

# CORPORATE OVERVIEW

April 2024



# Safe Harbor Statement & Trademarks

## CAUTIONARY STATEMENT

No representations or warranties, expressed or implied are given in, or in respect of, this presentation. To the fullest extent permitted by law, in no circumstances will Vincerx Pharma, Inc. (“Vincerx” or the “Company”) or any of its subsidiaries, stockholders, affiliates, representatives, partners, directors, officers, employees, advisers or agents be responsible or liable for any direct, indirect or consequential loss or loss of profit arising from the use of this presentation, its contents, its omissions, reliance on the information contained within it, or on opinions communicated in relation thereto or otherwise arising in connection therewith. Industry and market data used in this presentation have been obtained from third-party industry publications and sources as well as from research reports prepared for other purposes. Vincerx has not independently verified the data obtained from these sources and cannot assure you of the data’s accuracy or completeness. This data is subject to change. In addition, this presentation does not purport to be all-inclusive or to contain all of the information that may be required to make a full analysis of Vincerx. Viewers of this presentation should each make their own evaluation of Vincerx and of the relevance and adequacy of the information and should make such other investigations as they deem necessary.

This presentation contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended, that are intended to be covered by the “safe harbor” created by those sections. Forward-looking statements, which are based on certain assumptions and describe future plans, strategies, expectations and events, can generally be identified by the use of forward-looking terms such as “believe,” “expect,” “may,” “will,” “should,” “would,” “could,” “suggest,” “scheduled,” “seek,” “intend,” “plan,” “goal,” “potential,” “on-target,” “on track,” “project,” “estimate,” “anticipate,” or other comparable terms. All statements other than statements of historical facts included in this presentation are forward-looking statements. Forward-looking statements include, but are not limited to: Vincerx’s business model, pipeline, strategy, timeline, product candidates and attributes, and preclinical and clinical development, timing, and results. Forward-looking statements are neither historical facts nor assurances of future performance or events. Instead, they are based only on current beliefs, expectations, and assumptions regarding future business developments, future plans and strategies, projections, anticipated events and trends, the economy, and other future conditions. Forward-looking statements are subject to inherent uncertainties, risks, and changes in circumstances that are difficult to predict and many of which are outside of Vincerx’s control.

Actual results, conditions, and events may differ materially from those indicated in the forward-looking statements. Therefore, you should not rely on any of these forward-looking statements. Important factors that could cause actual results, conditions, and events to differ materially from those indicated in the forward-looking statements include, but are not limited to: general economic, financial, legal, political, and business conditions; risks associated with preclinical or clinical development and trials, including those conducted prior to Vincerx’s in-licensing; failure to realize the benefits of Vincerx’s license agreement with Bayer; risks related to the rollout of Vincerx’s business and the timing of expected business and product development milestones; changes in the assumptions underlying Vincerx’s expectations regarding its future business or business model; Vincerx’s ability to successfully develop and commercialize product candidates; Vincerx’s capital requirements, availability and uses of capital, and cash runway; and the risks and uncertainties set forth in Forms 10-K, 10-Q, and 8-K most recently filed with or furnished to the Securities and Exchange Commission by Vincerx. Forward-looking statements speak only as of the date hereof, and Vincerx disclaims any obligation to update any forward-looking statements.

## TRADEMARKS

Vincerx<sup>®</sup>, Vincerx Pharma<sup>®</sup>, the Vincerx Wings logo design, VersAptx<sup>™</sup> and CellTrapper<sup>®</sup> are trademarks or registered trademarks of the Company. This presentation may also contain trademarks and trade names of other companies, which are the property of their respective owners.



# OUR VISION

## WE ASPIRE TO CONQUER CANCER

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



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



**VersAptx™ NEXT-GENERATION  
PLATFORM** TO BIOCONJUGATE  
UNIQUE ADCs, SMDCs AND  
DELIVER ON THE PROMISE OF  
DRUG CONJUGATES



**R&D STRATEGY** STREAMLINED  
RESEARCH AND DEVELOPMENT  
FROM PRECLINICAL TO CLINICAL  
PROOF-OF-CONCEPT



**DIVERSE PIPELINE** WITH  
MULTIPLE CLINICAL FIRST-IN-  
AND BEST-IN-CLASS  
OPPORTUNITIES

# Seasoned Management Team



Ahmed Hamdy

CEO



Raquel Izumi

COO



Steven Bloom

CBO



Hans-Georg Lerchen

CSO



Alex Seelenberger

CFO



Beatrix Stelte-Ludwig

CDO



Tom Thomas

CLO



ZIOPHARM Oncology

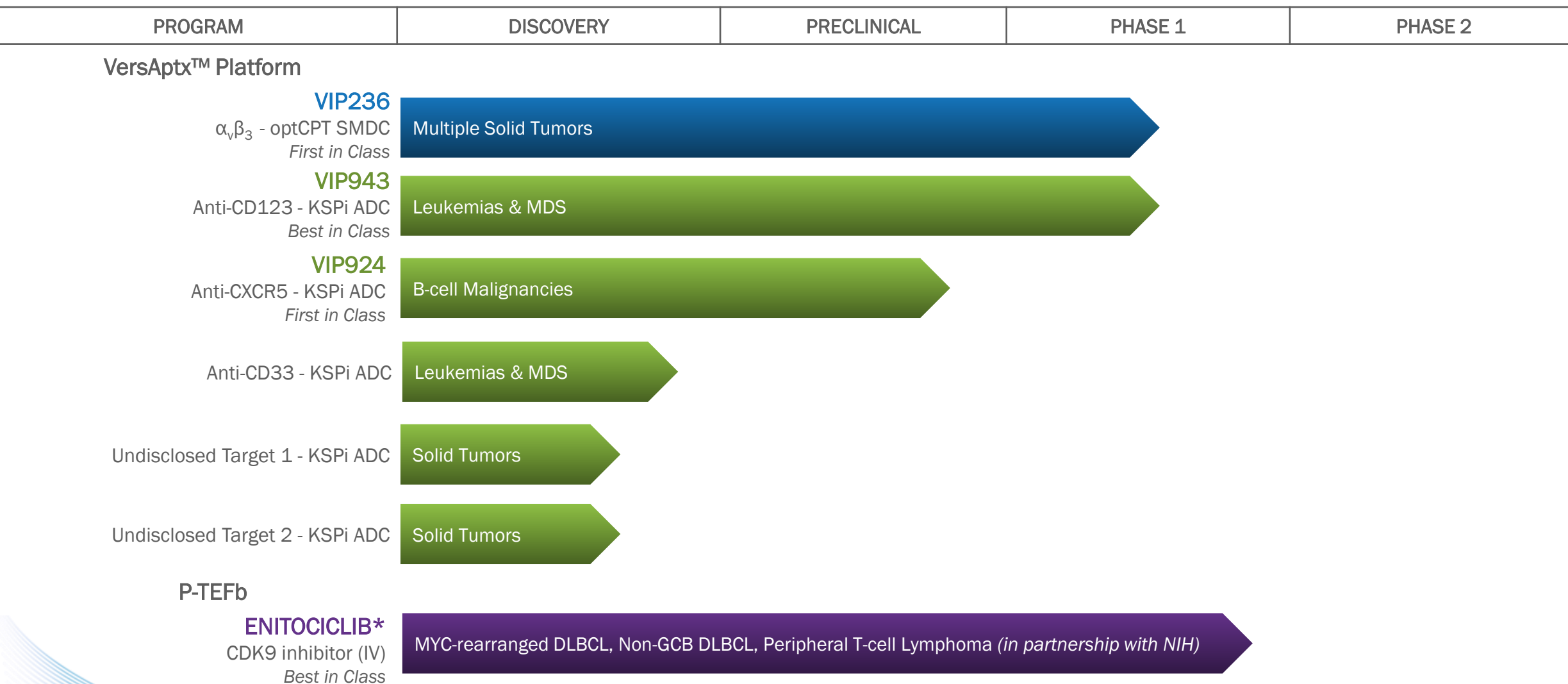


**\$975M partnership with Janssen in 2011 (\$150 up front, \$825M in milestones)**  
**\$21B acquisition of Pharmacyclics by AbbVie in 2015**  
**Management Team's Contribution:** Developed ibrutinib from preclinical through phase 2 in <3 years. All 3 phase 2 studies garnered break through therapy designation and accelerated approvals



**\$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

# Our Diverse Pipeline



\*Also known as VIP152.

ADC, antibody-drug conjugate; CDK, cyclin-dependent kinase; DLBCL, diffuse large B-cell Lymphoma; GCB, germinal center B-cell; 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.

# Demonstrating the Potential of the VersAptx™ Platform

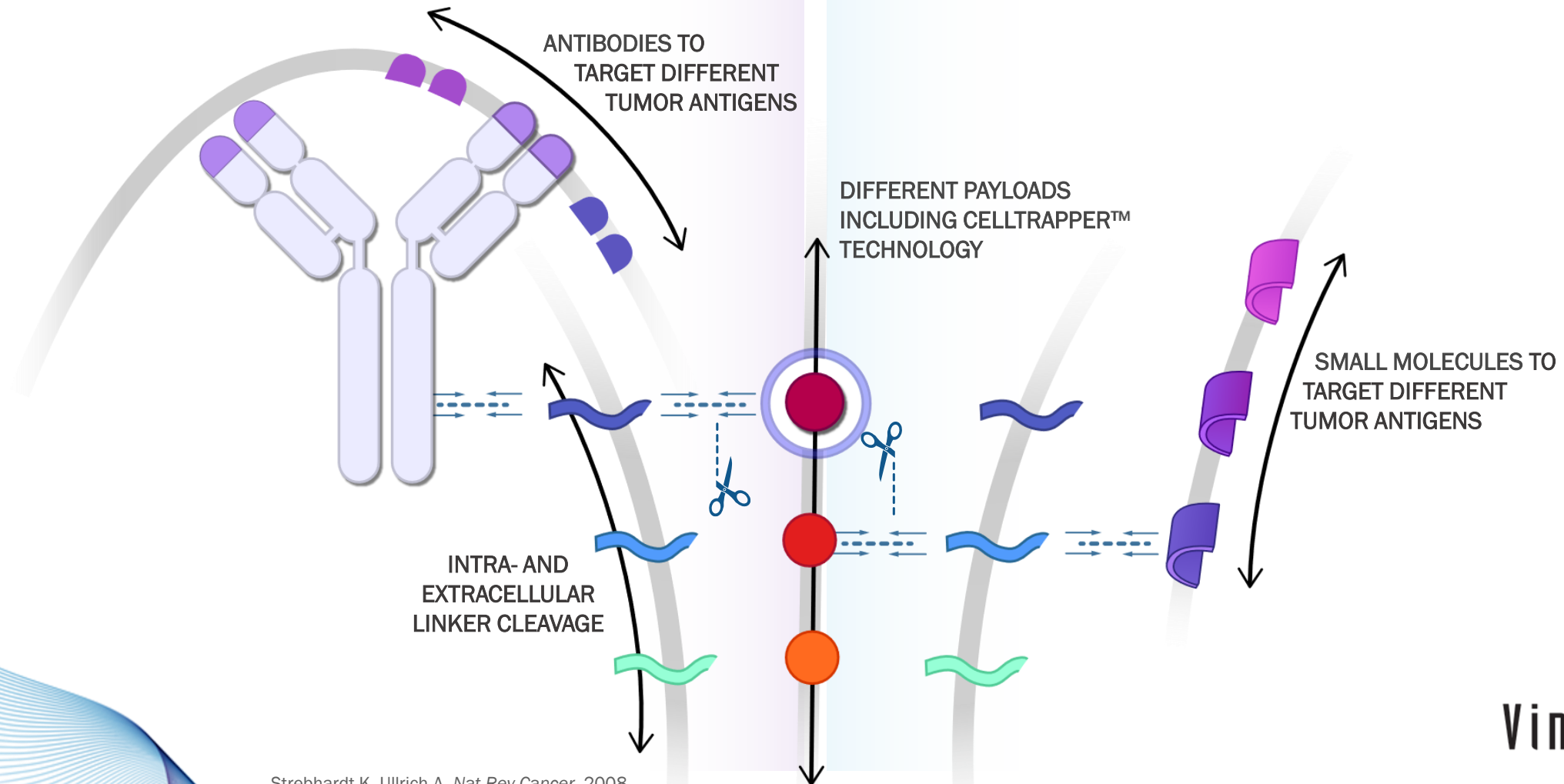


# VersAptx Platform™: A Versatile and Adaptable, Next-Generation Bioconjugation Platform

COMBINING DIFFERENT TARGETING, LINKER, AND PAYLOAD TECHNOLOGIES TO ADDRESS ALL CANCER BIOLOGIES

## Antibody Drug Conjugate

## Small Molecule Drug Conjugate



Strebhardt K, Ullrich A. *Nat Rev Cancer*. 2008.

# Solving ADC Challenges With Our Innovative VersAptx™ Platform

## INCREASING THE THERAPEUTIC WINDOW BY IMPROVING EFFICACY AND SAFETY

### Known ADC Challenges

Cell-permeable DNA- damaging payloads or microtubule inhibitors affect non-dividing, non-target cells

Premature release of cytotoxic payloads

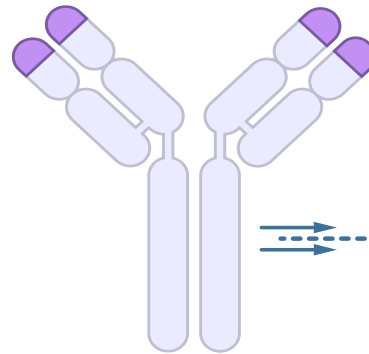
ADC aggregation and unspecific cellular uptake driven by hydrophobic payloads

Leading to severe side effects like myelosuppression, infections, peripheral neuropathy, hepatotoxicity, and others

### Vincerx Design Solutions

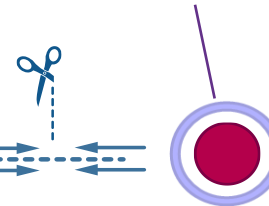
#### ANTIBODY

- High affinity to tumor-specific antigen
- Internalizing antibody



#### CELLTRAPPER®

- Reduced payload cell membrane permeability
- Released payload cannot enter healthy cells



#### LINKER

- Intracellular cleavage exclusively by legumain, a specific lysosomal protease overexpressed in tumors
- Site-specific or non-site-specific conjugation available

#### PAYLOAD

- KSPi, a novel, high-potency MoA payload specific for dividing cells

### Benefits

#### Legumain Linker

- Unique cleavage sequence post Asn (no unspecific cleavage)
- Second level of tumor targeting via specific ADC activation

#### KSPi payload + Cell Trapper

- High potency and novel MoA
- Low/no toxicity in nondividing cells, no neurotoxicity

Intracellular accumulation of the payload, no diffusion into non-target cells and long-lasting tumor accumulation of the payload

Flexibility, compatible with different linker designs

#### Hydrophilic linker-payload

- Efficacy: Allows for high DAR without affecting PK
- Safety: No side effects associated with aggregation



# CLINICAL FIRST-IN- AND BEST-IN- CLASS OPPORTUNITIES



# VIP236

$A_v\beta_3$  - Optimized Camptothecin  
Small Molecule Drug Conjugate

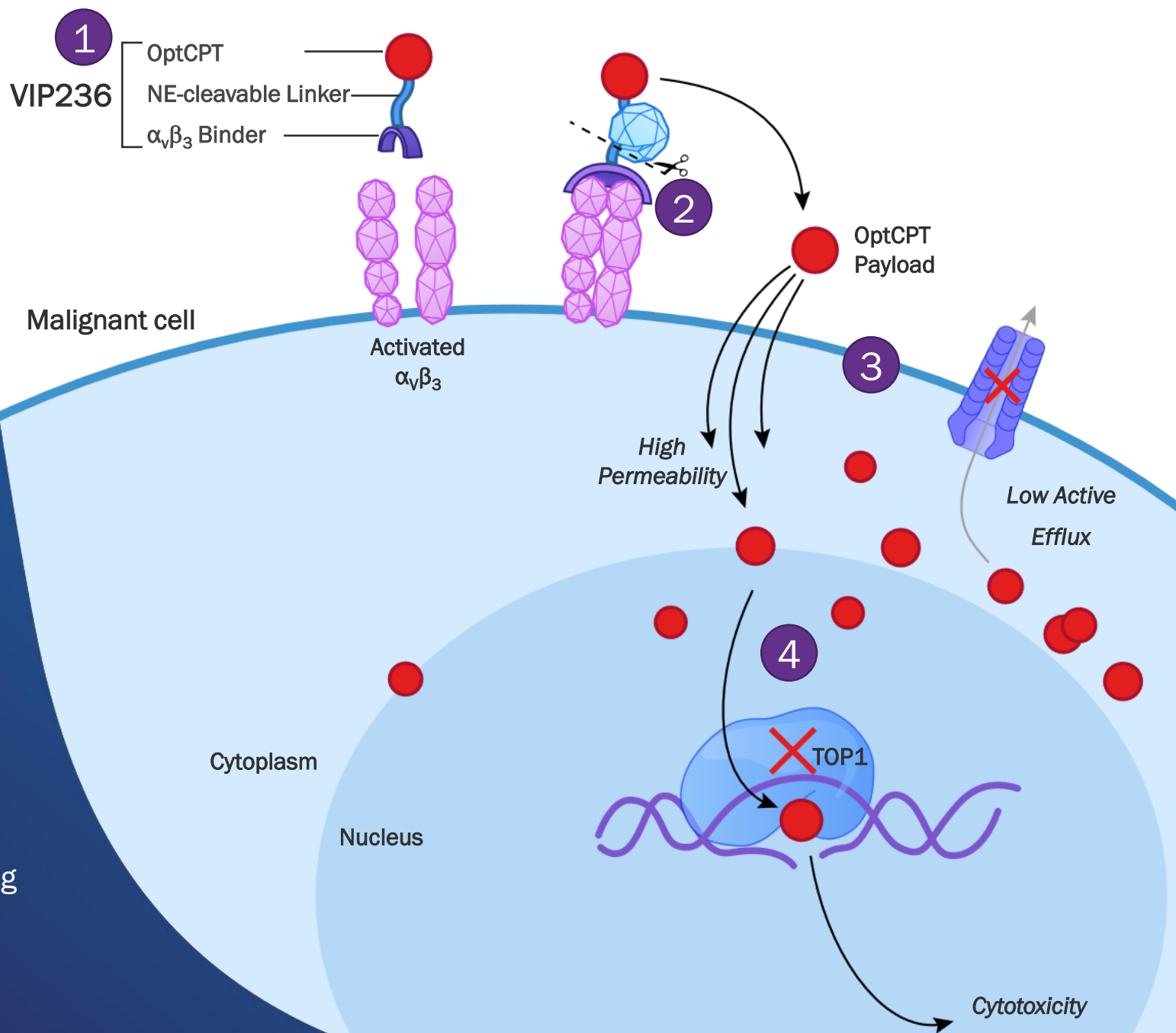


# VIP236

## $\alpha_v\beta_3$ Small Molecule Drug Conjugated to an optCPT

### ENHANCED SAFETY AND PRECISION PROFILE

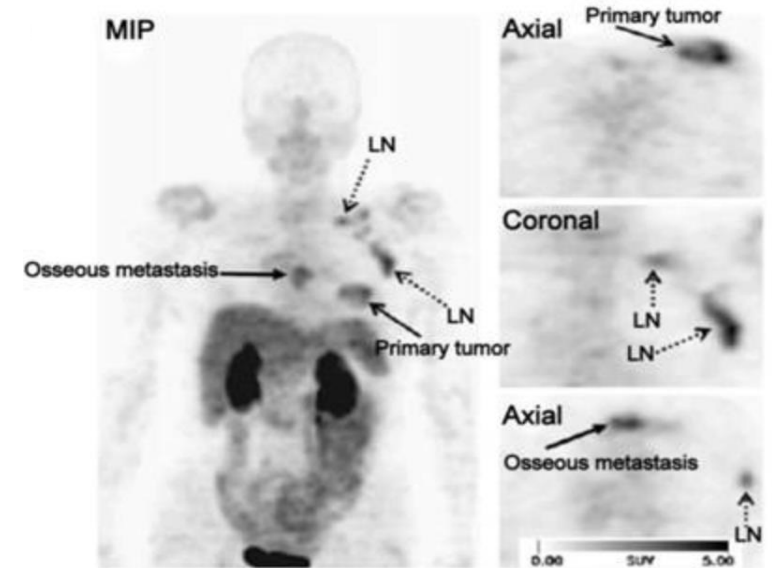
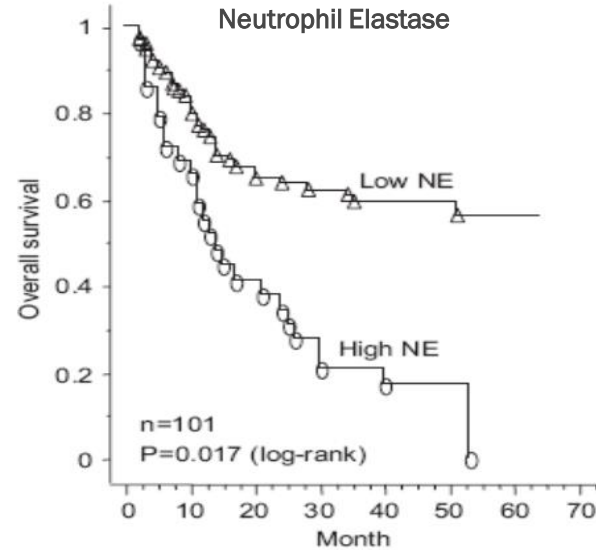
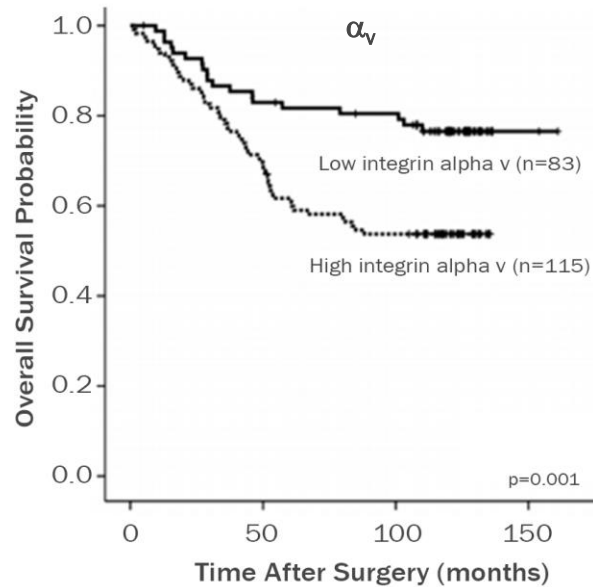
- 1 VIP236 is an  $\alpha_v\beta_3$  integrin binder linked to an optCPT payload
- 2 Payload is released by the enzyme NE in the tumor microenvironment
- 3 The payload accumulates in the tumor cell due to high permeability and resistance to drug transporters
- 4 The payload inhibits topoisomerase 1 causing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



# Expression of $\alpha_v\beta_3$ and Neutrophil Elastase is Associated With Poor Prognosis in Solid Tumor Indications

Kaplan-Meier Survival Curves of Overall Survival According to Integrin  $\alpha_v$  Expression Status in CRC and NE expression in Lung cancer

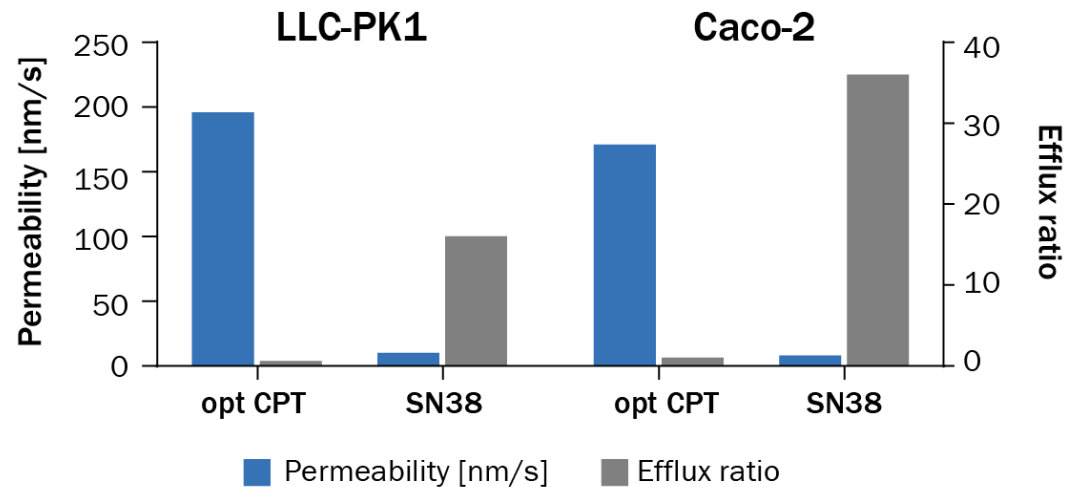
Imaging  $\alpha_v\beta_3$  With Radiolabeled [ $^{18}\text{F}$ ]Galacto-RGD Peptide in a Patient With Invasive Ductal Breast Cancer



- $\alpha_v\beta_3$  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
- Neutrophil infiltration into tumors and expression of neutrophil elastase is associated with poor survival statistics

# 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

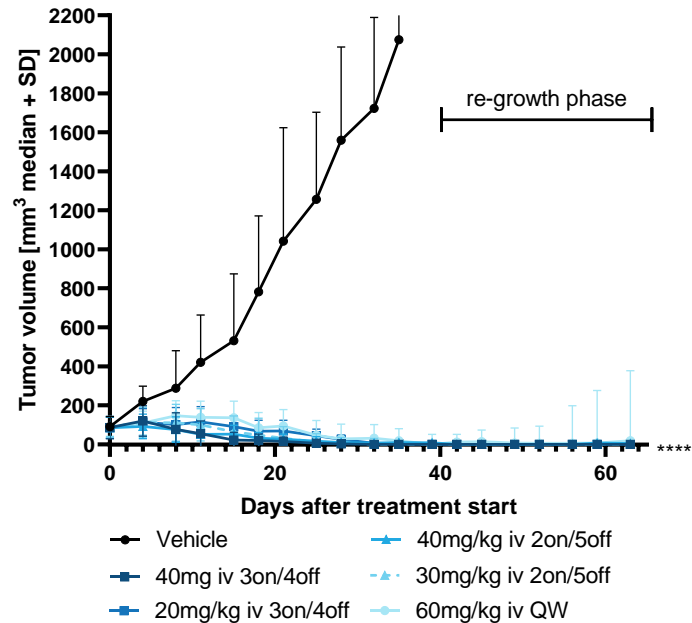
Compound	IC50 (nM)		
	NCI-H1975	NCI-H1975 – P-gp	NCI-H1975 – BCRP
SN38	45	141	512
OptCPT	19	34	27

- Payload: structurally related to the active metabolite of irinotecan known as SN38
- The payload of VIP236 is optimized for high permeability with low active 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
- OptCPT is also specifically designed to avoid enterohepatic recirculation, a common cause for severe diarrhea

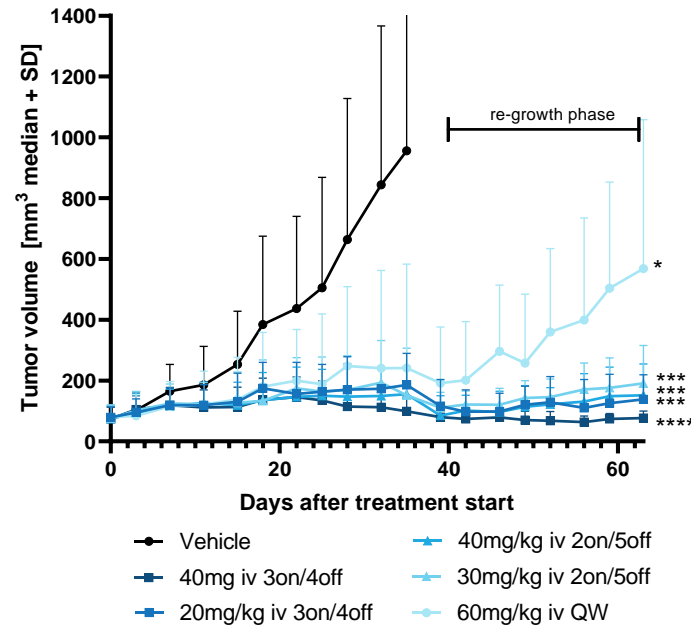
# VIP236 Induces Tumor Regression in Patient Derived Xenograft Models Across Indications

## DOSE AND SCHEDULE OPTIMIZED IN HARD TO TREAT & INVASIVE MODELS

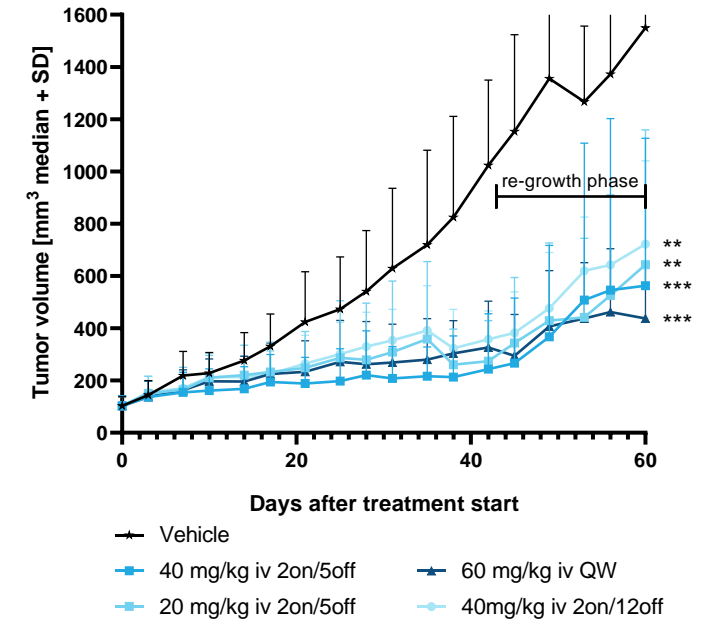
### NSCLC



### RCC



### Liver metastasis from CRC



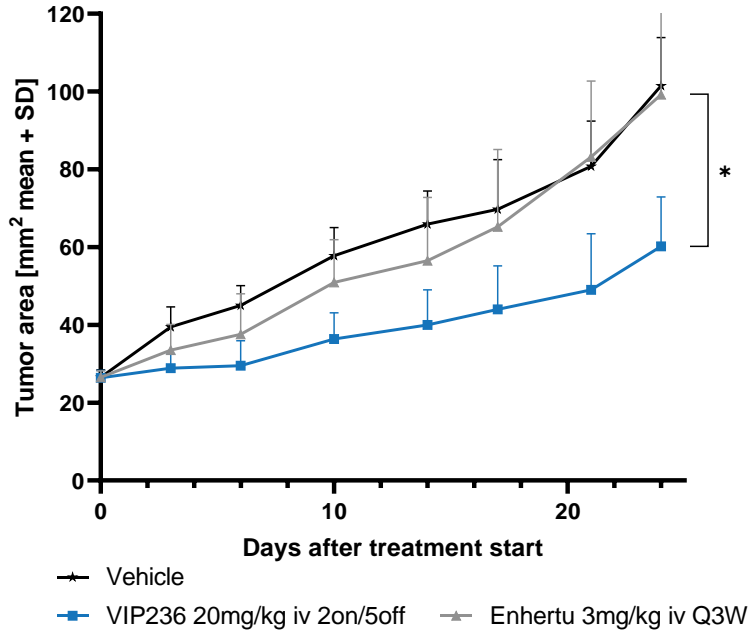
- Durable complete regression in a NSCLC PDX model with all schedules and doses tested
- Partial regression in renal PDX model with durable anti-tumor activity in the 3on/4off and 2on/5off schedules compared to once weekly treatment
- Statistically significant tumor growth inhibition in a liver metastasis CRC PDX model in all schedules with delayed re-growth at higher doses

NSCLC, non-small cell lung cancer; CRC, colorectal cancer; iv, intravenous; PDX, patient derived xenograft; QW, once weekly dosing; RCC, renal cell carcinoma; SD, standard deviation; TNBC, triple negative breast cancer;

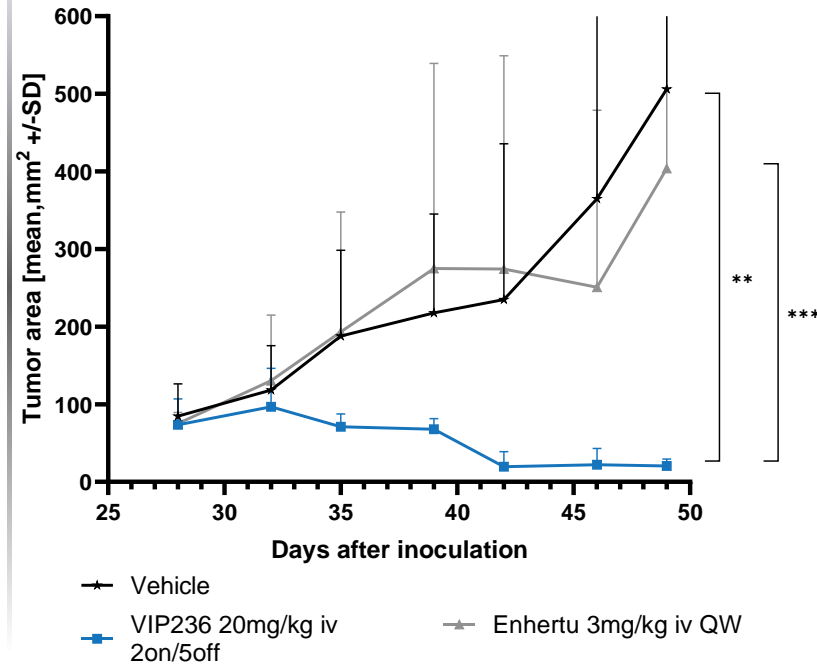
\* p < 0.05 compared with vehicle, \*\* p < 0.01 compared with vehicle, \*\*\* p < 0.001 compared with vehicle, \*\*\*\* p < 0.0001 compared with vehicle

# Improved in Vivo Efficacy of VIP236 Over ENHERTU in HER2 Negative, Low, and High Expressing Gastric CDX and PDX Models

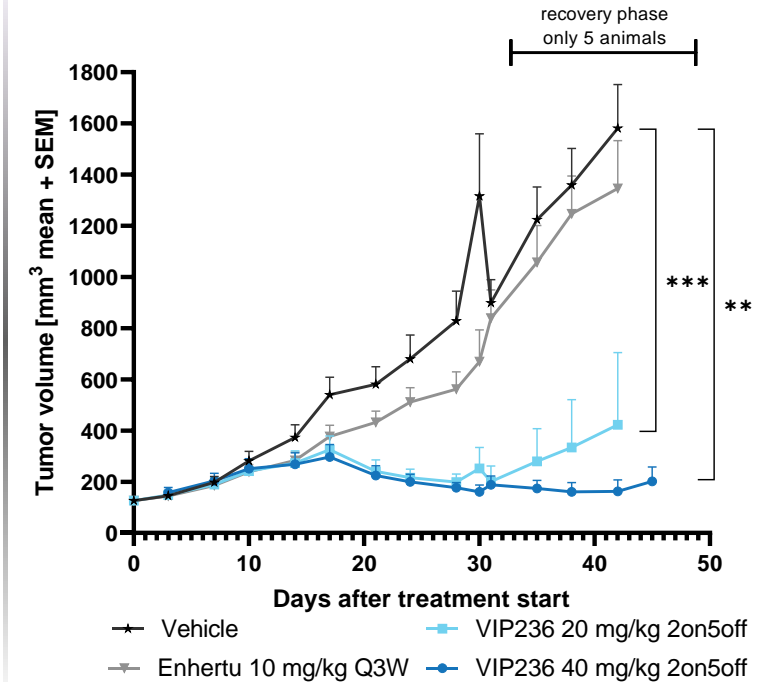
NCIN87 HER2<sup>high</sup>



SNU16 HER2<sup>neg</sup>



GXA3040 HER2<sup>low</sup>



- Statistically significant tumor growth inhibition with VIP236 treatment in CDX and PDX mouse models independent of HER2 status
- Partial regression is observed in the SNU16 HER2<sup>neg</sup>, whereas statistically significant tumor growth inhibition is seen in the NCI N87 HER2<sup>high</sup> CDX model
- In the GXA3040 HER2<sup>low</sup> PDX model the 2on/5off schedule with high doses of 40mg/kg shows tumor regression and reduced re-growth

# VIP236 Phase 1 Dose Escalation Clinical Study

NCT05712889





# Phase 1 Dose-Escalation Study in Patients With Solid Tumors

VNC-236-101

## PRIMARY ENDPOINTS

- Safety
- Tolerability

## SECONDARY ENDPOINTS

- Disease control rate
- PFS
- PK

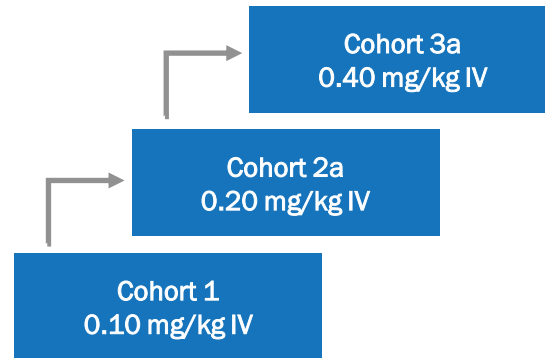
■ Original / Amendment 1

■ Amendment 2

■ Amendment 3 / Expansion cohorts

## ORIGINAL DOSING SCHEDULE

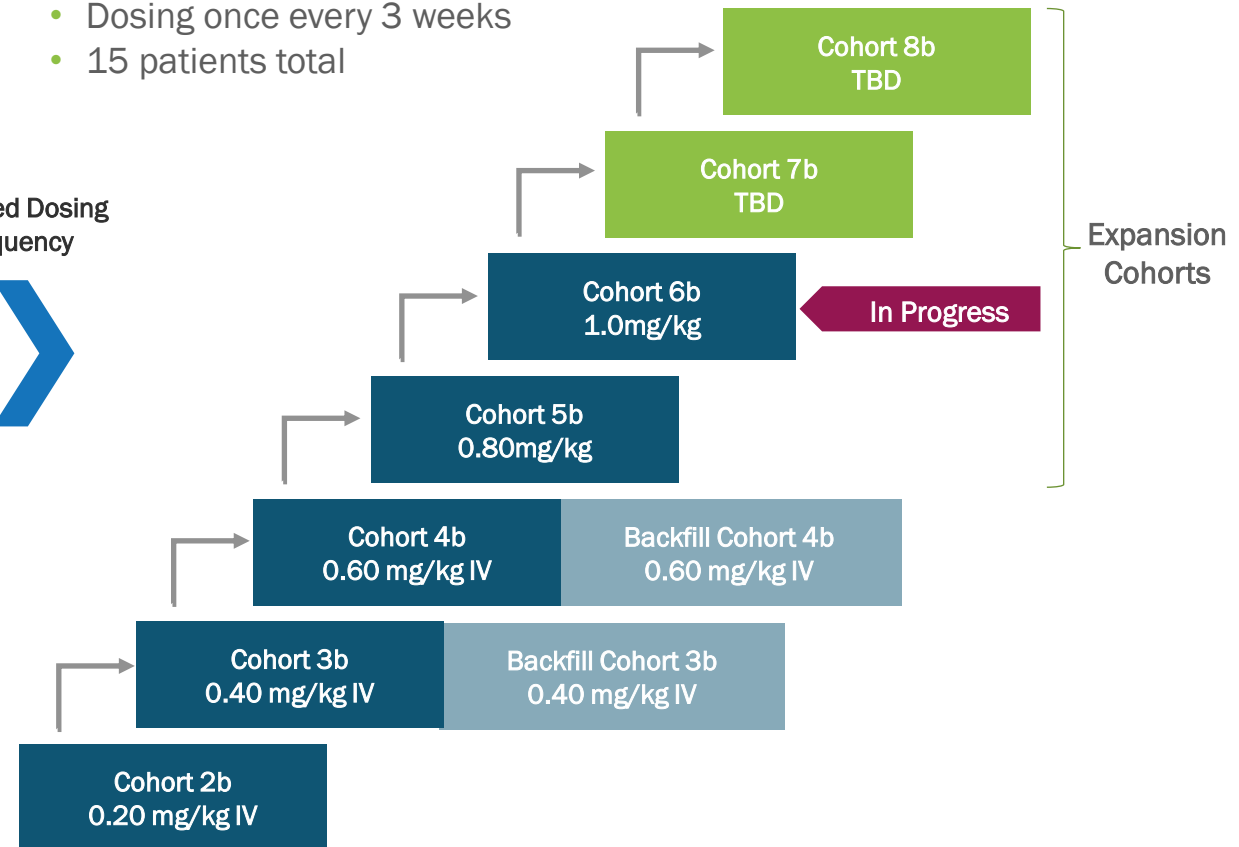
- Dosing 2 days on/5 days off
- 5 Patients total



## CURRENT DOSING SCHEDULE

- Dosing once every 3 weeks
- 15 patients total

Reduced Dosing Frequency

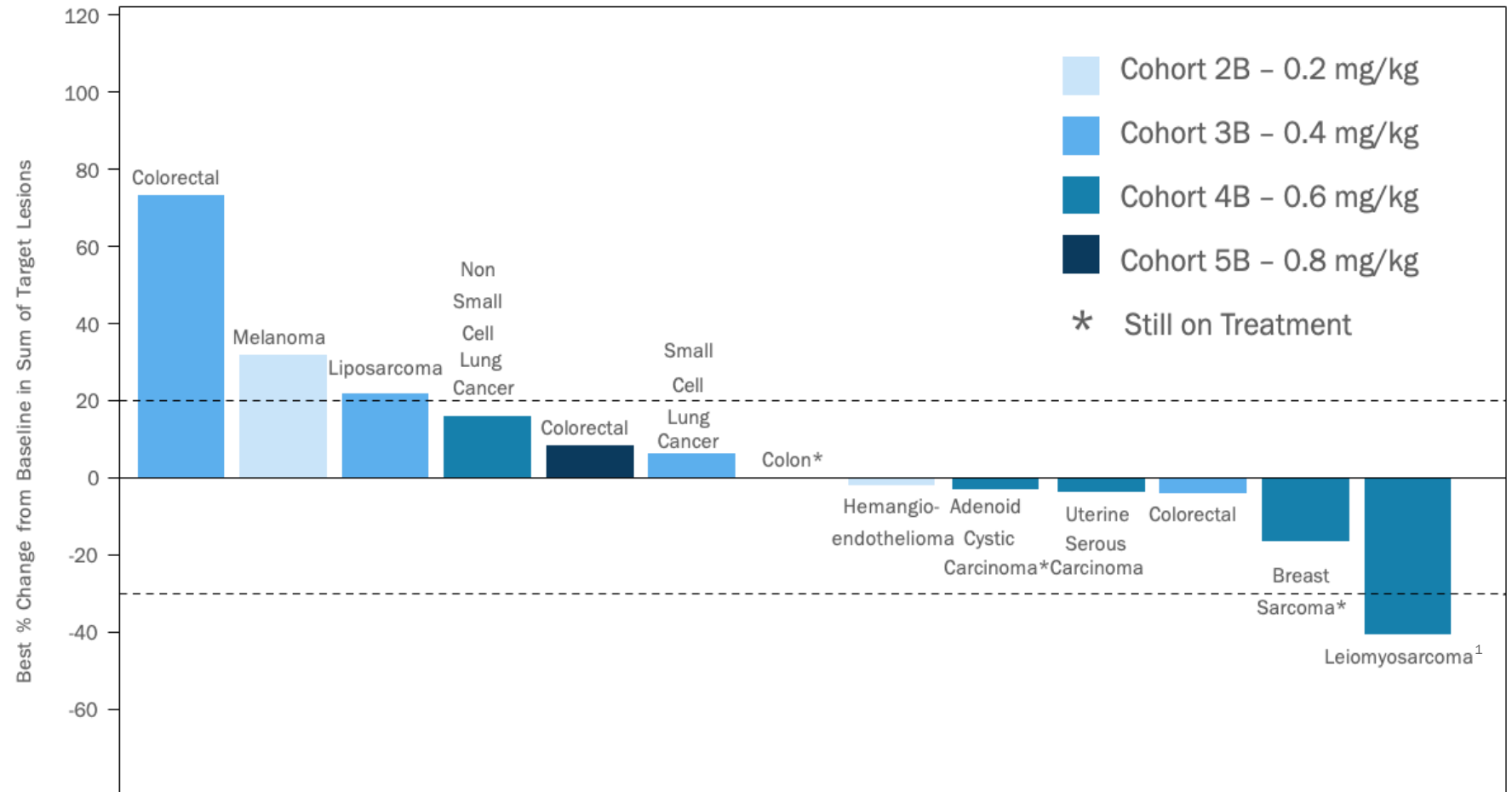


# Positive Signs of Clinical Activity with Tumor Reduction Starting at the Third Dosing Level With Q3W Schedule

N=13

All Comers Solid Tumor Study  
With Heavily Pretreated  
Patients

	Q3W (N=15)
M	8
F	7
Age in years median [range]	58 [35-80]
Prior Therapy ≤ 2	N=14 4 (29%)
≥ 3	10 (66.7%)



<sup>1</sup>The leiomyosarcoma patient had a 41% decrease in two target lesions, but a new 2cm lesion was detected at first scan

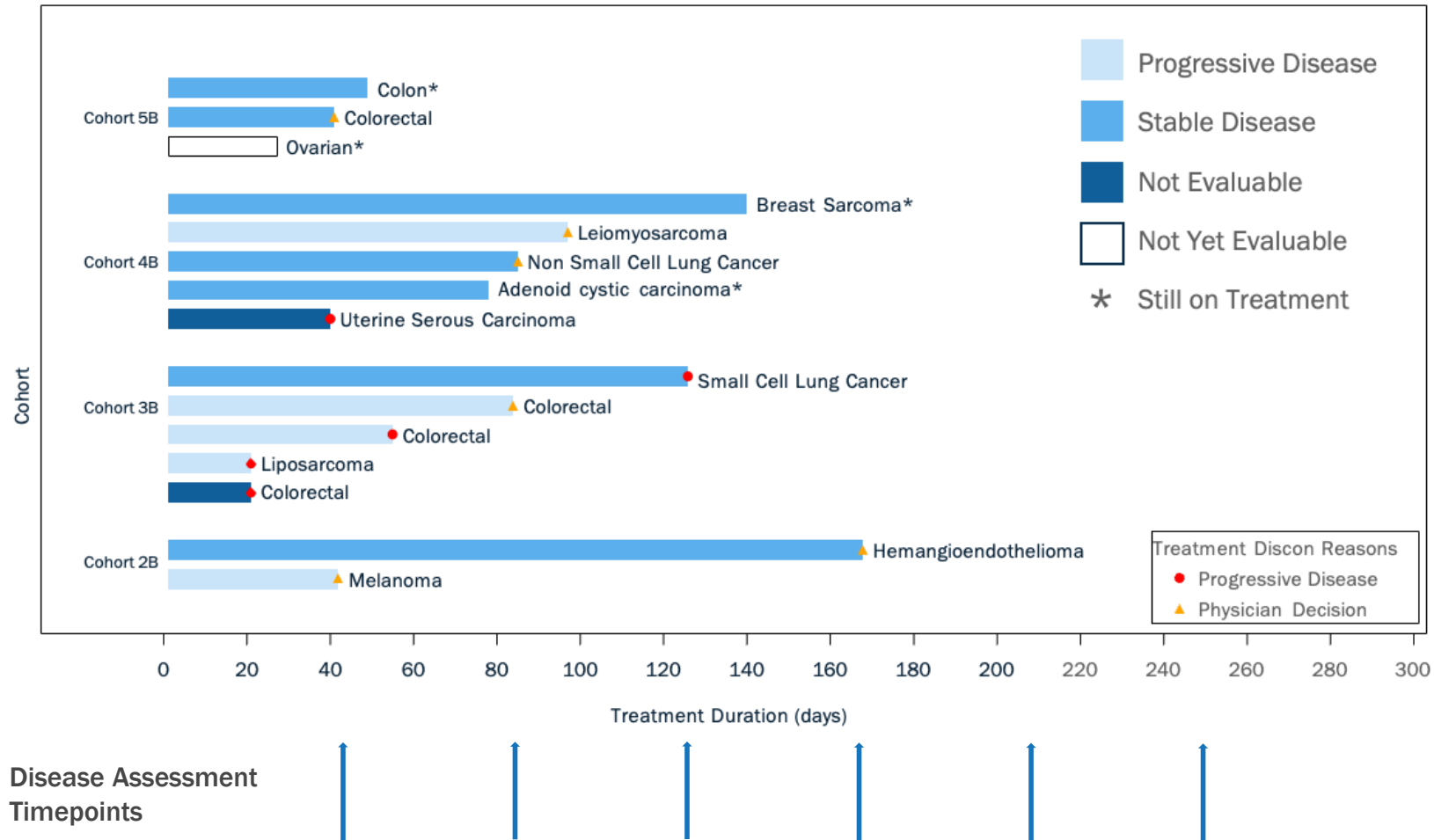
\*=Still on treatment

Data taken from data cut - 25MAR 24

Unaudited data subject to change

# Durable Disease Control Across Multiple Tumor Types With Q3W Schedule

21-DAY CYCLES WITH FIRST DISEASE ASSESSMENT AT THE END OF CYCLE 2



Data taken from data cut – 25MAR 24  
Unaudited data subject to change

# Differentiated and Favorable Safety With Q3W Schedule

## NO PATIENTS DISCONTINUED DUE TO AN ADVERSE EVENT

Drug-related Adverse Events	Q3W (n=15)			
	G1	G2	G3	G4
Preferred Term	G1	G2	G3	G4
Alopecia	5 (33.3%)	2 (13.3%)	0	0
White blood cell count decrease	0	1 (6.7%)	2 (13.3%)	1 (6.7%)
Fatigue	3 (20%)	1 (6.7%)	0	0
Nausea	5 (33.3%)	0	0	0
<b>Diarrhea</b>	<b>3 (20%)</b>	<b>1 (6.7%)</b>	<b>0</b>	<b>0</b>
Neutropenia	0	0	1 (6.7%)	2 (13.3%)
Vomiting	1 (6.7%)	3 (20%)	0	0
Anemia	0	1 (6.7%)	1 (6.7%)	0
Thrombocytopenia	0	1 (6.7%)	1 (6.7%)	0
Lymphocyte count decrease	0	1 (6.7%)	0	0

No Grade 3/4 Diarrhea

Data taken from data cut – 25MAR 24  
Unaudited data subject to change

# VIP943

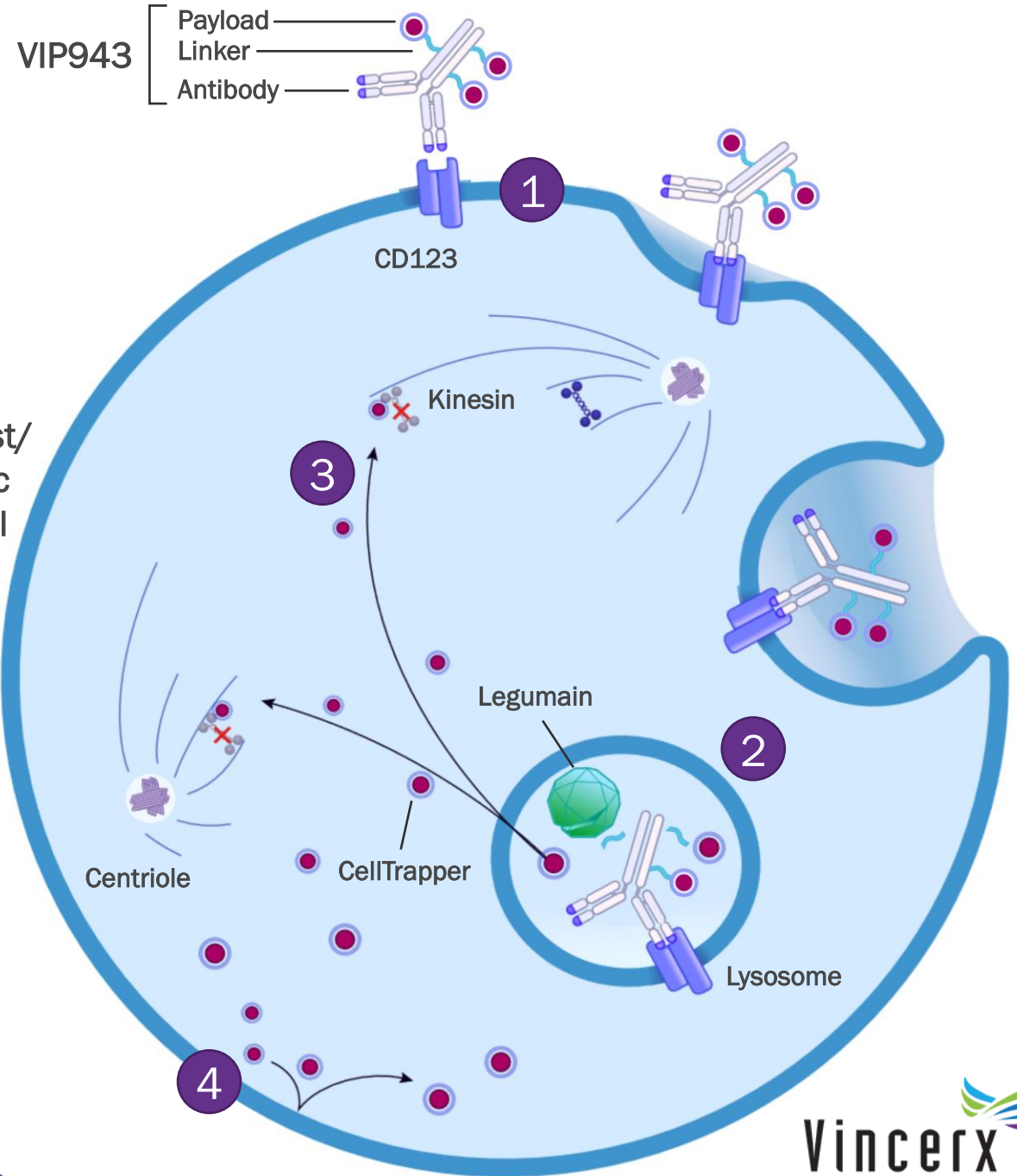
CD123-KSPi Antibody-Drug Conjugate



# VIP943 CD123-KSPi

## ANTIBODY-DRUG CONJUGATE FOR TREATMENT OF AML & MDS

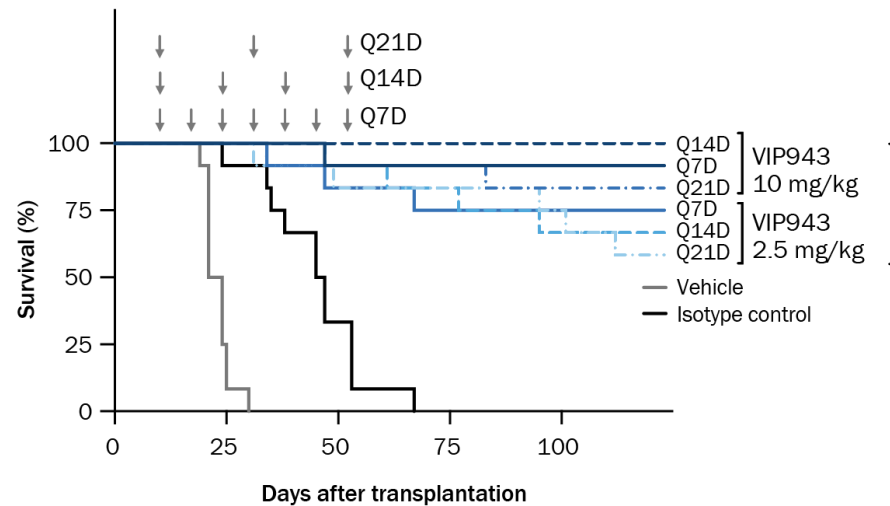
- 1 CD123 is a validated target in myeloid malignancies and a potential leukemic stem cell target
- 2 VIP943-targeting Ab is internalized upon binding to CD123 linked to a legumain released KSPi
- 3 Payload targets KSP stopping cell division and causing catastrophic cell death
- 4 CellTrapper® modified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



# VIP943 Increases Survival in AML Models

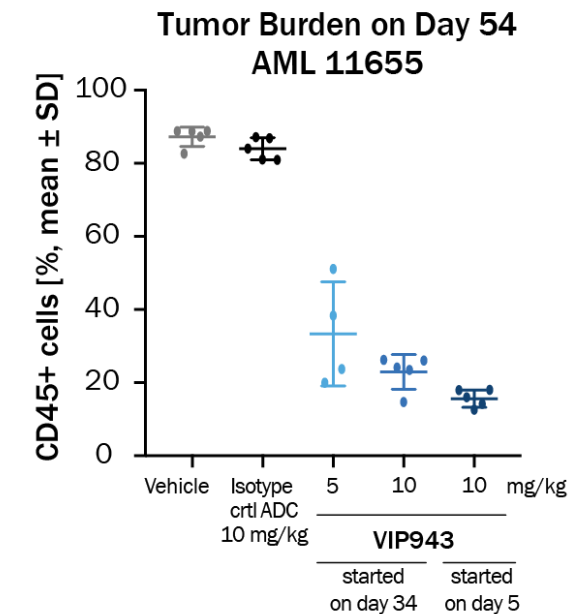
## AML CELL-LINE (CDX) AND PATIENT-DERIVED (PDX) TUMOR MODELS TREATED WITH TARGETED ADC VS ISOTYPE CONTROL ADC

### Striking Improved Survival in AML Model



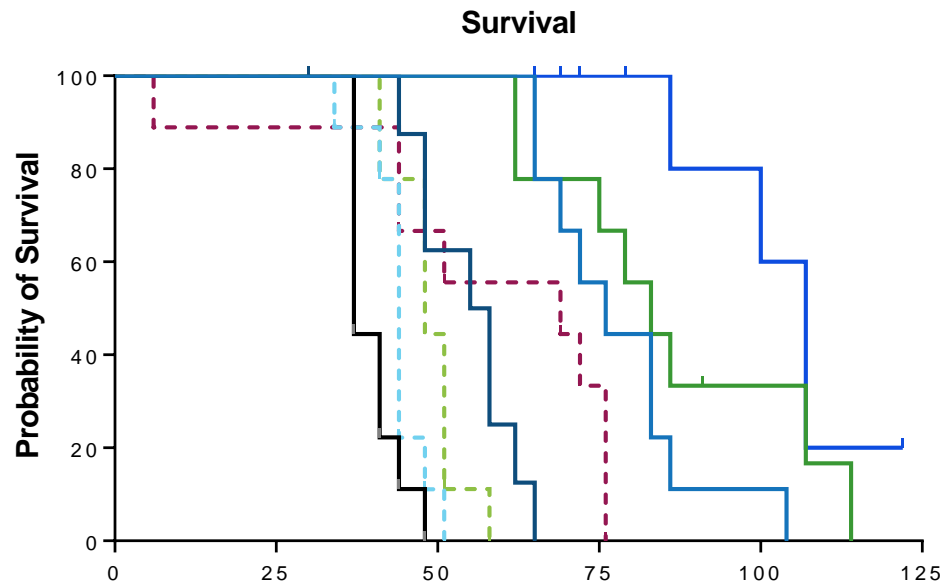
- Increased survival in disseminated CD123+ AML CDX model MOLM-13, treated Q7Dx7
- Improved efficacy of targeted vs isotype control ADC

### Reduction in Tumor Burden in AML PDX Model



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

# Triple Combination of VIP943 with VEN/AZA Achieved Complete Remission and Increased Survival in the AML6252 PDX Model



Treatment	Survival Time (Days)
Iso-ADC	37
VIP943+Ven	57
VIP943+Aza	76
Ven+ Aza	83
VIP943+Ven+Aza	>107

- Iso-ADC 5 mg/kg iv q7d
- - - VIP943 5 mg/kg iv q7d
- - - 5-Azacytidine 2.5 mg/kg sc d 1-5
- - - ABT-199 50 mg/kg po d1-5  
3<sup>rd</sup> cycle reduction to 25mg/kg
- VIP943+ 5-Aza
- VIP943+ Ven
- 5-Azacytidine+Ven
- VIP943+Ven+5-Aza

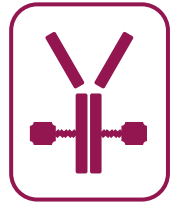
- Patient: FLT3, KDR, PTPN11 mutations, low to moderate CD123 expression, FAB-classification: M4
- The triple combination resulted in 5 complete remissions at the end of the treatment period while in the Aza/Ven group 2 CR were achieved
- Strong survival benefit is observed with triple combination

AZA, azacitidine; CR, complete response; PDX, patient derived xenograft; VEN, venetoclax.  
CR: No visible tumor



# VIP943 Displays an Improved Safety Profile in Monkeys When Compared to Mylotarg™ (Gemtuzumab-Ozogamycin)

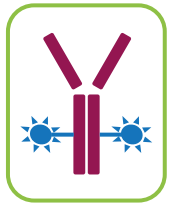
## LEGUMAIN-KSPI PAYLOAD CAN IMPROVE THE SAFETY PROFILE OF MYLOTARG



**Mylotarg**

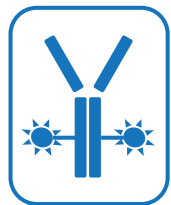
(Gemtuzumab-Ozogamycin)

- Anti-CD33 mAb
- Calicheamicin payload
- DAR: 2-3



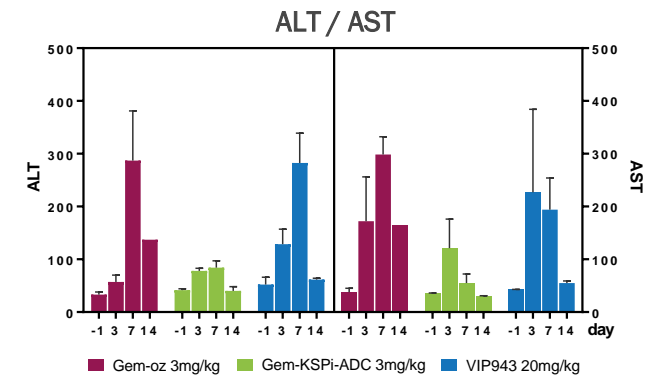
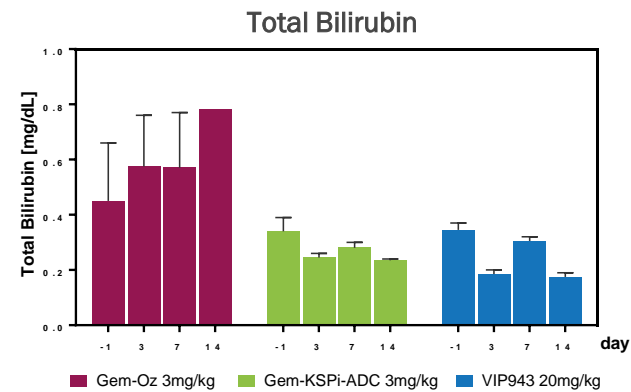
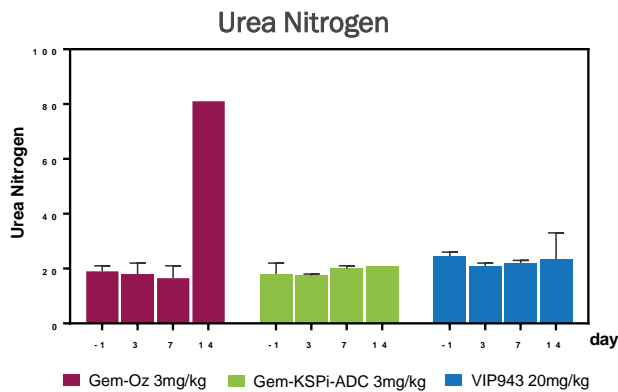
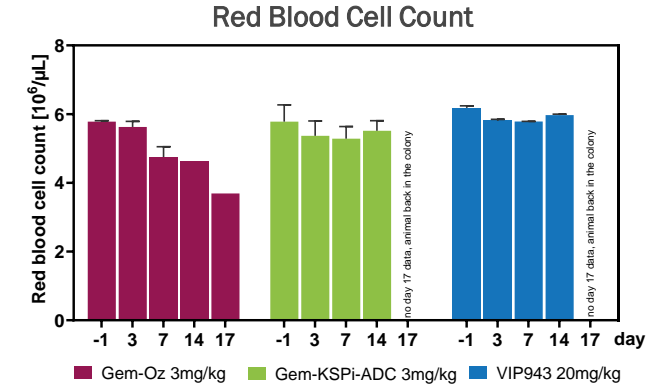
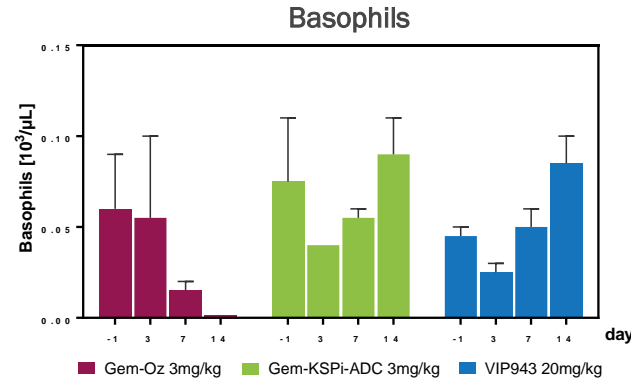
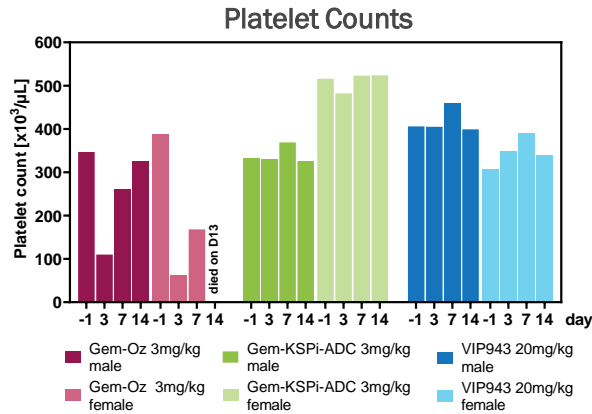
**Gem-KSPI-ADC**

- Anti-CD33 mAb
- Legumain-KSPI payload
- DAR: 5.3



**VIP943**

- Anti-CD123 mAb
- Legumain-KSPI payload
- DAR: 6



- Critical drop of platelet counts, and red blood cell count with insufficient recovery in the Mylotarg group
- Increased liver enzymes and severe increase in total bilirubin for animals treated with Mylotarg, indicating liver toxicity
- Extreme increase of urea nitrogen, indicating kidney toxicity in Mylotarg-treated animals
- No adverse events occurred with ADCs utilizing the legumain-KSPI payload; in contrast to two monkey deaths treated with Mylotarg

ADC, antibody-drug conjugate; KSPI, kinase spindle protein inhibitor; mAb, monoclonal antibody.

# VIP943 Phase 1 Dose Escalation Clinical Study

NCT06034275



# Phase 1 Dose-Escalation Study in Patients with CD123+ Relapsed/Refractory in Hematologic Malignancies

VNC-943-101

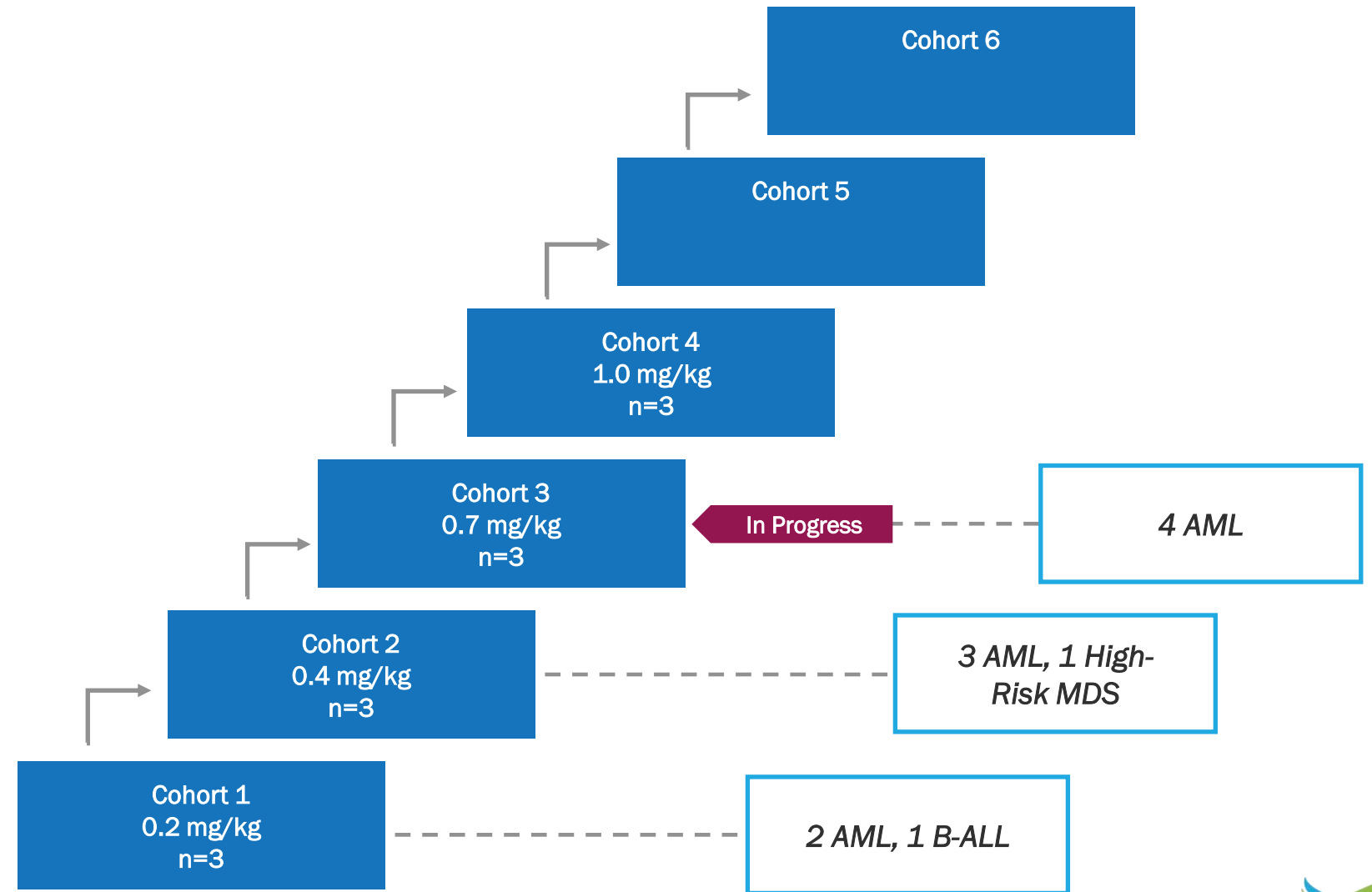
Enrolling adults with AML, Higher-Risk MDS, or B-ALL

**PRIMARY ENDPOINTS**

- Safety
- Tolerability

**SECONDARY ENDPOINTS**

- Response rate
- PK



# Preliminary Results Show VIP943 has Favorable Safety and Tolerability Profile to Date

	# of Patients Cohort 1 (0.2 mg/kg)	# of Patients Cohort 2 (0.4 mg/kg)
N	3	4
Disease	<ul style="list-style-type: none"> <li>• 1 de novo AML</li> <li>• 1 secondary AML</li> <li>• 1 B-ALL</li> </ul>	<ul style="list-style-type: none"> <li>• 1 de novo AML</li> <li>• 2 secondary AML</li> <li>• 1 MDS</li> </ul>
Completed 28-day DLT evaluation	3	4
Received Cycle 2 dose	2	3
Received Cycle 3 dose	1	1
Still on study in Cycle 3	0	1 MDS
DLTs	0	0
Drug-related AEs		2 <ul style="list-style-type: none"> <li>• 1 pt Grade 2 dry eye</li> <li>• 1 pt Grade 1 hot flush, Grade 1 confusion and Grade 3 diarrhea*</li> </ul>

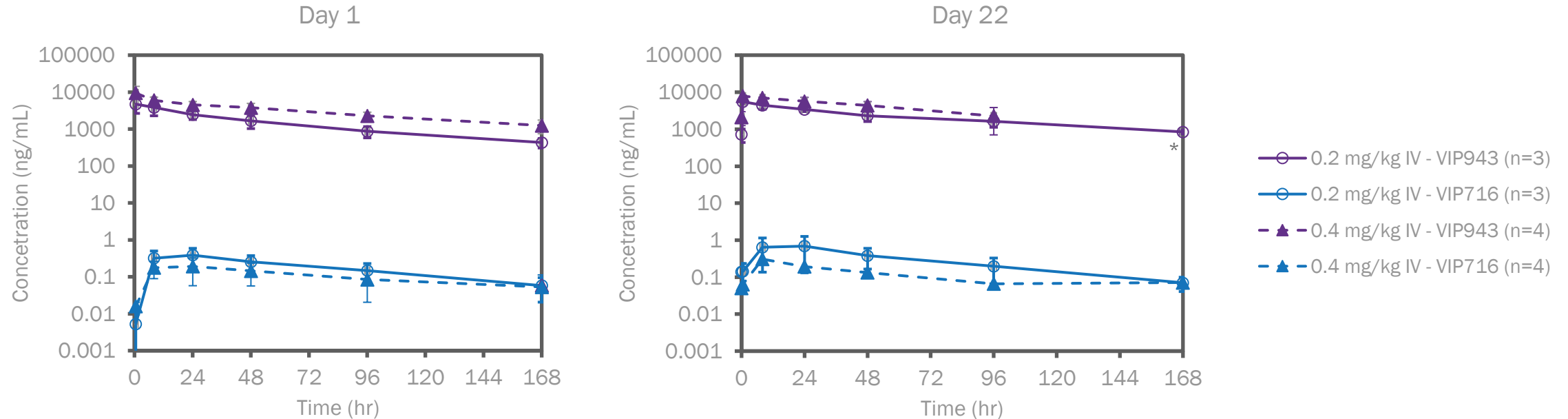
**7 OF 7 SEQUENTIALLY DOSED PATIENTS COMPLETED 28-DAY DLT REVIEW**

- No discontinuations due to AEs
- No dose reductions
- 4 patients enrolled in Cohort 3 are undergoing DLT assessment

\*Grade 3 diarrhea was serious adverse event.  
Data taken from data cut – 25MAR 24  
Unaudited data subject to change

# VIP943 PK Data Shows Very Little Free Payload in Circulation, Consistent With the Favorable Safety Profile Observed To Date

COHORT 1 (0.2 mg/kg) AND COHORT 2 (0.4 mg/kg)



- 0.7% - 3.0% free payload in circulation after four weekly doses indicative of our stable and selective legumain cleavable linker
- Low free payload after multiple doses is consistent with the favorable clinical safety profile observed to date and consistent with preclinical studies



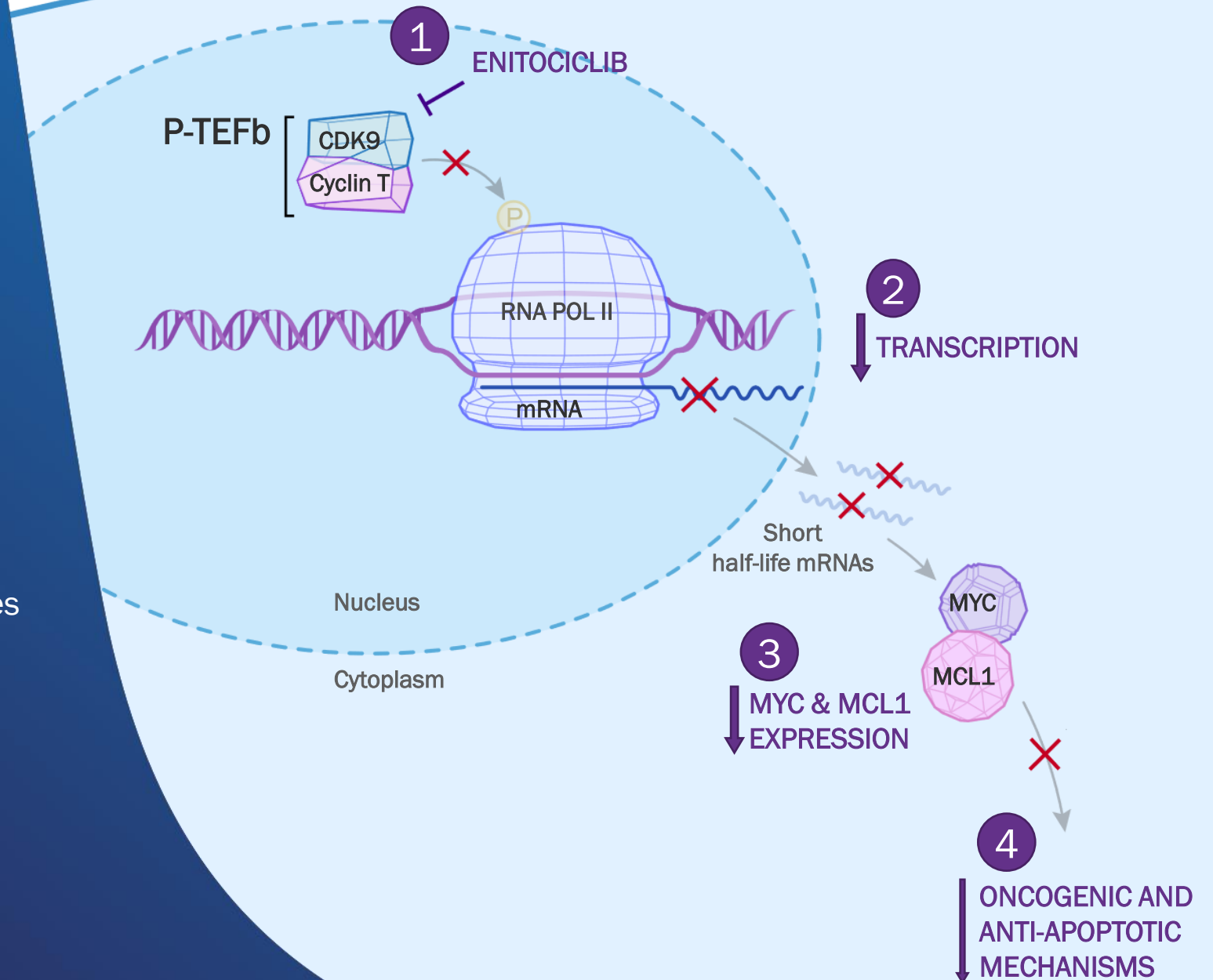
# Enitociclib

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”



# 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	<b>0.57</b>	<b>63</b>	<b>2.9</b>	<b>19</b>	<b>0.73</b>
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 $\mu$ M ATP	<b>4.52</b>	28.20	<b>5.96</b>	29.40	<b>3.20</b>
IC50 (nM) at 2 mM ATP	<b>11.80</b>	1.670	32.80	1.130	<b>4.22</b>

- Enitociclib is a highly selective CDK9 inhibitor that retains its potency in both high and low ATP environments
- Selectivity of CDK9 inhibitors is a known prerequisite for a tolerable safety profile

ATP, adenosine triphosphate; CDK, cyclin-dependent kinase; IC, inhibitory concentration; Kd, equilibrium dissociation constant.  
Lücking et al. J. Med. Chem. 2021, Boffo et al. J Exp Clin Cancer Res. 2018, Frigault, et al. EHA 2022.; Diamond, et al. CCR 2022.

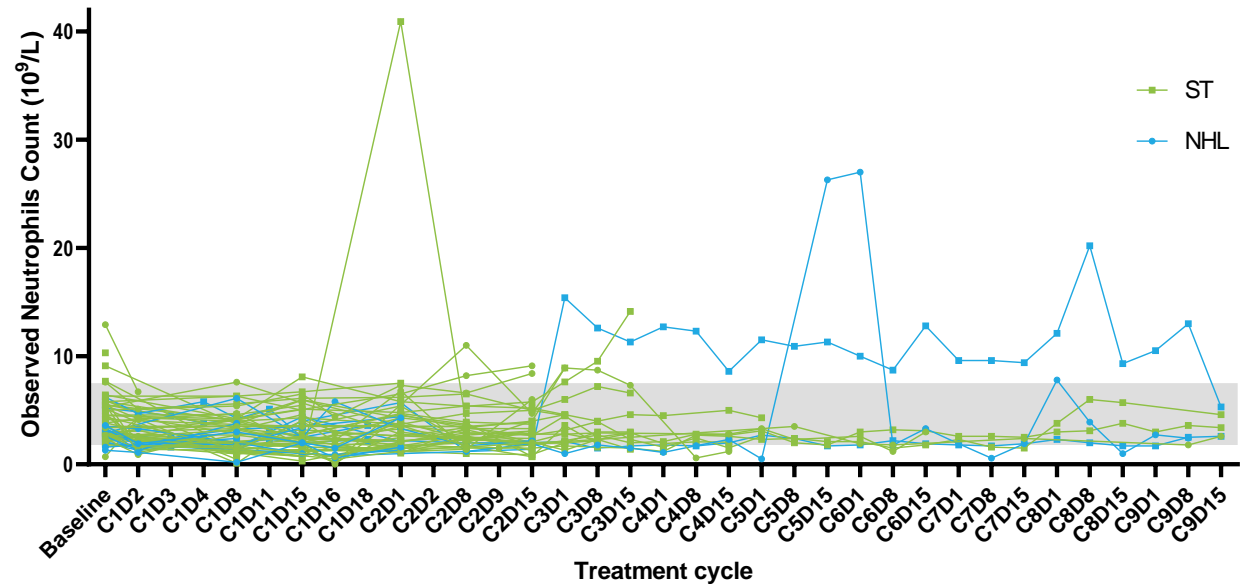


# Enitociclib Has a Favorable Safety Profile in Patients With Solid Tumors and Lymphoma

Treatment-Emergent Adverse Events (n=63)

Adverse Events (>15%)	Any Gr	Gr 1	Gr 2	Gr 3	Gr 4	Gr 5
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
Nausea	41(65.1)	24(38.1)	17(27.0)	0	0	0
Vomiting	32(50.8)	21(33.3)	11(17.5)	0	0	0
Fatigue	21(33.3)	10(15.9)	10(15.9)	1(1.6)	0	0
Anemia	20(31.7)	6(9.5)	8(12.7)	6(9.5)	0	0
Diarrhea	22(34.9)	17(27.0)	5(7.9)	0	0	0
Neutropenia	14(22.2)	0	5(7.9)	5(7.9)	4(6.3)	0
Constipation	12(19.0)	9(14.3)	2(3.2)	1(1.6)	0	0

Neutropenia Is an On-Target (CDK9) Toxicity and Is Monitorable and Manageable With Supportive Care (n=63 patients)



## 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

# Enitociclib Is Well Tolerated and Induces Durable Complete Remissions (n=63)

## Monotherapy Activity

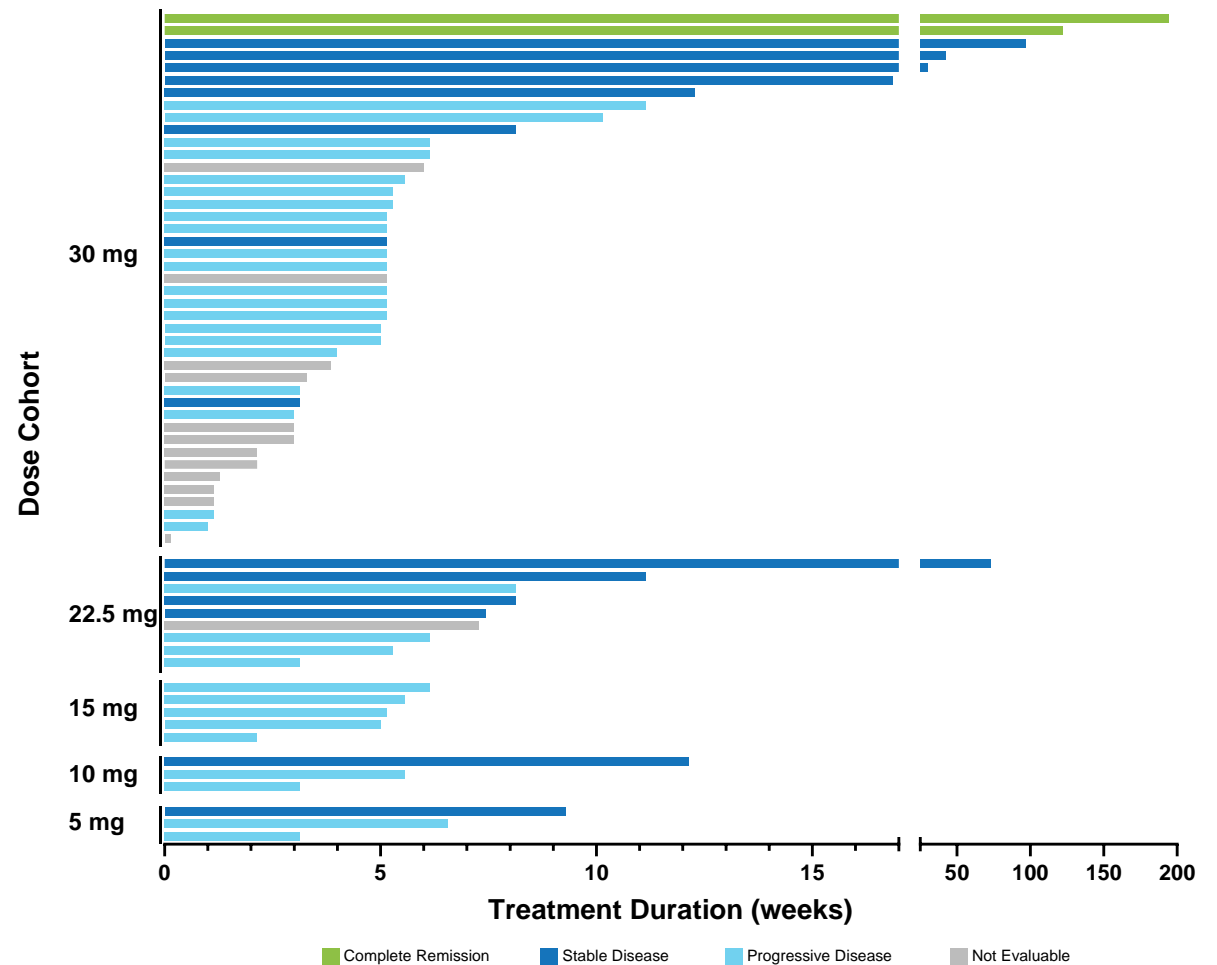
### 2 CRs of 7 DH-DLBCL (29% CR rate)

- 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

### 14 patients had stable disease as best response

- 1 transformed follicular, 31 cycles
- 5 ovarian cancer, 1 to 10 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, 4 cycles

As of January 2024, total number of patients dosed with enitociclib: 95



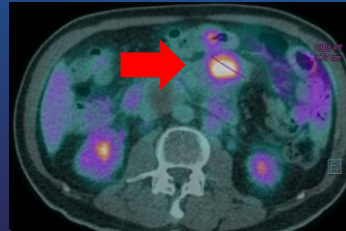
# Enitociclib Induces Durable Complete Metabolic Remission and Tumor Regression

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

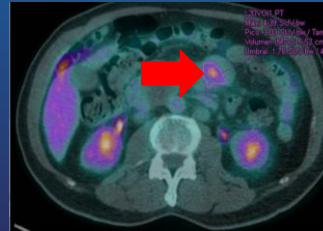
Age: 58 years  
Cell of origin: GCB  
Prior therapy (response):

- R-CHOP (PR)
- Radiotherapy (PR)
- R-GemOx (PD)

NOVEMBER 2016



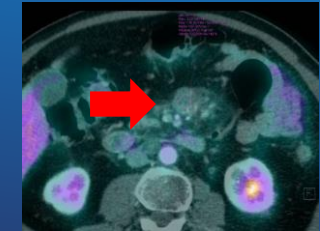
APRIL 2017



SEPTEMBER 2017



MARCH 2021



Date of 1st dose: 08 NOV 2016

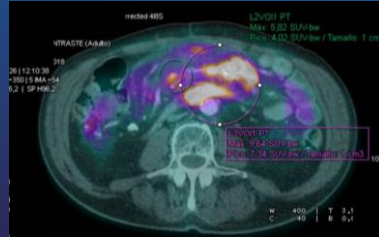
Date of last dose: 16 JUL 2020

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

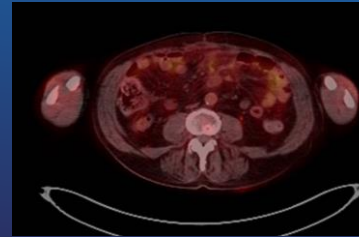
Age: 78 years  
Cell of origin: GCB  
Prior therapy (response):

- R-EPOCH (PR)
- R-DHAP (PD)
- Palliative radiotherapy

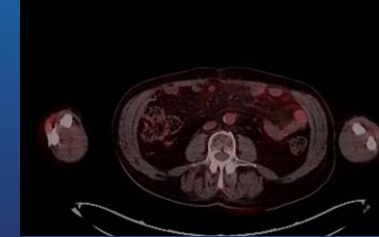
MARCH 2018



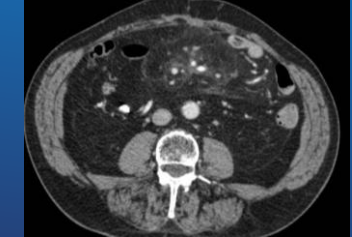
OCTOBER 2018



JUNE 2020



FEBRUARY 2021



Date of 1st dose: 03 APR 2018

Date of last dose: 23 JUL 2020

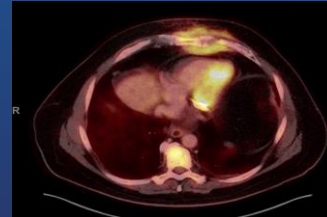
**PATIENT:** Diagnosis tFL

Age: 72 years  
Cell of origin: GCB  
Prior therapy (response):

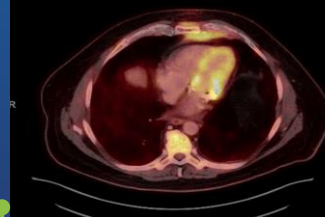
- R-EPOCH (CR for 5 years)

Status: Active in Cycle 24  
Latest Response: SD  
Next Scan: Jan2024

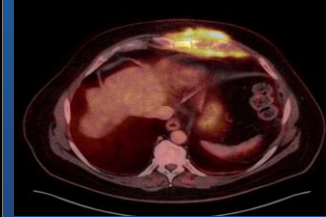
MAY 2022



NOVEMBER 2022

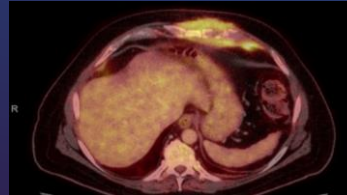


JANUARY 2023

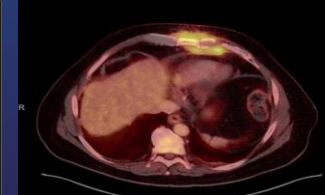


Date of 1st dose: 08 JUNE 2022

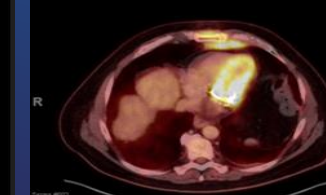
MAY 2023



JULY 2023



OCTOBER 2023



Cycle 23: 51% reduction in 2 TL

# Enitociclib Induces Tumor Regression with Partial Responses in Combination with Venetoclax and Prednisone

## NIH SPONSORED TRIAL IN R/R LYMPHOID MALIGNANCIES (NCT05371054)

### OBJECTIVES:

- **Phase 1:** To determine the MTD, RP2D, and the safety and toxicity profile of the combination of enitociclib with venetoclax and prednisone (VVIP)
  - MYC-rearranged DLBCL
  - Non-GCB DLBCL
  - Peripheral T-cell lymphoma
- **Phase 2:** To determine the complete response rate of the combination of enitociclib with venetoclax and prednisone

### PATIENT 2: Diagnosis R/R AITL

- 91% decrease in tumor burden
- Partial response on dose level 1

### PATIENT 5 : Diagnosis Refractory HGBCL-DH-BCL2

- ~80% decrease in tumor burden
- Partial response on dose level 2

### PATIENT 4: Diagnosis EBV+ PTCL

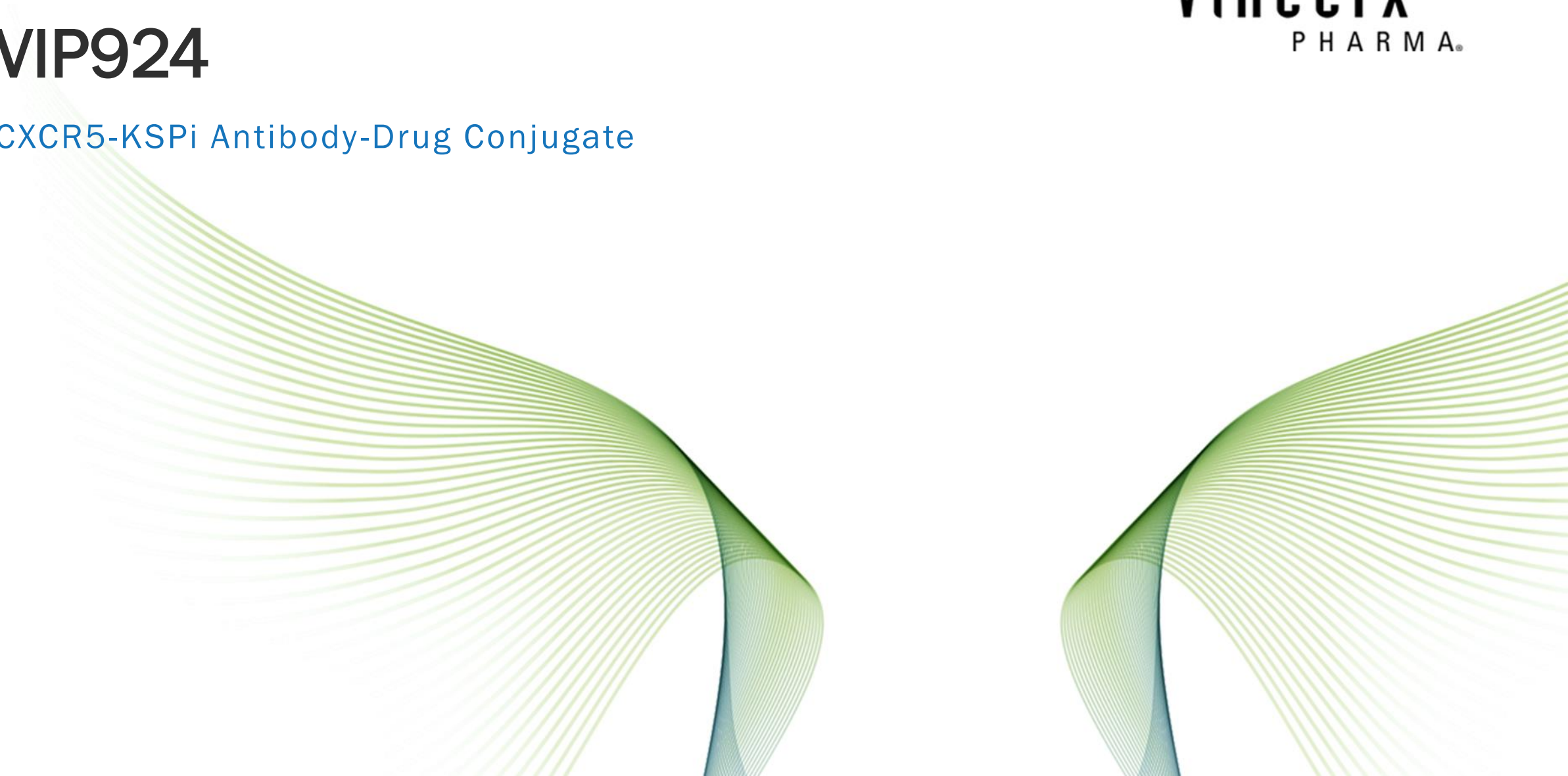
- 80% decrease in pulmonary lesion
- Partial response on dose level 2

# Preclinical Assets



# VIP924

CXCR5-KSPi Antibody-Drug Conjugate

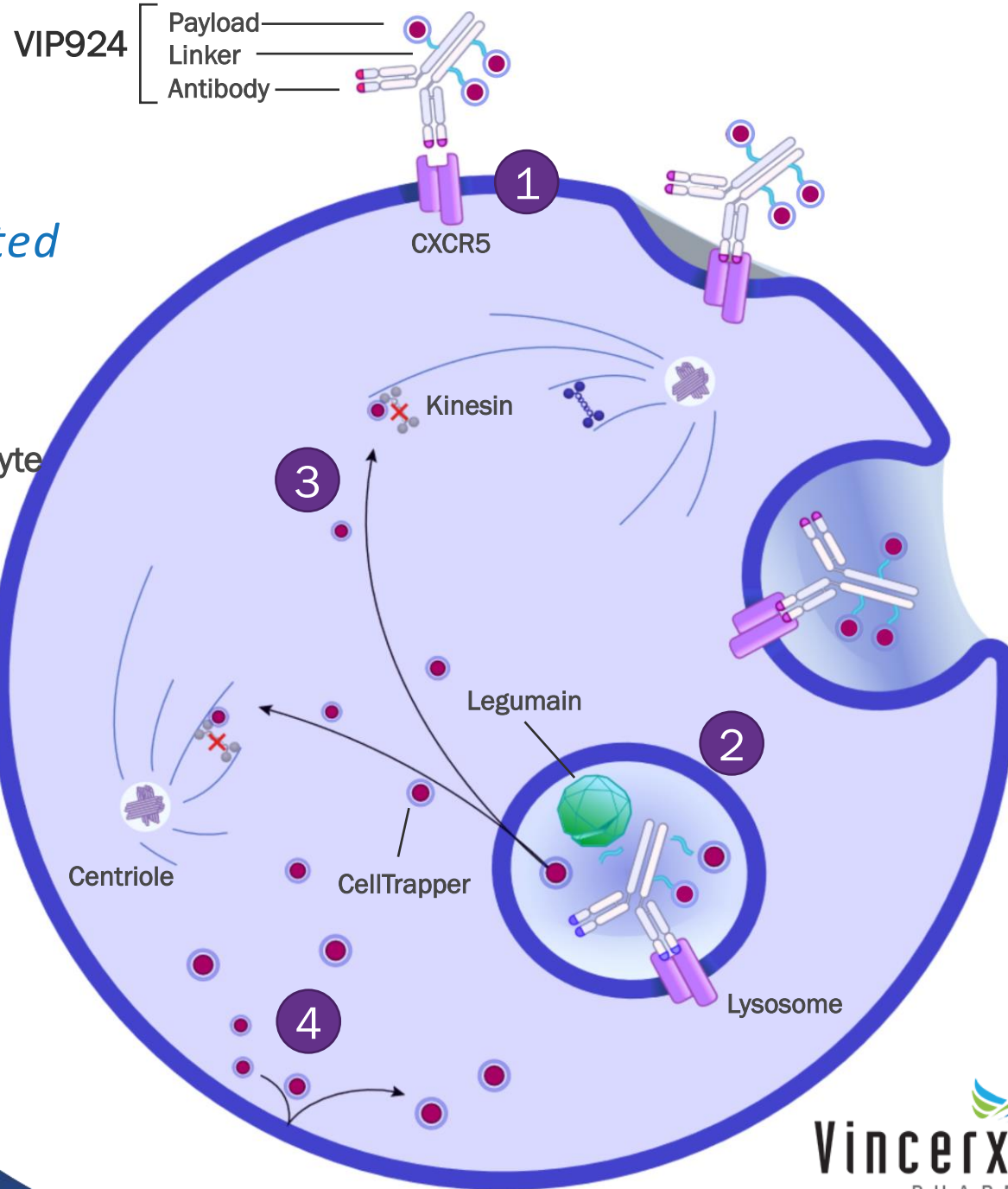


# VIP924 CXCR5-KSPi

## FOR TREATMENT OF B-CELL MALIGNANCIES

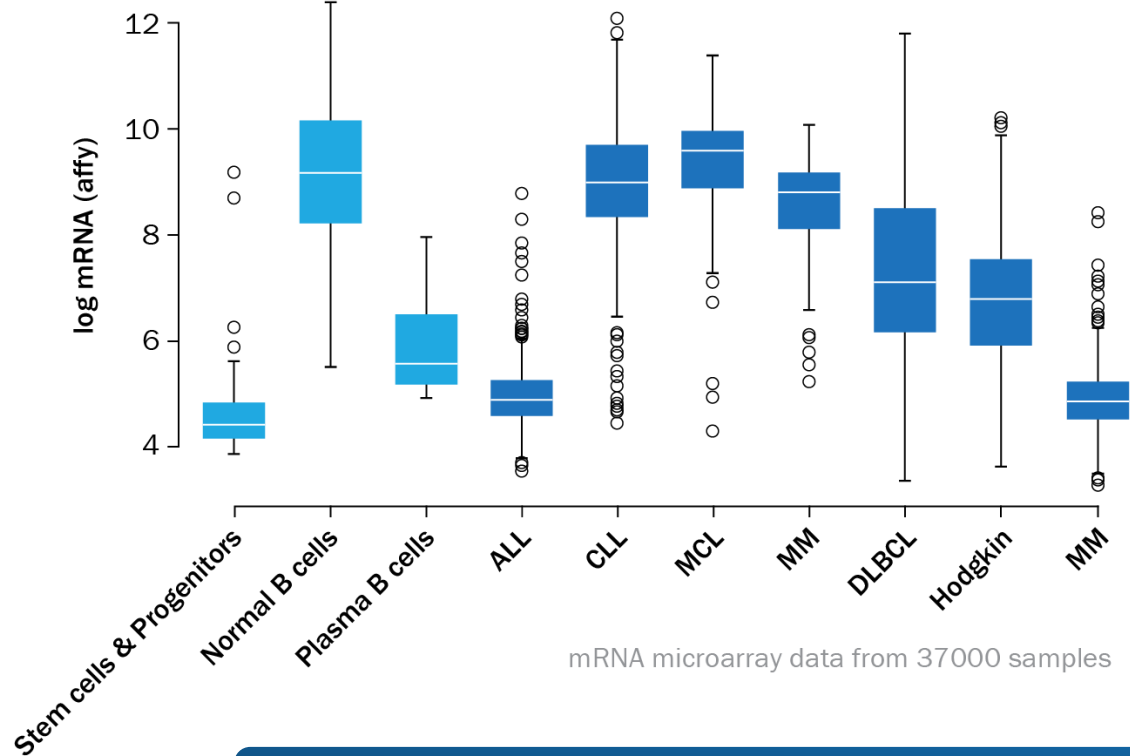
- 1 CXCR5 regulates chemotaxis, germinal center formation, and plasma and memory B-cell differentiation
- 2 VIP924 has an internalizing Ab upon binding to CXCR5 which is linked to a legumain released KSPi that drives cell death during cell division
- 3 Payload targets KSP stopping cell division and causing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

*VIP924 is a first-in-class CXCR5 targeted therapy*

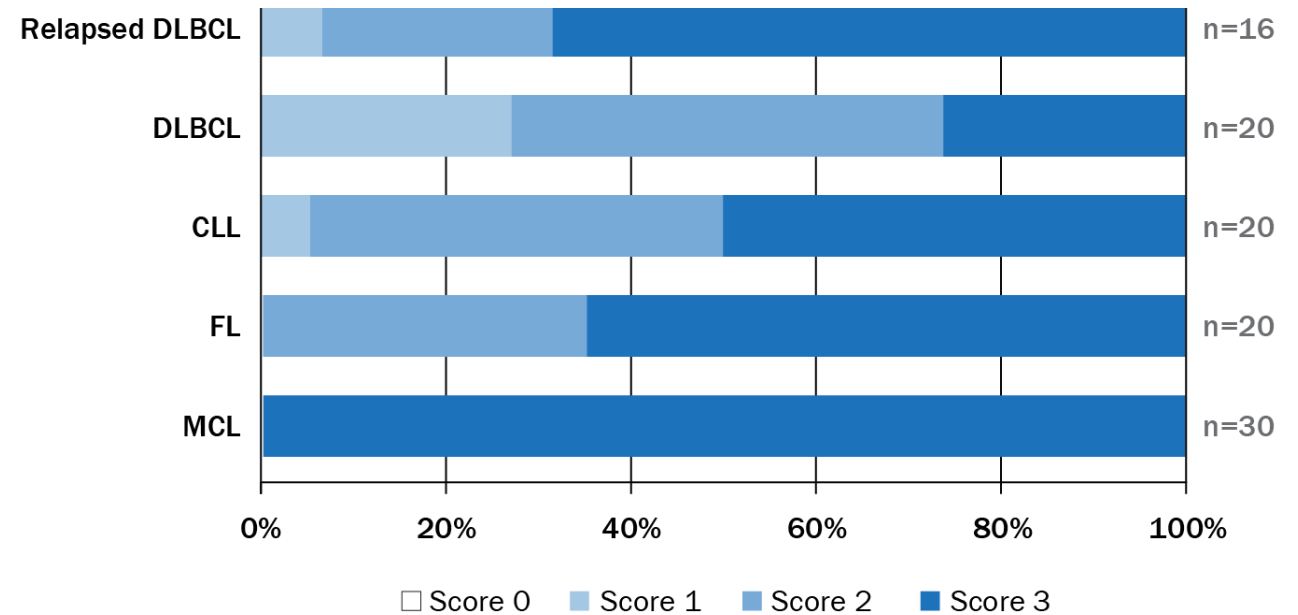


# CXCR5 Is Expressed in B-Cell Malignancies

High CXCR5 mRNA Across NHL



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



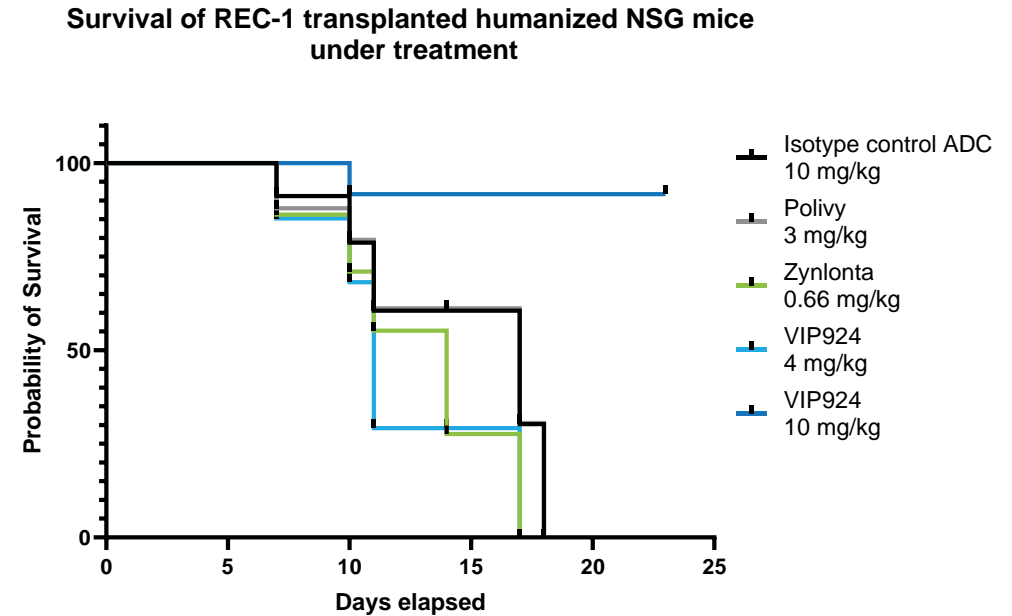
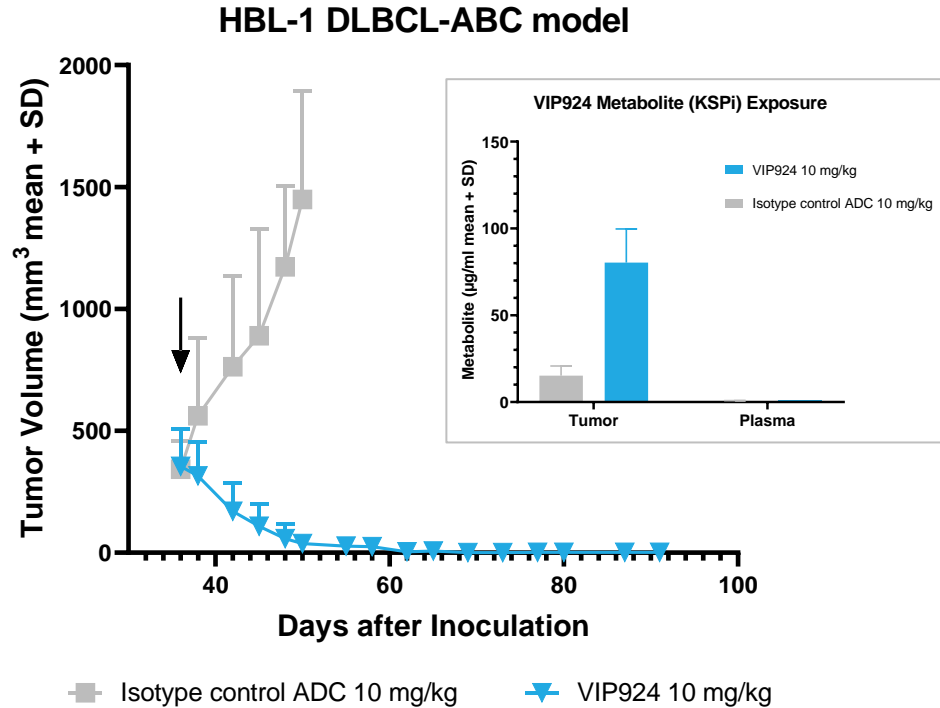
IHC analysis of samples from patients with hematologic malignancies shows CXCR5 expression in MCL, DLBCL, FL and CLL

affy, Affymetrix; CHOP, cyclophosphamide, doxorubicin, vincristine, prednisone; CLL, chronic lymphocytic leukemia; DLBCL, diffuse large B-cell lymphoma; FL, follicular lymphoma; IHC, immunohistochemistry; MCL, mantle cell lymphoma; MM, multiple myeloma; NHL, non-Hodgkin lymphoma; R, rituximab.  
Schomber et al, AACR 2023. Poster.





# Significant Anti-Tumor Efficacy of VIP924 in Established Large Tumor in the HBL-1 Lymphoma CDX Model and in Mantle Cell Lymphoma Mouse Model



- Durable complete response in 67% of treated mice in an HBL-1 CDX model treated with a single dose of 10 mg/kg VIP924
  - Metabolite exposure confirms selective tumor enrichment of the payload in VIP924 treated animals
- Only animals treated with VIP924 showed a significant tumor growth inhibition and a survival benefit as compared to control treated animals in a humanized REC-1 NSG mice

# Shaping the Future of Cancer Treatment Through Patient-Centric Drug Innovation

## Building Strong Partnerships

- Collaborative and flexible partnerships that bring mutual benefit

## Best-in-Class Team

- R&D Team with 30+ years of drug development and ADC experience
- Seasoned BD Team ready to quickly align on commercial and scientific deal structure

## VersAptx™ Platform: Bioconjugation Innovation

- Versatile and adaptable platform for rapid development of bespoke bioconjugates
- Tailored solutions for diverse cancer biologies, ensuring precision in treatment