATE FOR TREATMENT OF AML & MDS

1. CD123 is a validated target in myeloid malignancies and a potential leukemic stem cell target
2. VIP943-targeting Ab is internalized upon binding to CD123 linked to a legumain released KSPi
3. Payload targets KSP stopping cell division and causing catastrophic cell death
4. CellTrapper™ modified payload is hydrophilic and accumulates in the tumor cell for improved safety and tolerability for long-term therapy and targeting leukemic stem cells



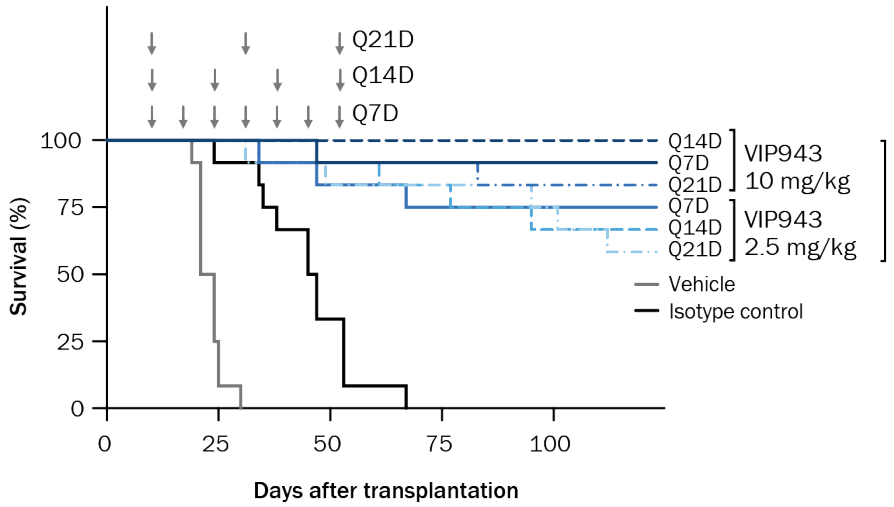
Vincerx PHARMA

Ab, antibody.

VIP943 Increases Survival in AML Models

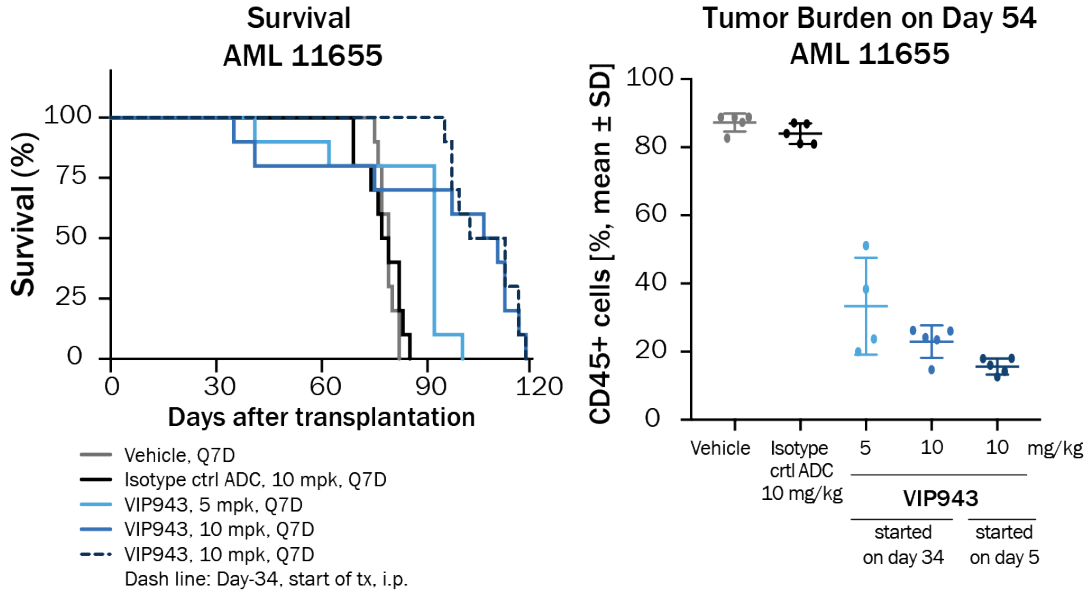
AML CELL-LINE (CDX) AND PATIENT-DERIVED (PDX) TUMOR MODELS TREATED WITH TARGETED ADC VS ISOTYPE CONTROL ADC

Striking Improved Survival in AML Model



- Increased survival in disseminated CD123+ AML CDX model MOLM-13, treated Q7Dx7
- Improved efficacy of targeted vs isotype control ADC

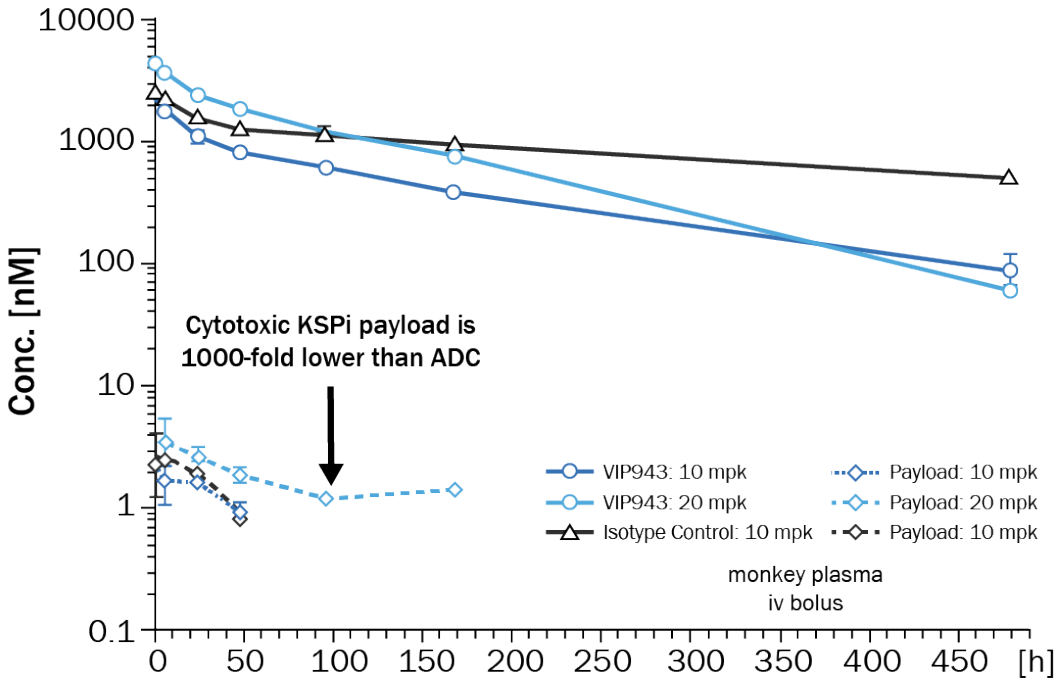
Improved Survival and Reduction in Tumor Burden in AML PDX Model



- Increased survival in disseminated CD123+ AML PDX model AML11655, treated Q7D
- Reduction of CD45+ AML tumor burden

VIP943 Favorable Safety in Monkey and Optimal DMPK Profile

DOSE LINEARITY AND HIGH ADC EXPOSURE; LOW PLASMA CONC. OF FREE PAYLOAD



DMPK In Vitro Profile
No CYP inhibition up to 20 µM • Low-risk DDI
No induction of CYP3A4 and CYP1A2 up to 30000 µg/L • Low-risk DDI
No payload metabolism found in plasma and in rat and human hepatocytes
Very low permeability in Caco-2 cell assay (2.7 nm/s) of intestinal permeability
No effect on hERG K^+ channel (IC50 > 10µmol/L)

VIP943 shows typical IgG PK profile by iv bolus:

- Low CL
- Low V_{ss}
- $T_{1/2} \geq 100$ h

VIP943 shows favorable toxicity profile:

- No VIP943-induced cytokine release observed
- Non-GLP single (20mg/kg) and repeated dose (10mg/kg,3 cycles) monkey toxicity study
- Well tolerated – No neutropenia, thrombocytopenia, mucositis, or liver toxicity

CL, clearance; CYP, cytochrome P450; DDI, drug-drug interaction; DMPK, drug metabolism and pharmacokinetics; GLP, good laboratory practices; IgG, immunoglobulin G; $T_{1/2}$, half life; Vss, volume of distribution.

VIP943 Is Designed to Address Safety Liabilities of ADCs Approved in Hematologic Malignancies

	MYLOTARG™	BESPONSA®	POLIVY™	ADCETRIS®	VIP943		
PRECLINICAL TARGET ORGAN TOXICITY					Cynomolgus Macaque		
Bone Marrow/Lymph Nodes	+	+	+	+	Not observed		
Liver	+	+	+	+	Not observed		
CLINICAL TRIAL SEVERE ADVERSE EVENTS					Linker	KSPi	CELLTRAPPER™
Myelosuppression		++	++	++	✓	✓	✓
Infections/PML			++	+++	✓	✓	✓
Hepatotoxicity/VOD	+++	+++	++	++	✓		✓
Peripheral Neuropathy			++	++		✓	

+: Present
++: Warnings & precautions
+++: Black box warning
✓: Designed to address AEs

PML, progressive multifocal leukoencephalopathy; VOD, veno-occlusive disease.

Source: Drugs@FDA

Vincerx PHARMA

VIP943 Potential Indications

CD123 IS A VALIDATED TARGET IN MYELOID MALIGNANCIES INCLUDING LEUKEMIC STEM CELLS

- High-Risk, Elderly AML
- Secondary AML
- Myelodysplastic Syndrome
- AML With TP53 Mutations
- Combination With Venetoclax in Second-Line AML
- Combination With Azacitidine and Venetoclax in Front-Line AML

Key Features of VIP943

SAFETY

HIGHLY FAVORABLE SAFETY PROFILE IN MONKEY TOXICITY STUDIES

NO NEUTROPENIA, THROMBOCYTOPENIA, MUCOSITIS, OR LIVER TOXICITY FINDINGS IN MONKEY TOXICITY

LINEAR PK PROFILE WITH LOW FREE PAYLOAD EXPOSURE IN PLASMA

EFFICACY

BEST-IN-CLASS ADC, USING KSPi AS NOVEL PAYLOAD

ABSENCE OF MYELOSUPPRESSION SUPPORTS DEVELOPMENT IN AML/MDS

DOSE-DEPENDENT TUMOR REDUCTION OBSERVED IN SEVERAL IN VIVO MODELS

COMMERCIAL POTENTIAL

OPPORTUNITY FOR IMPROVEMENT IN SAFETY AND EFFICACY OVER CURRENT SOC

POTENTIAL AS MONOTHERAPY OR AS PREFERRED COMBINATION PARTNER

OPPORTUNITY FOR ACCELERATED APPROVAL

POTENTIAL FOR FIRST-LINE TREATMENT IN AML AND MDS

SOC, standard of care.

VIP924
CXCR5-KSPi ADC

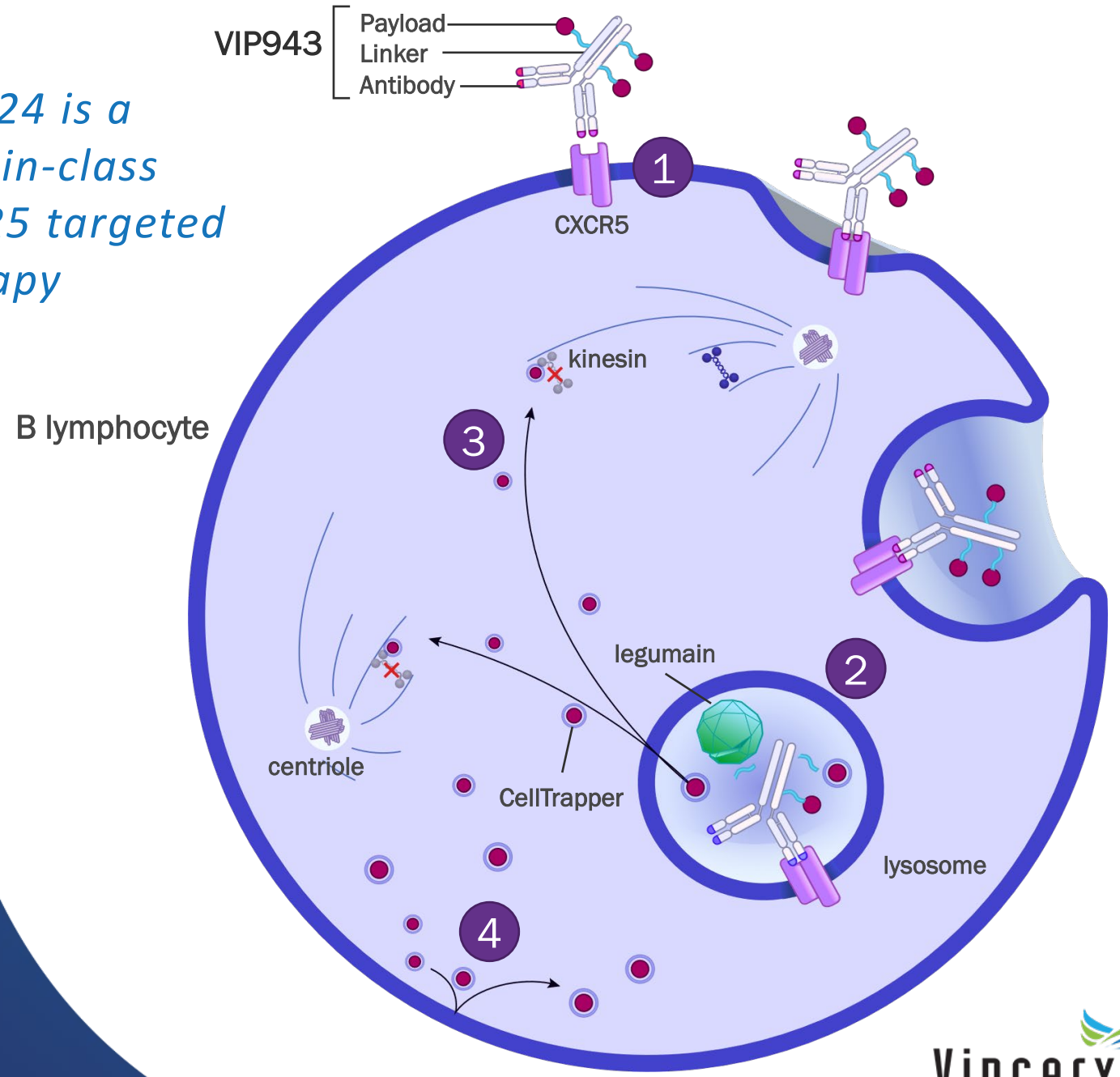
Vincerx PHARMA

VIP924
CXCR5-KSPi

FOR TREATMENT OF B-CELL MALIGNANCIES

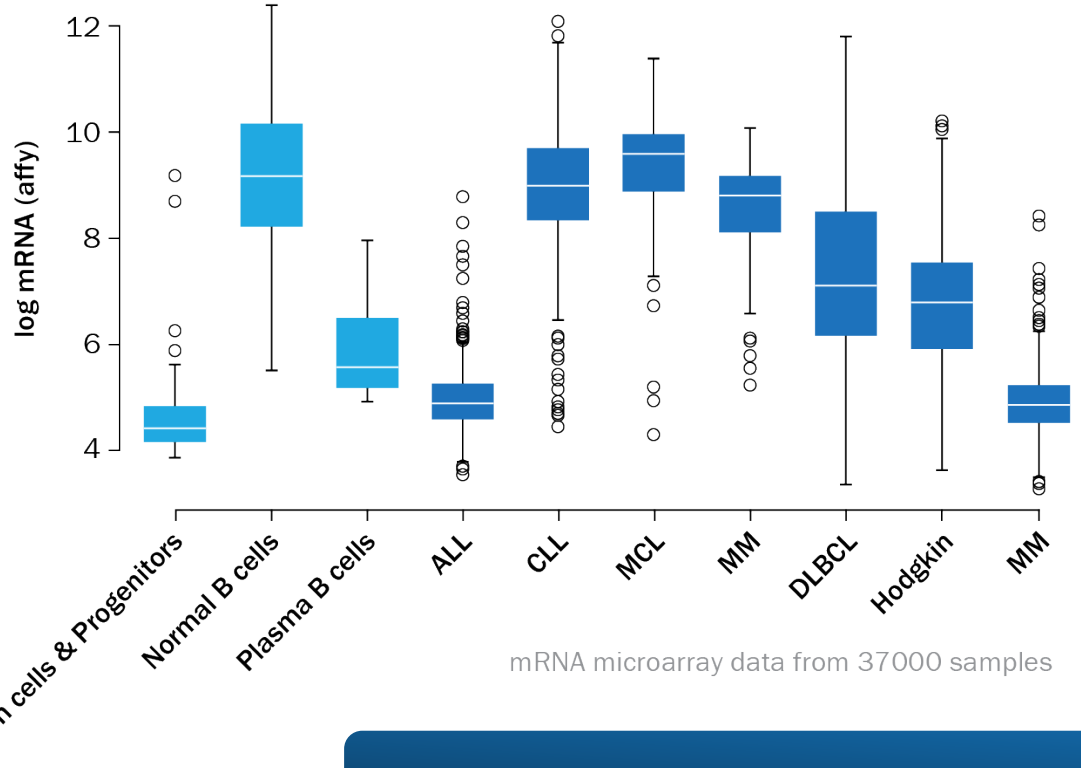
VIP924 is a first-in-class CXCR5 targeted therapy

1. CXCR5 regulates chemotaxis, germinal center formation, and plasma and memory B-cell differentiation
2. VIP924 has an internalizing Ab upon binding to CXCR5 which is linked to a legumain released KSPi that drives cell death during cell division
3. Payload targets KSP stopping cell division and causing catastrophic cell death
4. CellTrapper™ modified payload is hydrophilic and accumulates in the tumor cell for improved safety and tolerability for long-term treatment of B-cell malignancies



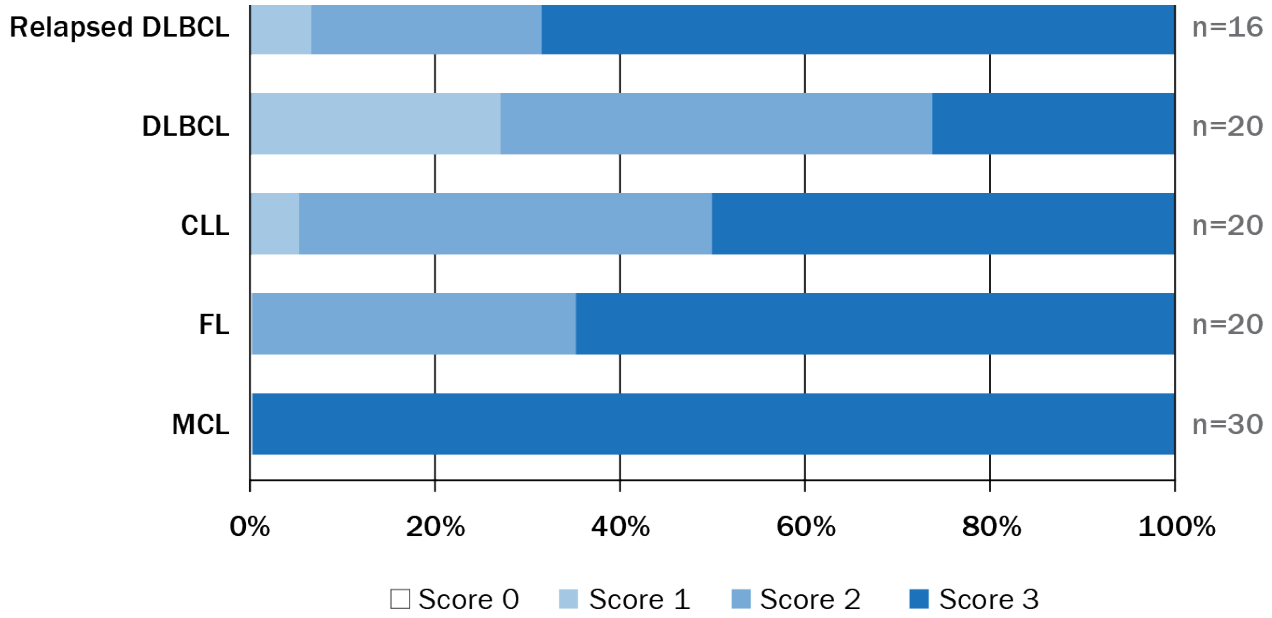
CXCR5 Is Expressed in B-Cell Malignancies

High CXCR5 mRNA Across NHL



mRNA microarray data from 37000 samples

CXCR5 IHC Staining Is Present in 16/16 Relapsed DLBCL Samples (Post–R-CHOP therapy)

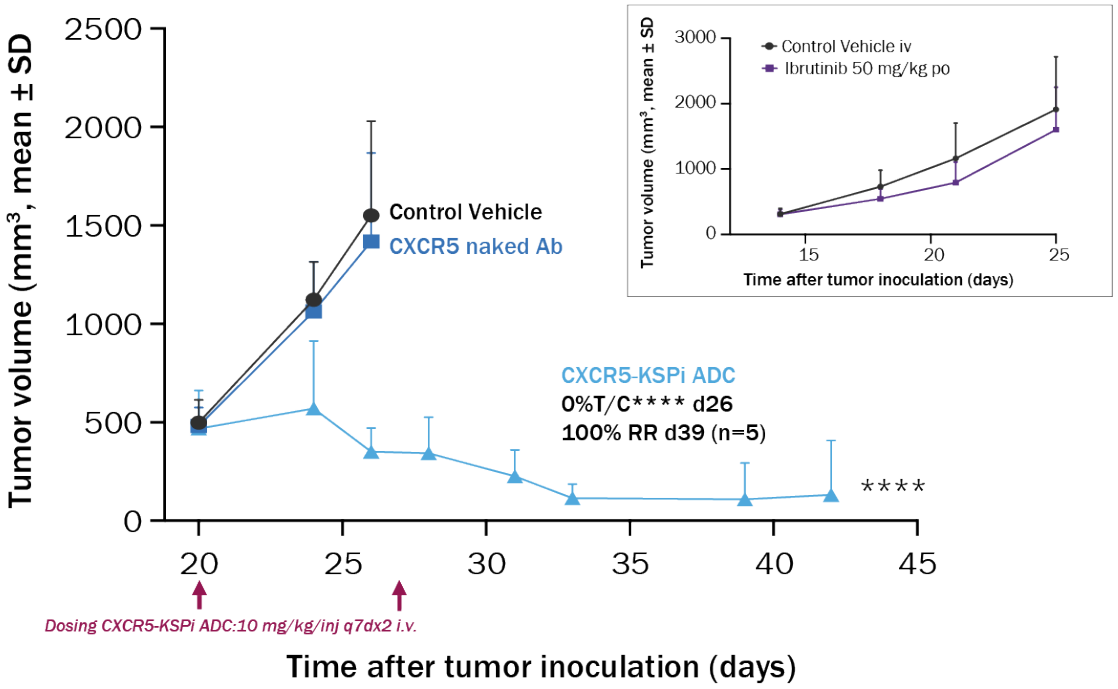


Potential indications for VIP924 are MCL, DLBCL, FL, and CLL

IHC, immunohistochemistry; MM, multiple myeloma.

VIP924 Induces Sustained Tumor Regression in MCL and DLBCL Models

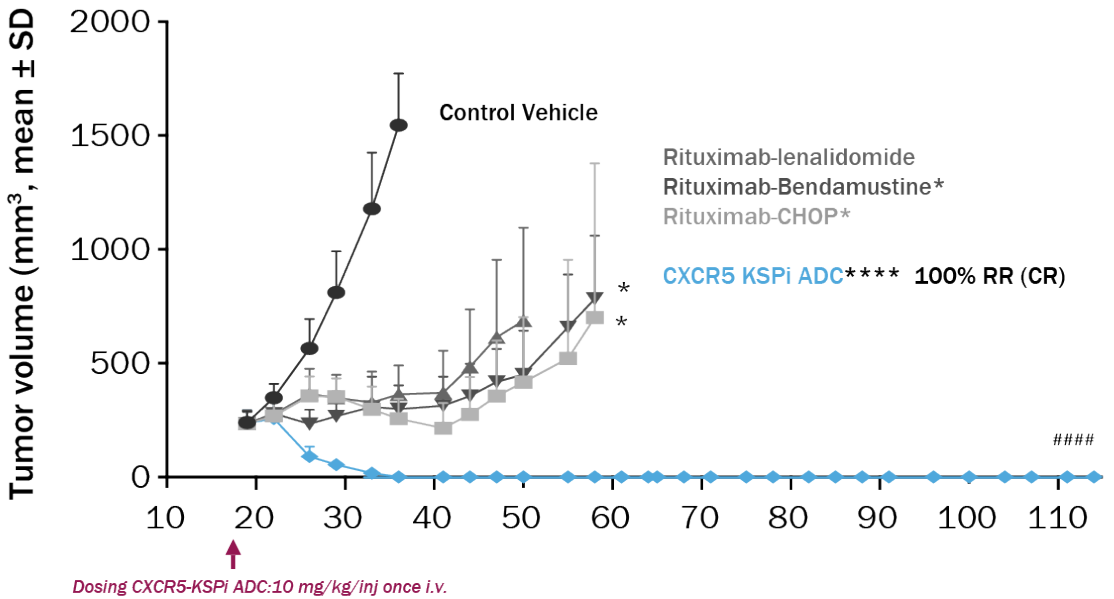
VIP924 Is Active in Ibrutinib-Refractory MCL In Vivo Model



- Ibrutinib-refractory MCL CDX CXCR5+ REC-1 model (inset)
- VIP924 achieved complete remission after 2 doses

****P=0.0001 vs vehicle one-way ANOVA, Dunnett-method on Log transformed tumor volumes on day 26.

Single Dose of VIP924 in DLBCL In Vivo Model Achieved Durable Complete Regressions



- Complete regression with single dose of VIP924 in CXCR5+ model OCI-LY1 (day 114)
- Superior activity versus SOC

*P<0.05. ****P=0.0001 vs vehicle. ####P<0.0001 vs rituximab-bendamustine/ lenalidomide or CHOP. One-way ANOVA, Tuckey-method on Log transformed tumor volumes on day 36. RR, response rate.

VIP924 Potential Indications

CXCR5 IS EXPRESSED IN B-CELL MALIGNANCIES

- Diffuse Large B-Cell Lymphoma
- Chronic Lymphocytic Leukemia
- Follicular Lymphoma
- Mantle Cell Lymphoma

Key Features of VIP924

SAFETY

SURROGATE DATA FROM VIP943, WITH SAME LINKER PAYLOAD, SUGGEST A HIGHLY FAVORABLE SAFETY PROFILE WITHOUT NEUTROPENIA, THROMBOCYTOPENIA, MUCOSITIS, OR LIVER TOXICITY

EFFICACY

CXCR5 IS A NOVEL B-CELL MALIGNANCY TARGET

UTILIZES A KSPi AS A PROPRIETARY PAYLOAD WITH CELLTRAPPER™ MOIETY

COMPLETE REMISSION IN CXCR5+ LYMPHOMA IN VIVO MODELS INCLUDING IBRUTINIB-REFRACTORY MCL AND DLBCL

COMMERCIAL POTENTIAL

OPPORTUNITY FOR IMPROVEMENT IN SAFETY AND EFFICACY OVER CURRENT SOC

POTENTIAL AS MONOTHERAPY OR AS PREFERRED COMBINATION PARTNER

OPPORTUNITY FOR ACCELERATED APPROVAL POTENTIAL FOR FIRST-LINE TREATMENT IN B-CELL MALIGNANCIES

Expected Upcoming Milestones

Vincerx PHARMA®

A STRONG MANAGEMENT TEAM WITH A PROVEN TRACK RECORD OF SUCCESSES

HIGHLY EXPERIENCED IN VALUE CREATION BY DELIVERING MULTIPLE APPROVALS AND SUCCESSFUL M&As

>20 YEARS OF EXPERIENCE IN CDK9 SPACE

>15 YEARS OF ADC DEVELOPMENT EXPERIENCE FROM DISCOVERY TO CLINICAL DEVELOPMENT

PHASE 1 ASSET WITH PROOF OF CONCEPT IN MULTIPLE INDICATIONS

ENITOCICLIB WITH SINGLE-AGENT DURABLE REMISSIONS IN AGGRESSIVE DH-DLBCL

ACCELERATED APPROVAL OPPORTUNITIES AS A POTENTIAL BEST-IN-CLASS MONOTHERAPY

SAFETY PROFILE WILL SUPPORT FUTURE COMBINATION STUDIES

PHASE 2 STUDIES IN 2023

INNOVATIVE, NEXT-GENERATION BIOCONJUGATION PLATFORM

MODULAR TECHNOLOGY DESIGNED TO ADDRESS CHALLENGES OF APPROVED ADCs

PROPRIETARY LINKER PAYLOAD TECHNOLOGY REPRESENT POTENTIAL FIRST-IN-CLASS AND BEST-IN-CLASS OPPORTUNITIES

VIP236 IND EXPECTED IN 2H2022, VIP943 AND VIP924 INDs EXPECTED IN 2H2023 AND 1H2024

M&As, mergers and acquisitions.