CORPORATE OVERVIEW

February 2024

Vincerx PHARMA®

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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

Vincerx PHARMA

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

Roche · AMGEN · AstraZeneca · Acerta Pharma · ONYX PHARMACEUTICALS · NOVARTIS · Verastem Oncology · Genentech · PDL BioPharma

pharmacyclics An AbbVie Company · GILEAD · Bristol Myers Squibb · Lilly · janssen Pharmaceutical Companies of Johnson & Johnson · Seagen · ZIOPHARM Oncology · BAYER · Solasia Brightening the Future with Better Medicines for Cancer · CSL Behring Biotherapies for Life®

imbruvica® (ibrutinib)
560, 420, 280, 140 mg tablets | 140, 70 mg capsules
70 mg/mL oral suspension

$975M partnership with Janssen in 2011 ($150 up front, $825M in milestones)$21B acquisition of Pharmacyclics by AbbVie in 2015

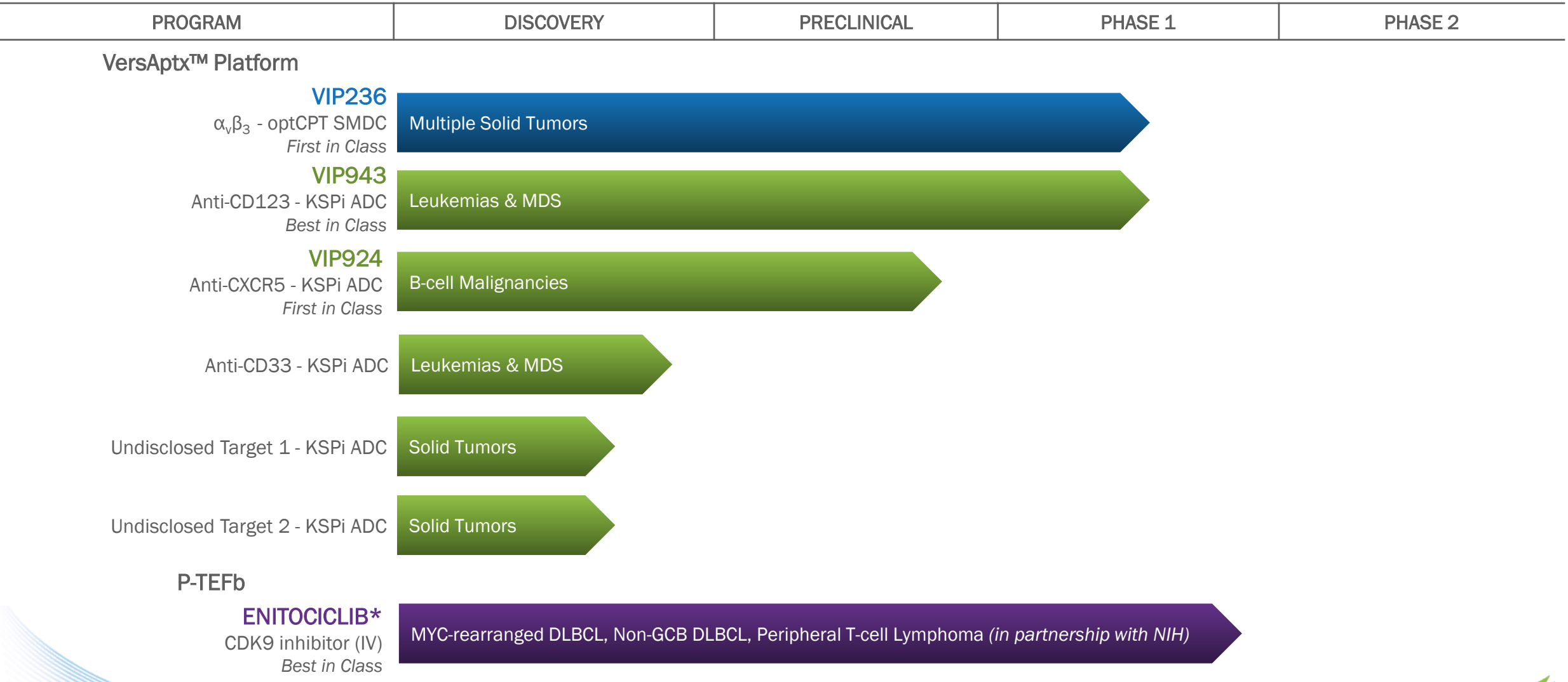
Management Team's Contribution: Developed ibrutinib from preclinical through phase 2 in <3 years. All 3 phase 2 studies garnered break through therapy designation and accelerated approvals

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

Our 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.

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Accelerating Programs Towards Strategic Milestones and Value Creation

VIP236

- In licensed from Bayer at candidate selection phase
- Completed all CMC steps for GMP material
- Completed and filed all IND studies
- Phase 1 dose escalation nearly complete
  - Favorable safety profile with Q3W schedule
  - Early signs of clinical activity with monotherapy
  - Monotherapy and combination therapy to be explored in Phase 1b/2

VIP943

- In licensed from Bayer at candidate selection phase
- Completed all CMC steps for GMP material
- Completed and filed all IND studies
- Phase 1 dose escalation in progress
  - Favorable safety and PK payload profile in initial cohorts
  - Early signs of target engagement (PK and PD data)
  - Monotherapy and combination therapy to be explored in Phase 1b/2

Enitociclib

- IND transferred from Bayer (Phase 1)
- 95 patients dosed across 4 studies
- Durable monotherapy efficacy established in DH-DLBCL and tFL
- Combination therapy potential in:
  - Ovarian cancer (clinical activity with monotherapy)
  - PTCL (high response rate with venetoclax/prednisone combination)
  - Safety profile = partner of choice for combo Tx

VIP924

- In licensed from Bayer at candidate selection phase
- Pharmacology studies suggest improved safety and efficacy compared with currently approved heme ADCs
- IND enabling studies in progress

ADC, Antibody–drug conjugates; CMC, Chemistry, manufacturing, and controls; DH-DLBCL, Double-hit Diffuse large B-cell lymphoma; GMP, Good Manufacturing Practices; IND, Investigational New Drug; PD, Pharmacodynamics; PK, Pharmacokinetics; PTCL, peripheral T-cell lymphoma; tFL, Transformed Follicular; Tx, Treatment.

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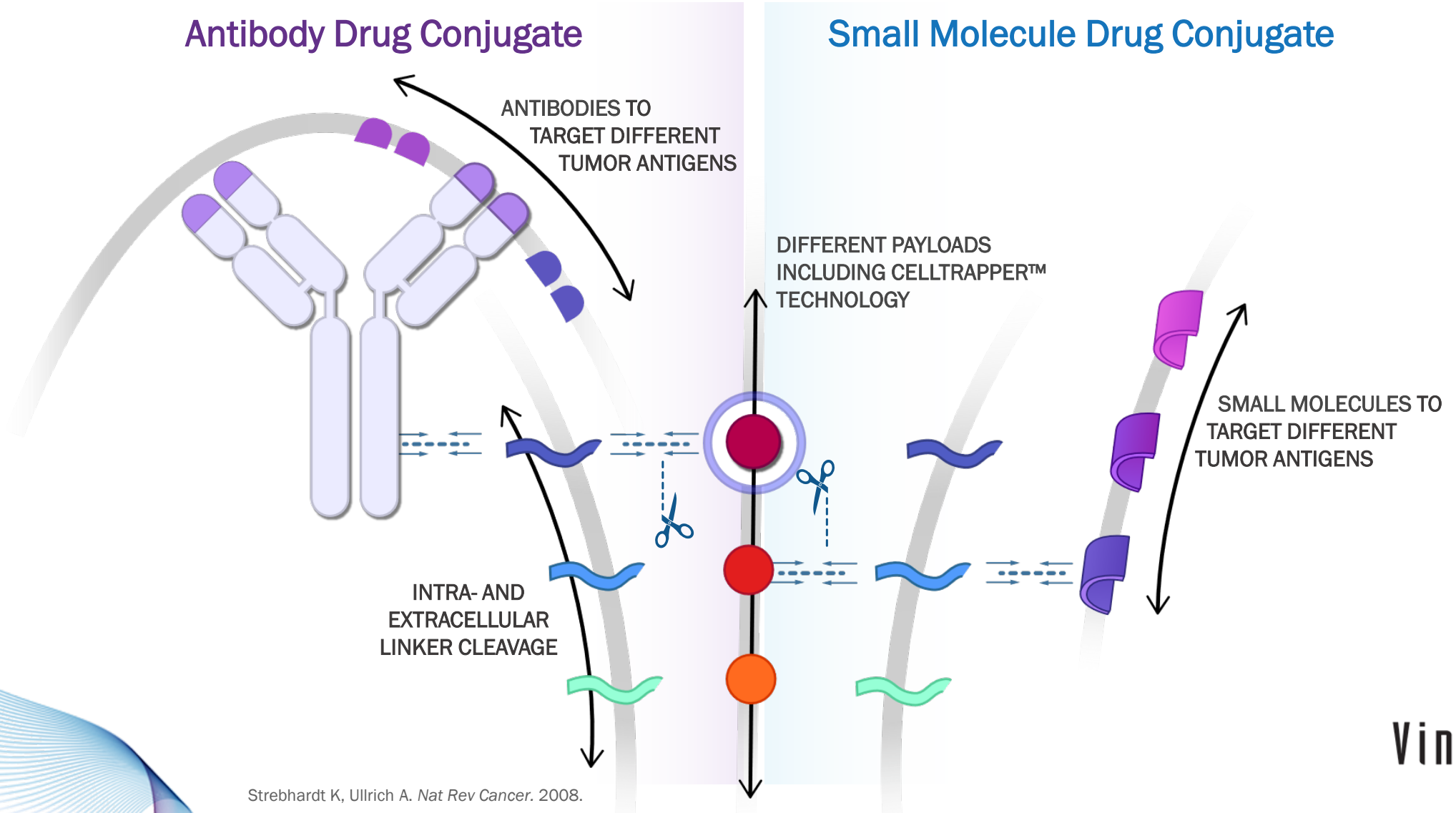
VersAptx™ PLATFORM

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

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



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

ADC Technology

ADC, antibody-drug conjugate.

The Promise Of Bioconjugate Therapies Has Not Been Realized

AS EVIDENCED BY SEVERE SIDE EFFECTS

	ASTRAZENECA	SEAGEN	IMMUNOGEN	ADC THERAPEUTICS	MERSANA THERAPEUTICS	LEGOCHEM BIOSCIENCES	BICYCLE
Binder	mAbs	mAbs	mAbs	mAbs	mAbs	mAbs	Bicyclic peptides
Linker	Cathepsin B cleavable, Furin cleavable, Stable	Cathepsin B cleavable, Glucuronidase cleavable	Stable (DM1), Cleavable (Disulfide bond)	Cathepsin B cleavable	pH sensitive self-cleaving	Glucuronidase cleavable	Cathepsin B cleavable
Payloads	MMAE, MMAF, Tubulysine, Camptothecin, PBD	MMAE, MMAF, PBD	DM1, DM4	PBD	Auristatin (MMAE)	MMAF, PBD	MMAE, DM1
Permeability	Permeable, Non-permeable (MMAF)	Permeable, Non-permeable (MMAF)	Permeable	Permeable	DolaLock (Time dependent)	Permeable, Non-permeable (MMAF)	Permeable
Highest Development status	Approved	Approved	Approved	Approved	Phase 1	Phase 3	Phase 1/2

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

ADC, antibody-drug conjugate; mAbs, monoclonal antibodies; MMAE, monomethyl auristatin E; DM1/4, maytansinoid payloads; MMAF, monomethyl auristatin F; PBD, pyrrolobenzodiazepine.

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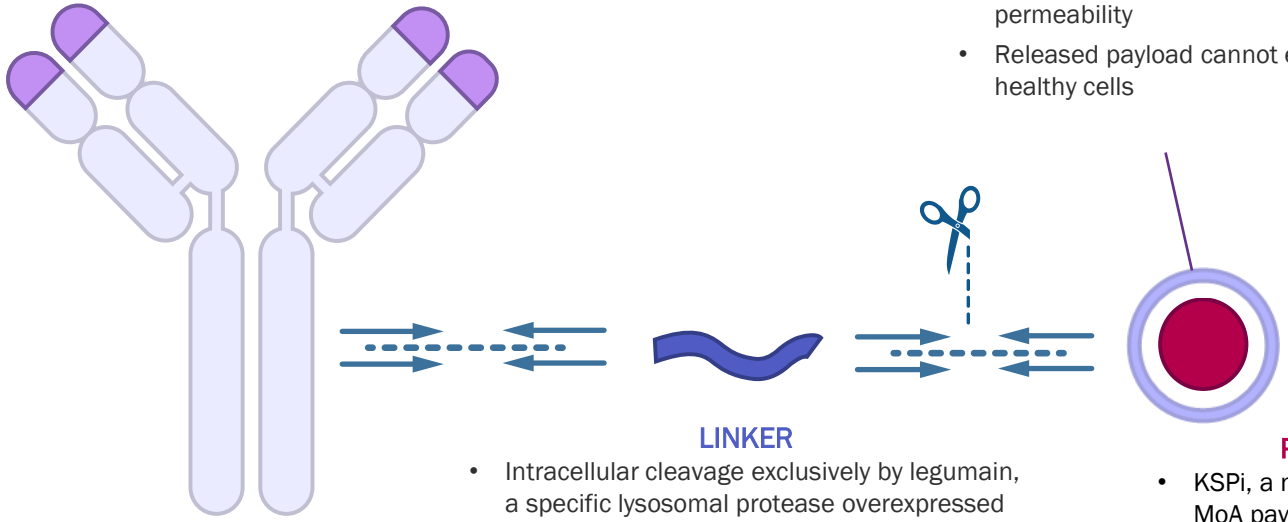
Solving ADC Challenges With Our Innovative VersAptx™ Platform

INCREASING THE THERAPEUTIC WINDOW BY IMPROVING EFFICACY AND SAFETY

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

ADC, antibody-drug conjugate; DAR, drug antibody ratio; KSPi, kinesin spindle protein inhibitor; MoA, mechanism of action; PK, pharmacokinetics.

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Sum of All Parts of Our Technology Is Designed to Address Safety Liabilities of Approved ADCs

	MYLOTARG™	BESPONSA®	POLIVY®	ADCETRIS®	ENHERTU®	Legumain - KSPi ADCs		
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

ADC, antibody-drug conjugate; KSPi, kinesin spindle protein inhibitor; PML, progressive multifocal leukoencephalopathy; VOD, veno-occlusive disease.

Source: Drugs@FDA

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VIP943
CD123-KSPi ADC

ADC, antibody-drug conjugate; KSPi, kinesin spindle protein inhibitor.

VIP943 Overview

Best-in-Class ADC targeting CD123 with a novel and differentiated linker and payload combination positioned to deliver improved safety and efficacy

Clinical Path

- Acute myeloid leukemia (AML)
- Myelodysplastic syndrome (MDS)
- Other CD123+ heme malignancies

Current Trials

Phase 1 dose escalation in AML and MDS

Market Opportunity

- AML incidence: 16.3K (US)/ 21.1K (EU)
- MDS incidence: 9.9K (US)/ 9.3K (EU)

Near Term Milestones

- Preliminary data: mid 2024
- Dose optimization: Late 2024 based on funding/partnering

Market opportunity in patients / year. Data based on CI5XII, RKI and SEER*

VIP943
CD123-KSPi

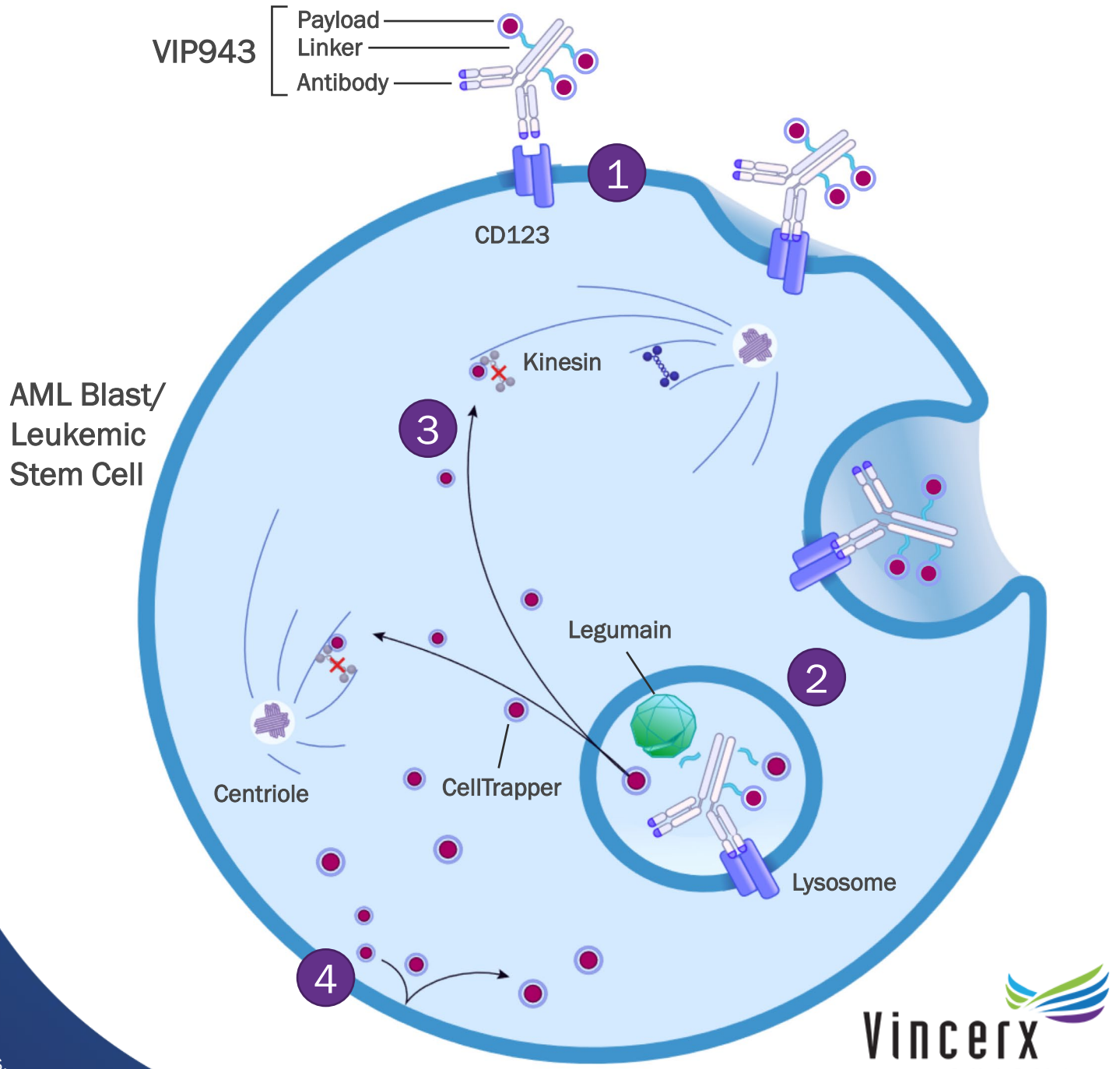
ANTIBODY-DRUG CONJUGATE FOR TREATMENT OF AML & MDS

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

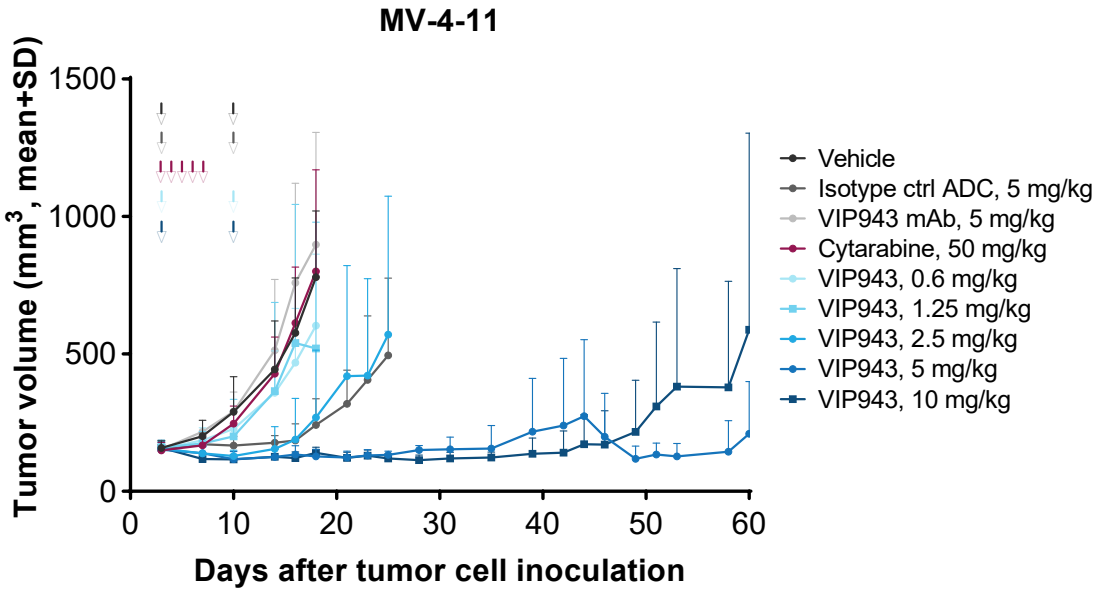
4. CellTrapper® modified payload is hydrophilic and accumulates in the tumor cell for improved safety and tolerability for long-term therapy and targeting leukemic stem cells

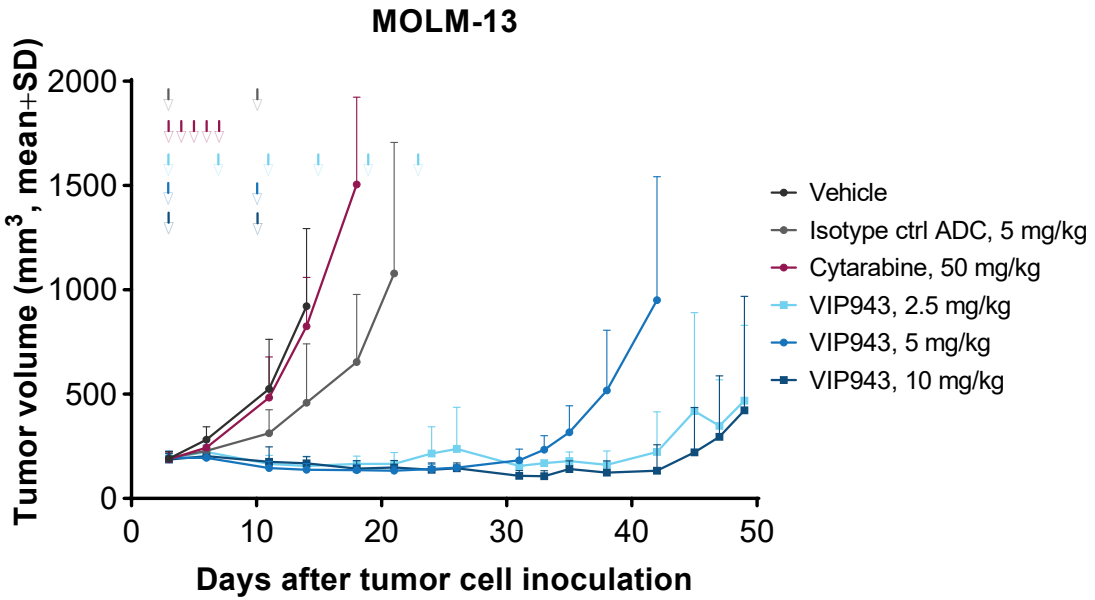


Ab, antibody; AML, acute myeloid leukemia; KSPi, kinesin spindle protein inhibitor; MDS, myelodysplastic syndrome.

VIP943 Shows Dose-Dependent Anti-Leukemia Efficacy in In Vivo Mouse Models of AML

ACTIVITY IN CELL-DERIVED XENOGRAFT AML MODELS OF VIP943 WITH LONG DURABILITY





- Models tested limited duration treatment and showed dose-dependent tumor regression
- Potent cytotoxic activity on multiple hematological cell-derived xenograft models
- High tolerability as measured by lack of weight loss is observed in all studies

CDX, cell-derived xenograft.

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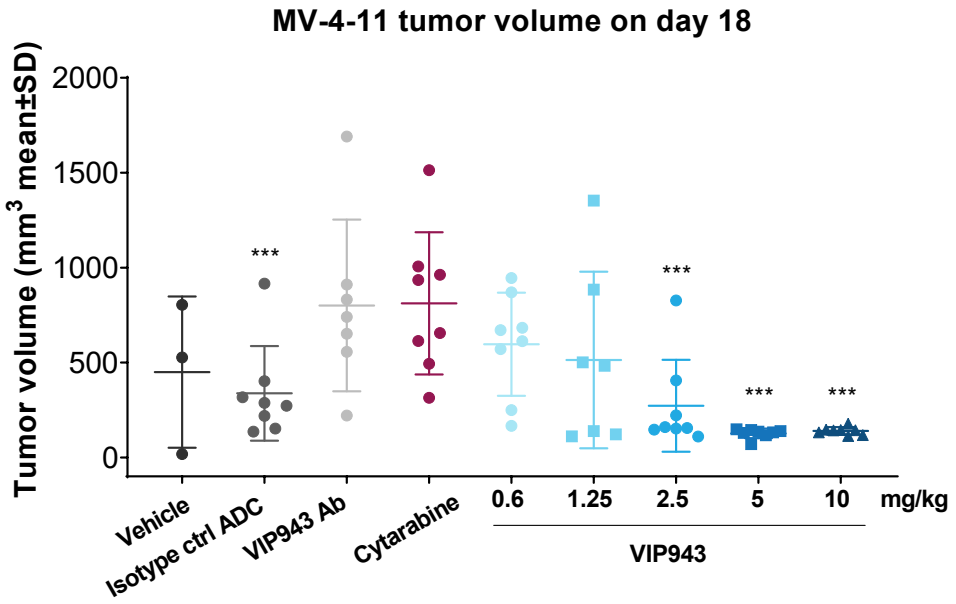
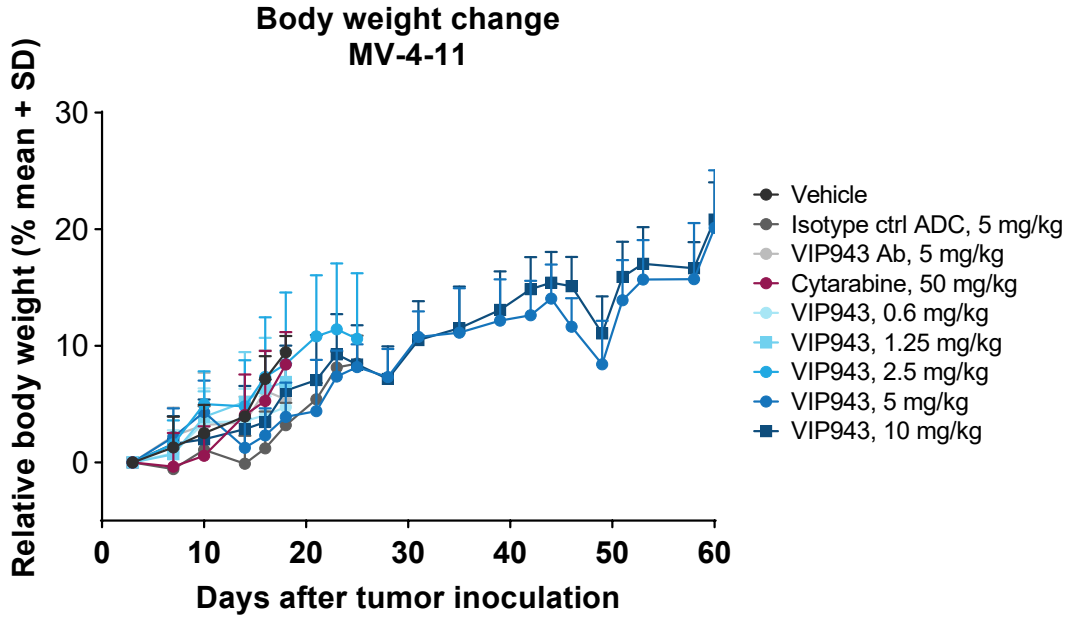
High Tolerability of VIP943 Treatment is Observed with AML Models





- Concentration-dependent reduction in tumor volume
- No negative impact on body weight

AACR Poster VIP943 2019

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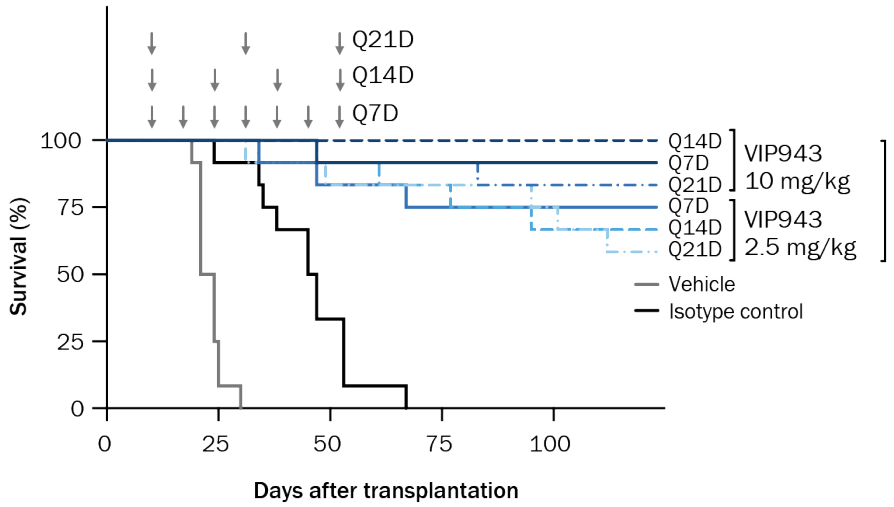
VIP943 Increases Survival in AML Models

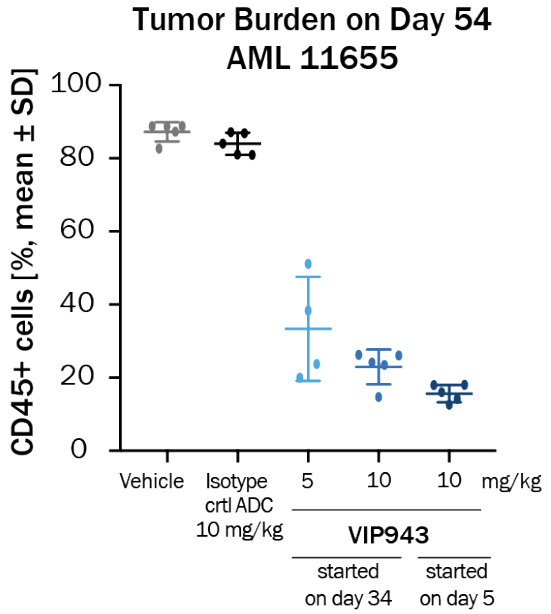
AML CELL-LINE (CDX) AND PATIENT-DERIVED (PDX) TUMOR MODELS TREATED WITH TARGETED ADC VS ISOTYPE CONTROL ADC

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

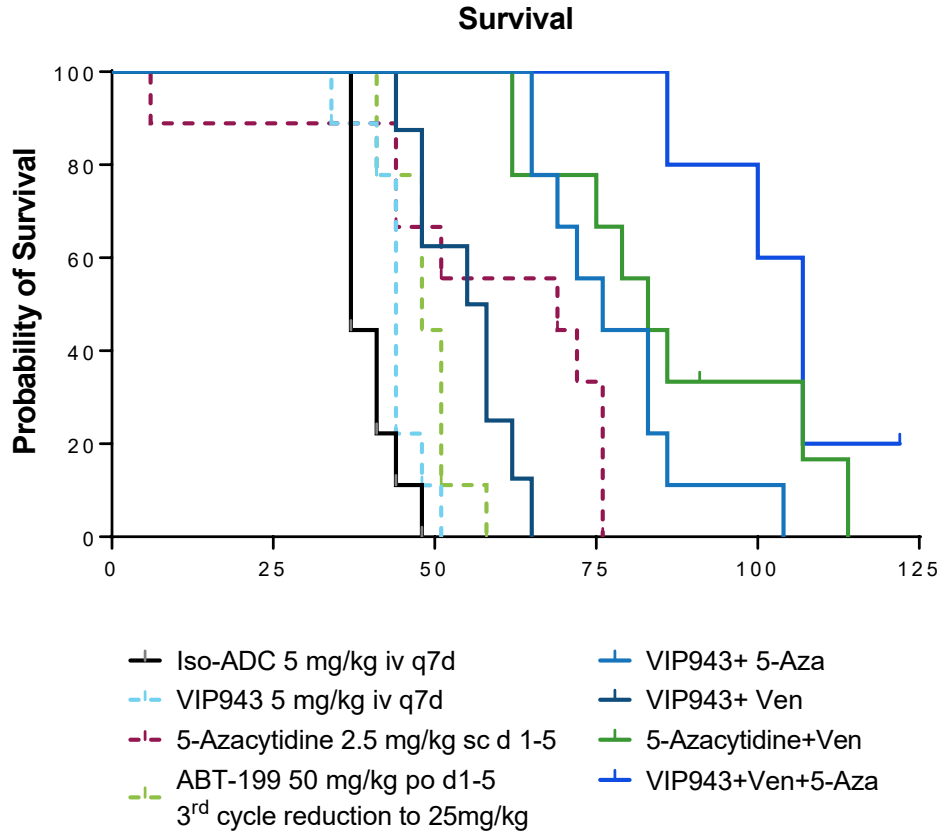
Reduction in Tumor Burden in AML PDX Model



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

ADC, antibody-drug conjugate; AML, acute myeloid leukemia; QXD, every X days.

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



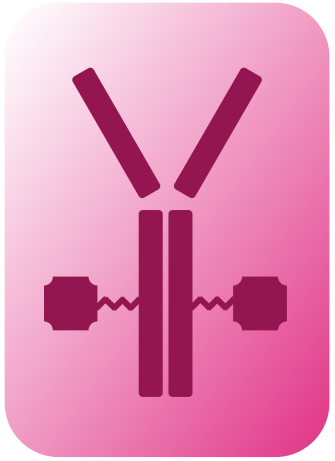
Treatment	Survival Time (Days)
Iso-ADC	37
VIP943+Ven	57
VIP943+Aza	76
Ven+ Aza	83
VIP943+Ven+Aza	>107

- 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

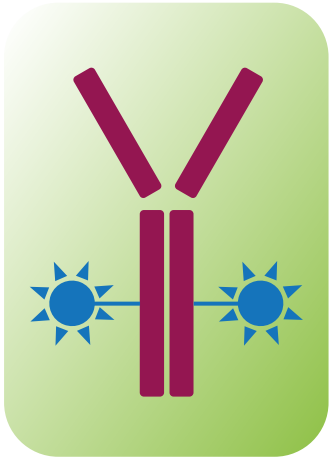
AZA, azacitidine; CR, complete response; PDX, patient derived xenograft; VEN, venetoclax.

CR: No visible tumor

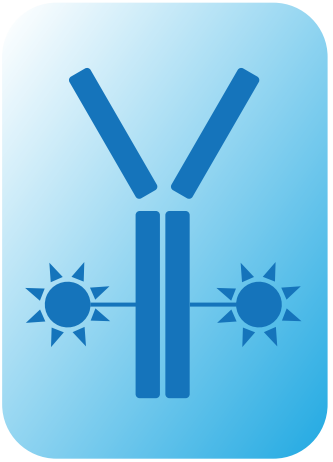
Safety Study in Monkeys Comparing VIP943 to Mylotarg™



Mylotarg (Gemtuzumab-Ozogamicin)
- 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

Study to compare the safety profile of VIP943, Mylotarg and the Mylotarg anti-CD33mAb combined with the legumain-KSPi payload in monkeys after a single dose

DAR, drug antibody ratio; KSPi, kinase spindle protein inhibitor; mAb, monoclonal antibody.

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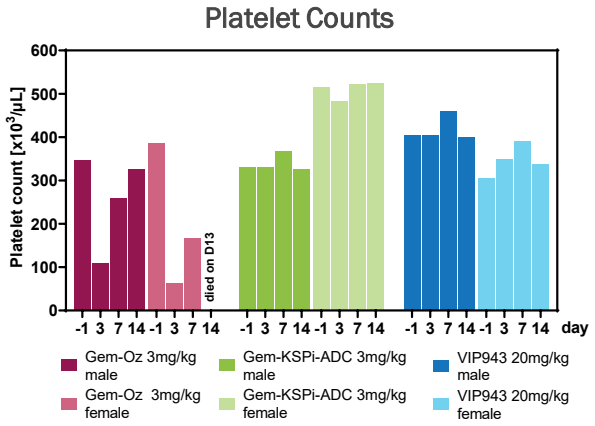
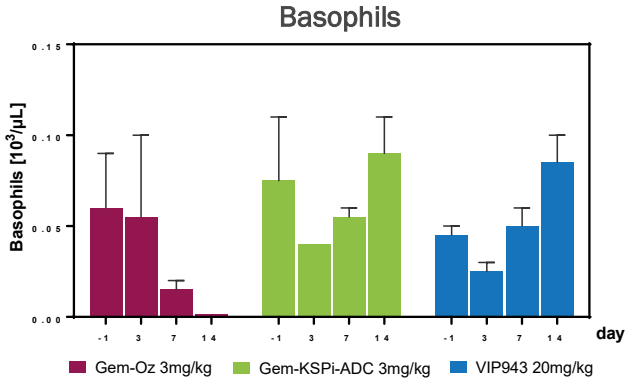
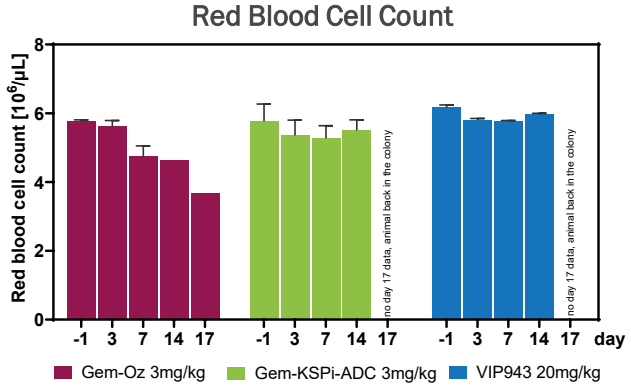
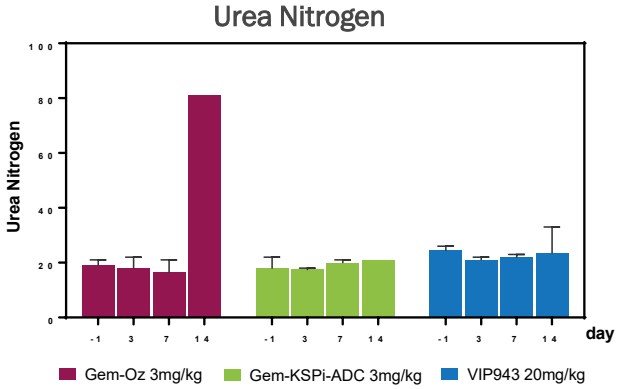
VIP943 Displays an Improved Safety Profile in NH-Primates When Compared to Mylotarg (Gemtuzumab-Ozogamycin)

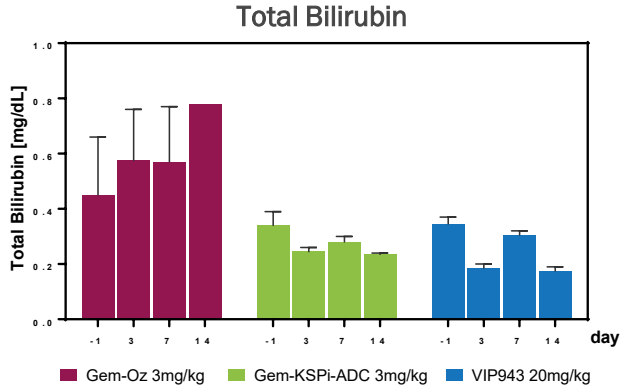
LEGUMAIN-KSPi PAYLOAD CAN IMPROVE THE SAFETY PROFILE OF MYLOTARG

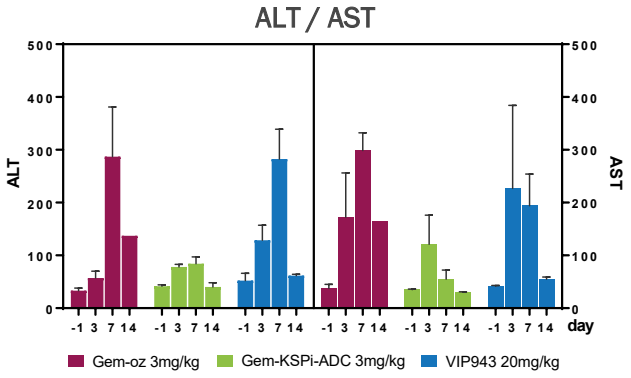












- Critical drop of platelet counts and red blood cell count with insufficient recovery in the Mylotarg group
- Increased liver enzymes and severe increase in total bilirubin for animals treated with Mylotarg, indicating liver toxicity
- Extreme increase of urea nitrogen, indicating kidney toxicity in 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.

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VIP943 First-in-Human Dose-Escalation Study

VNC-943-101

Open-label, multicenter, Phase 1, first-in-human (FIH), dose-escalation and dose-optimization study of VIP943 in subjects with relapsed or refractory CD123+ acute myeloid leukemia (AML), myelodysplastic syndrome (MDS) and B-cell acute lymphoblastic leukemia (B-ALL) [NCT06034275]

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VIP924
CXCR5-KSPi ADC

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VIP924 Overview

First-in-class ADC with novel CXCR5 target delivers enhanced efficacy and safety

Clinical Path

- Non–Hodgkin lymphoma (NHL)
- Chronic Lymphocytic Leukemia (CLL)

Current Trials

N/A

Market Opportunity

- Non–Hodgkin lymphoma 81K (US)/ 38K (EU)
- CLL 15K (US)/20K (EU)

Near Term Milestones

IND 2025 pending funding/partnerships

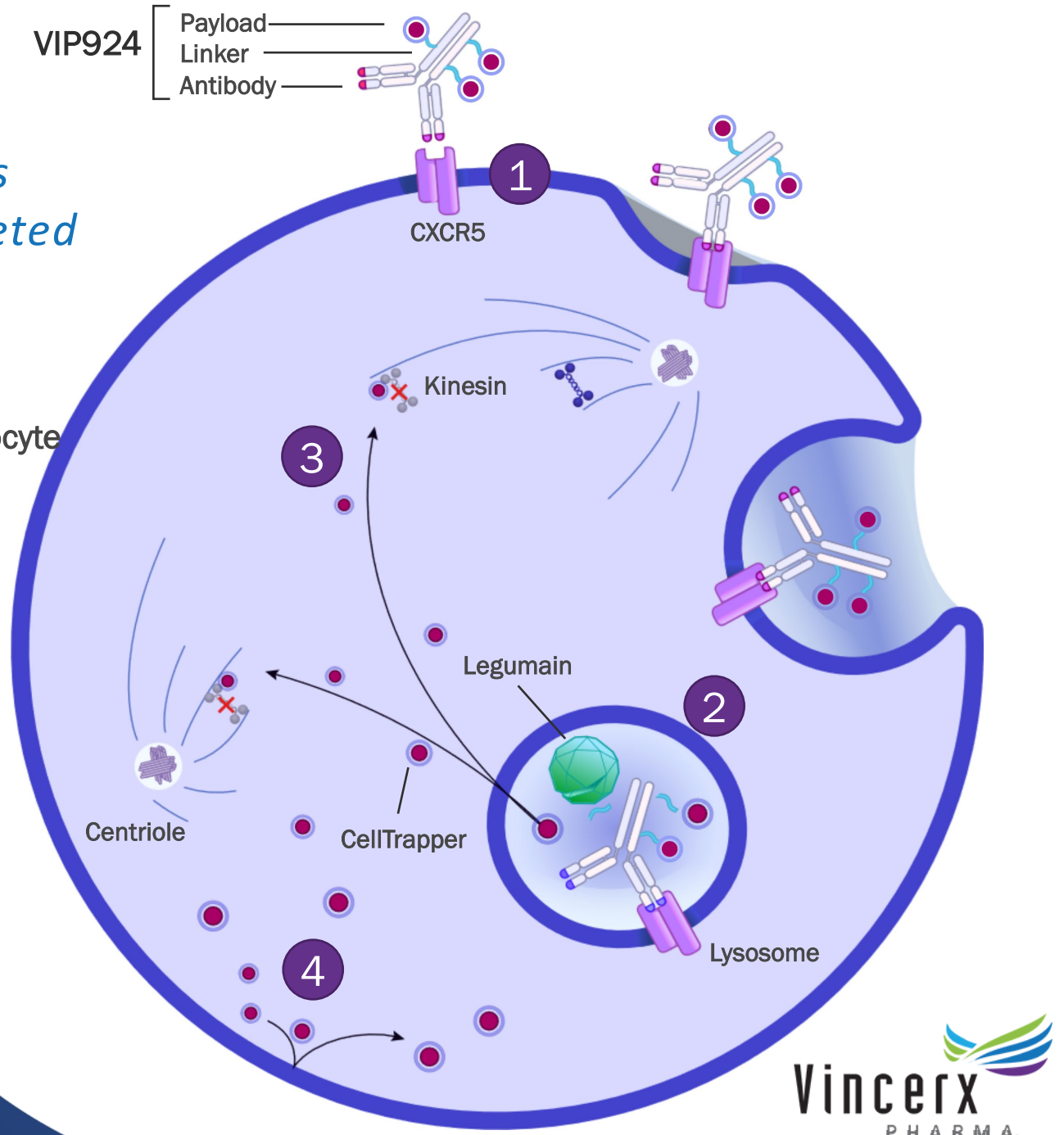
Market opportunity in patients / year. Data based on CI5XII, RKI and SEER*

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



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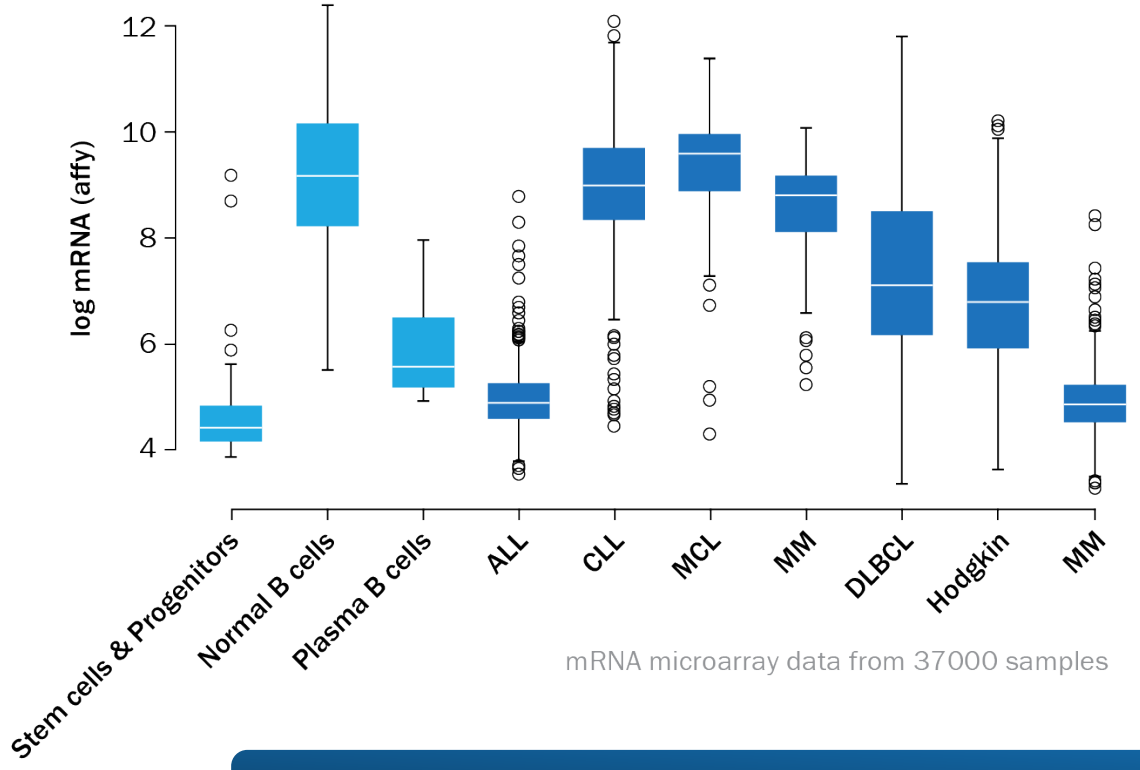
Ab, antibody; KSPi, kinesin spindle protein inhibitor.

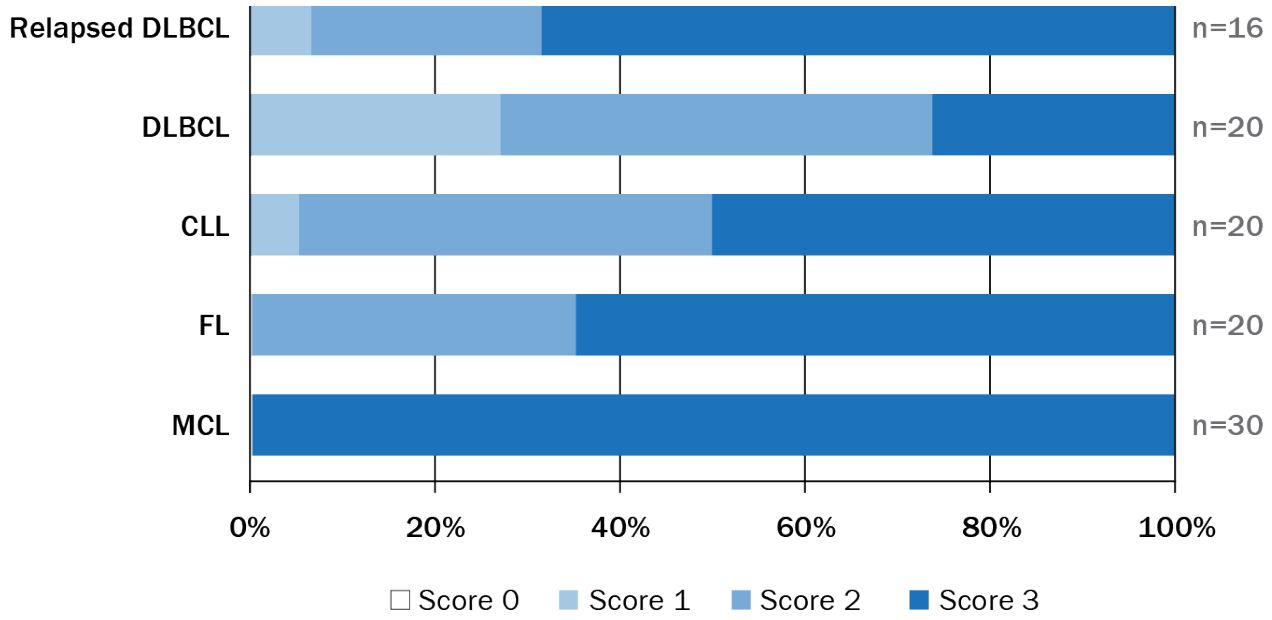
CXCR5 Is Expressed in B-Cell Malignancies

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)



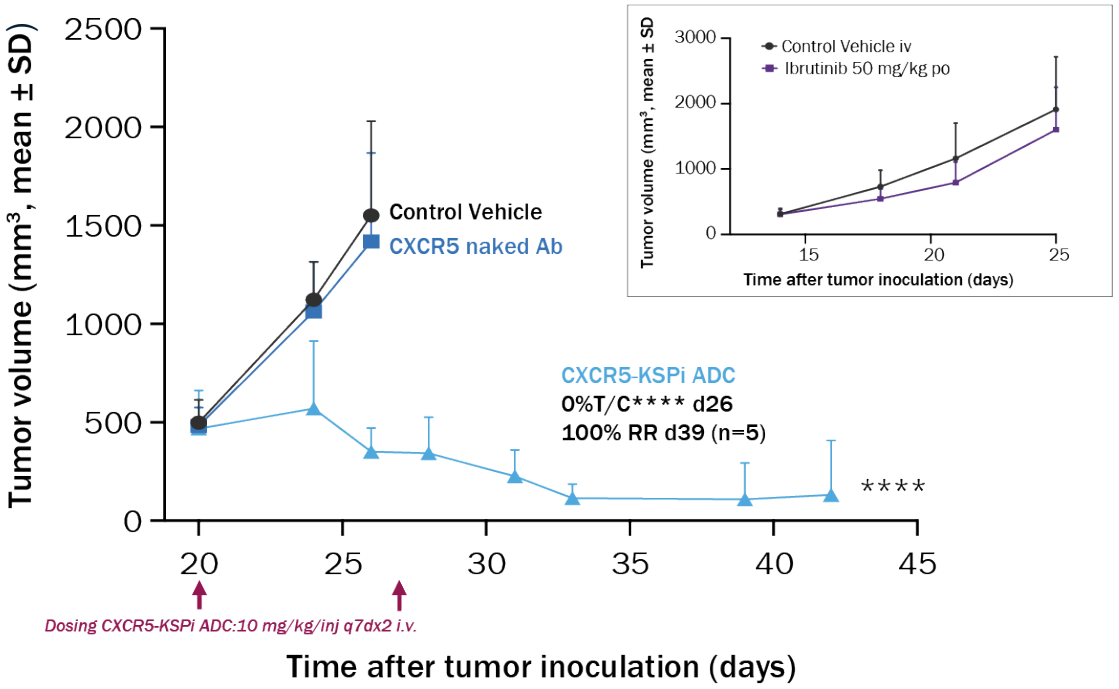
IHC analysis of samples from patients with hematologic malignancies shows CXCR5 expression in MCL, DLBCL, FL and CLL

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.

VIP924 Induces Sustained Tumor Regression in MCL and DLBCL Models

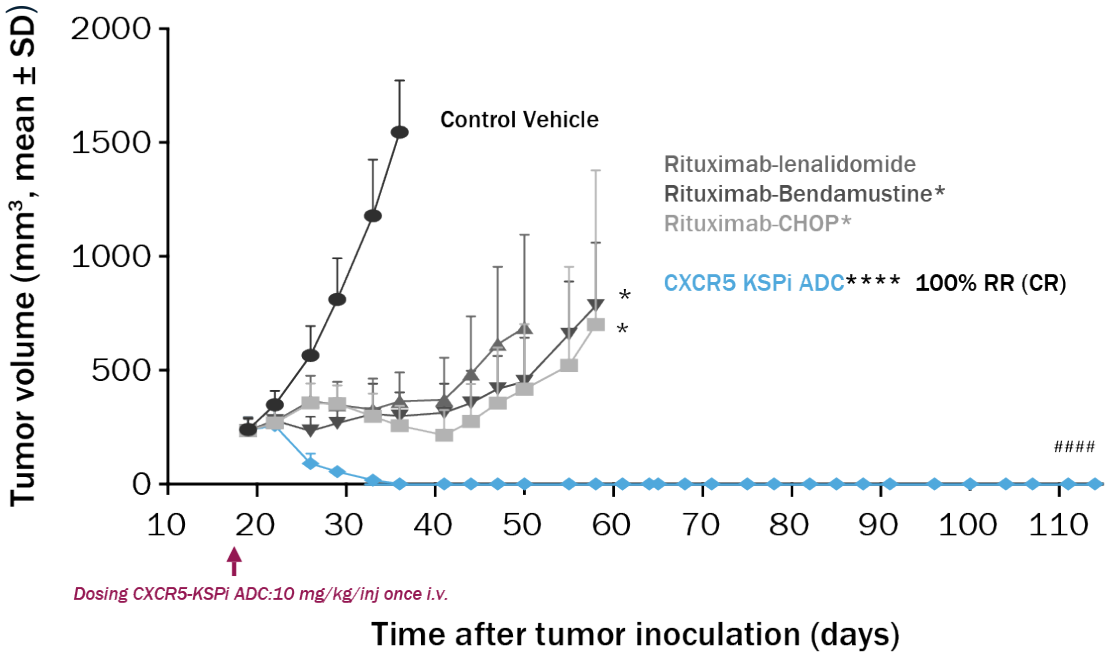
VIP924 Is Active in Ibrutinib-Refractory MCL In Vivo Model



- 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

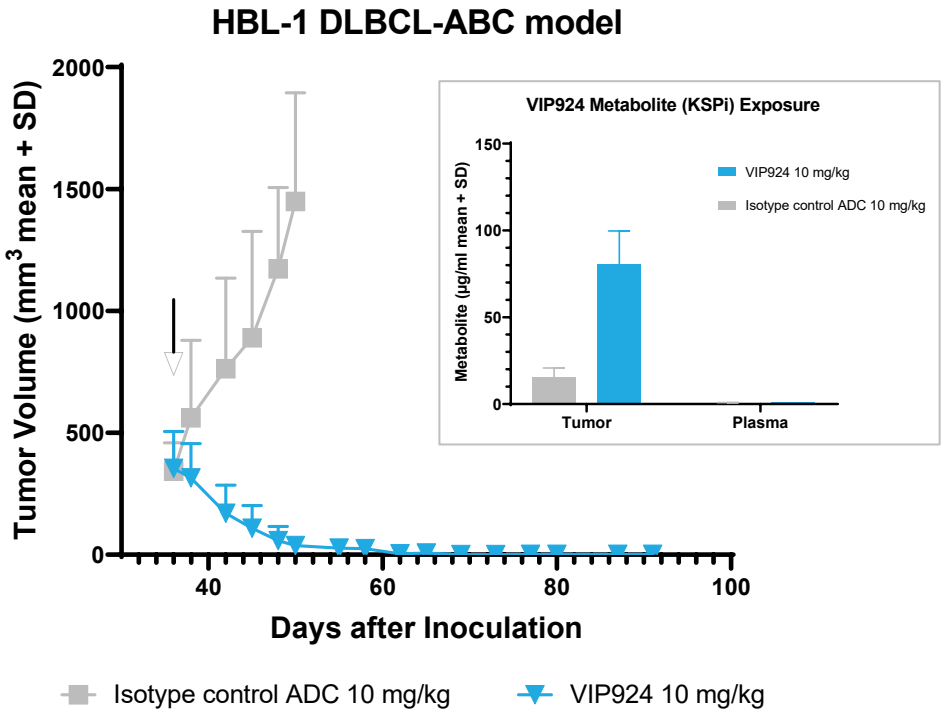


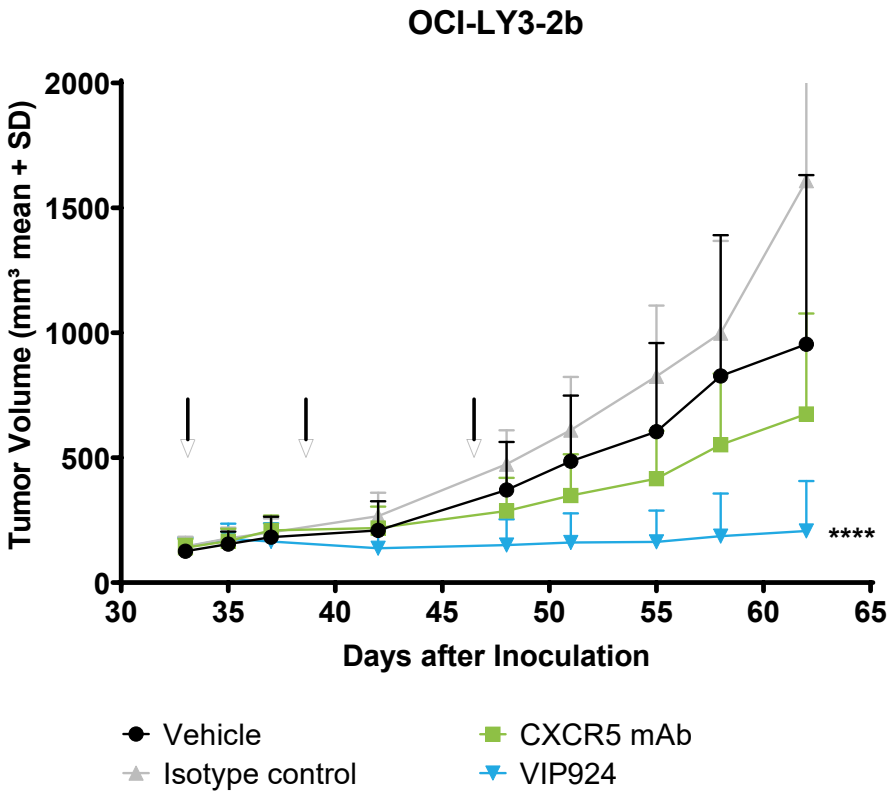
- Complete regression with single dose of VIP924 in CXCR5+ model OCI-LY1 (day 114)
- Superior activity versus SOC

*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.

Ab, antiobody; ADC, antibody-drug conjugate; CDX, cell-line; CHOP, cyclophosphamide, doxorubicin, vincristine, prednisone; CR, complete response; DLBCL, diffuse large B-cell lymphoma; i.v., intravenous; KSPi, kinesin spindle protein inhibitor; MCL, mantle-cell lymphoma; SOC, standard of care; T/C, treatment-to-control ratio.

Significant Anti-Tumor Efficacy of VIP924 in Established Large Tumor in the HBL-1 Lymphoma CDX 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
- VIP924 induced potent anti-tumor effect in the ABC-like DLBCL OCI-LY3-2b model which shows weak to moderate CXCR5 expression

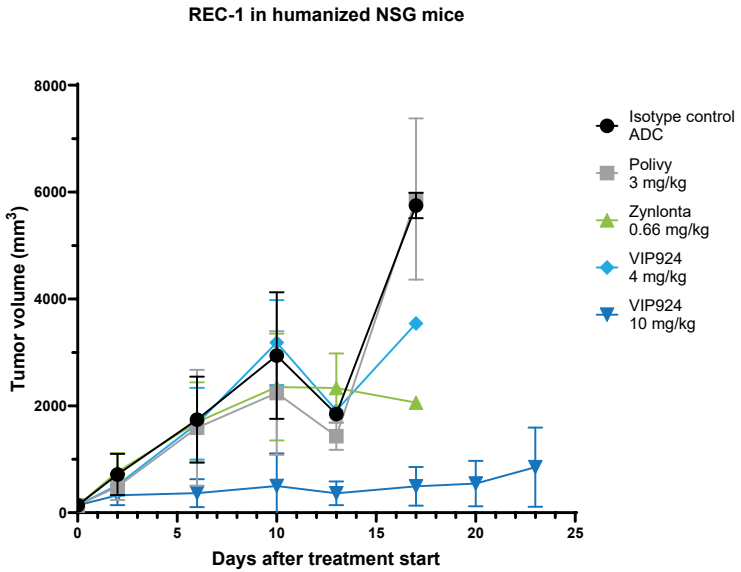
AACR Poster 2023

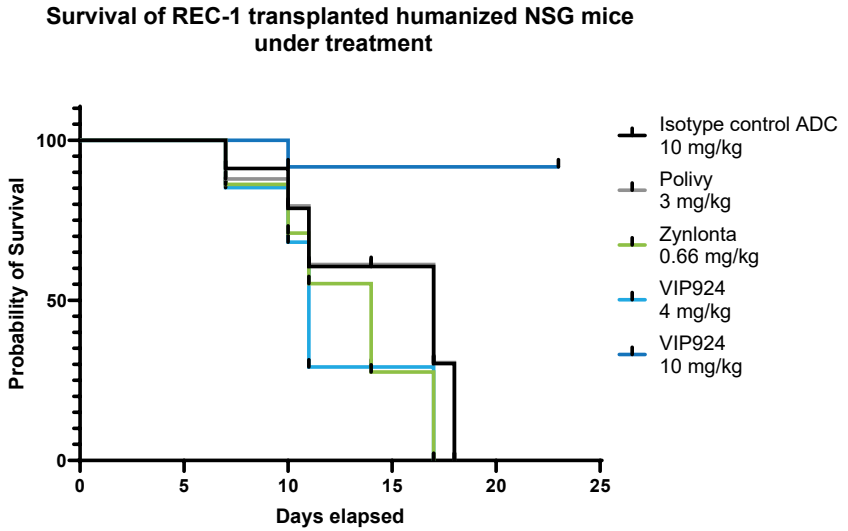
CDX, cell derived.xenograft

Vincerx PHARMA

In Vivo Evaluation of VIP924 in Mantle Cell Lymphoma Mouse Model Shows Superiority over Polivy® and Zynlonta®

REC-1 CELL LINE IN HUMANIZED NSG MICE





- Only animals treated with VIP924 showed a significant tumor growth inhibition and a survival benefit as compared to control treated animals.
- Polivy and Zynlonta doses were selected based on the literature as effective doses in mouse xenograft experiments and higher doses lead to toxicity in these models.
  - VIP924 data are based on a dose level of 10 mg/kg compared with 3 mg/kg for Polivy and 0.66 mg/kg for Zynlonta.
  - In humans, Polivy is given with 1.8mg/kg every 21 days, Zynlonta with 0.15mg/kg also every 21 days.
- In Zynlonta-treated animals, white blood counts, monocytes, and lymphocytes were reduced at the end of treatment. VIP924 treatment showed only minor to no effects on these cell populations.

VIP236
$\alpha_v\beta_3$-optCPT SMDC

optCPT, optimized camptothecin; SMDC, small molecule drug conjugate.

VIP236 Overview

First-in-class small molecule drug conjugate designed for deep penetration in the tumor tissue with fast uptake

Clinical Path

- Ovarian & Endometrial
- Gastric
- Bone metastasis (TNBC and Lung)
- Glioblastoma

Current Trials

Phase 1 dose escalation in metastatic solid tumors

Market Opportunity

- Ovarian 26K (US)/ 40K (EU)
- Endometrial 65K (US)/ 34K (EU)
- Gastric 14K (US)/ 21K (EU)
- GBM 15K (US)/ 21K (EU)
- Bone Mets 62K (US)

Near Term Milestones

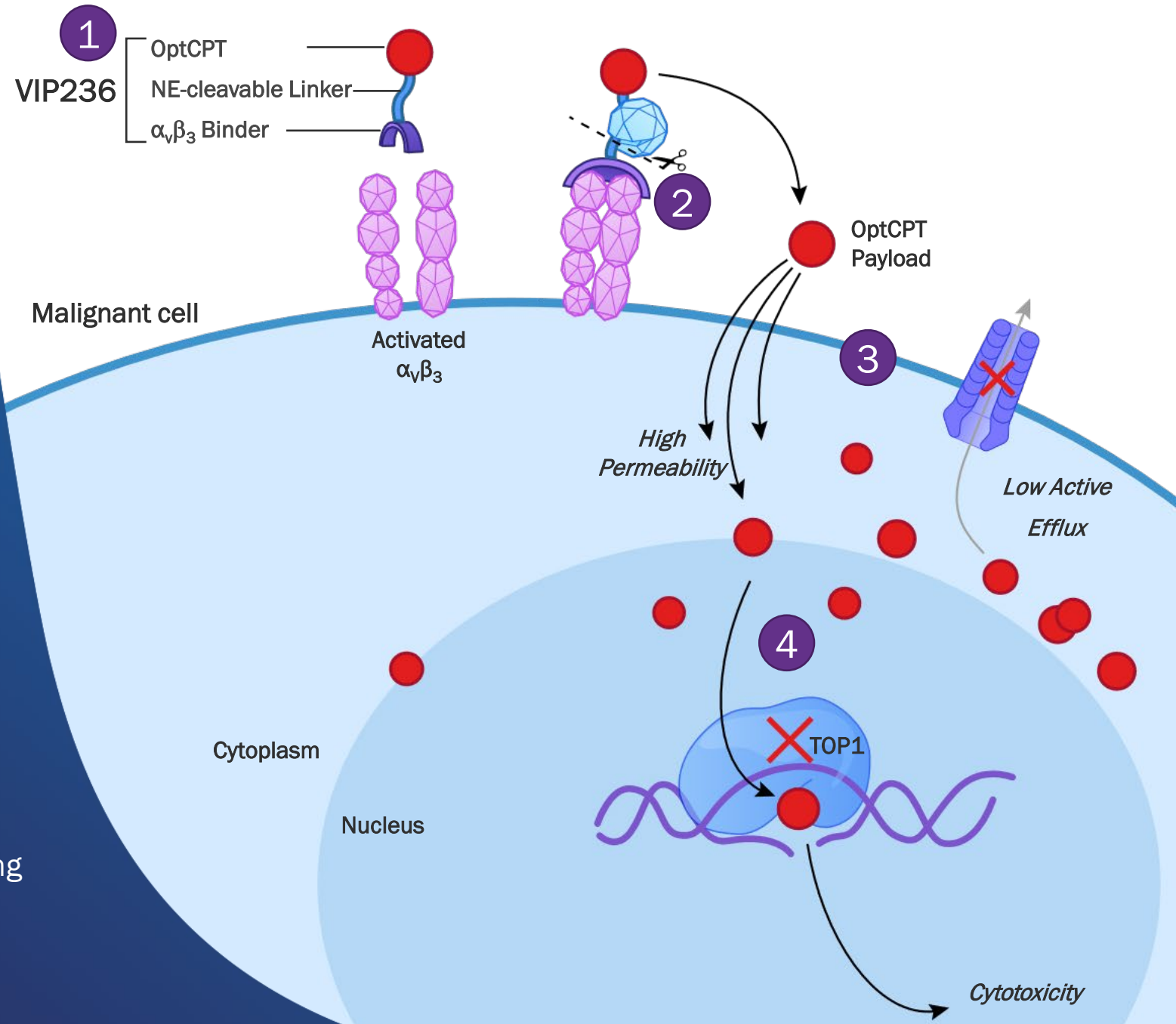
- PK and Safety: Early 2024
- Dose optimization: Late 2024 based on funding/partnering

Market opportunity in patients / year. Data based on CI5XII, RKI and SEER*

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

ENHANCED SAFETY AND PRECISION PROFILE

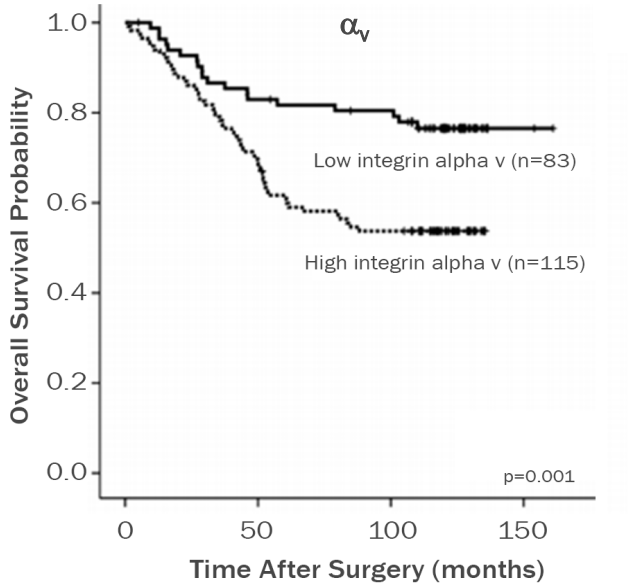
1. VIP236 is an $\alpha_V\beta_3$ integrin binder linked to an optCPT payload
2. Payload is released by the enzyme NE in the tumor microenvironment
3. The payload accumulates in the tumor cell due to high permeability and resistance to drug transporters
4. The payload inhibits topoisomerase 1 causing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

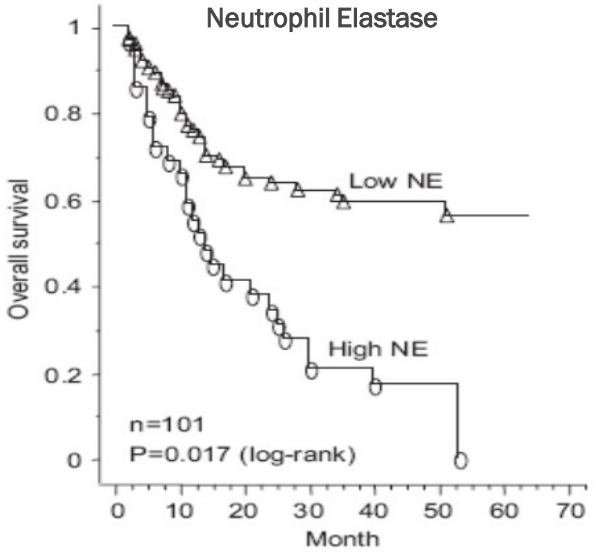


NE, neutrophil elastase; optCPT, optimized camptothecin; TOP1, topoisomerase 1.

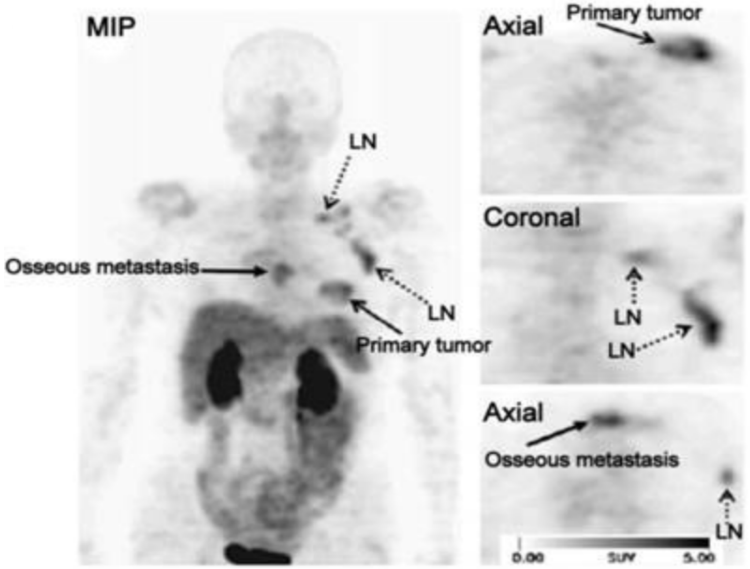
Expression of $\alpha_v\beta_3$ and Neutrophil Elastase is Associated With Poor Prognosis in Solid Tumor Indications

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





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



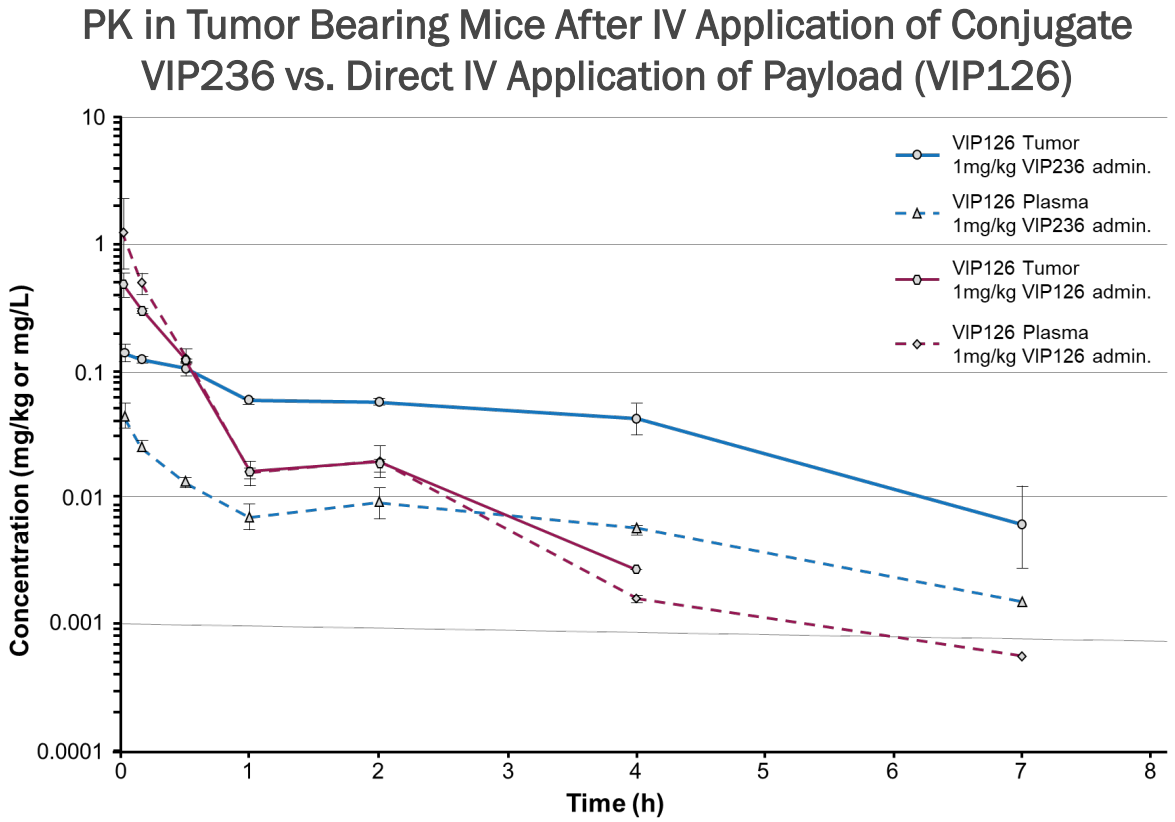
- $\alpha_v\beta_3$ is absent on resting endothelial cells and healthy organs
- High expression on activated endothelial cells and in advanced and metastatic tumors
- Expression correlates with poor prognosis in CRC and in other indications
- Anti-angiogenic therapies targeting $\alpha_v\beta_3$ showed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

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

Ha SY, et al. *J Clin Pathol.* 2014.
Chen H, et al. *Theranostics.* 2016.

Vincerx PHARMA

Administration of VIP236 Leads to Higher Tumor Exposure to the Payload (VIP126)

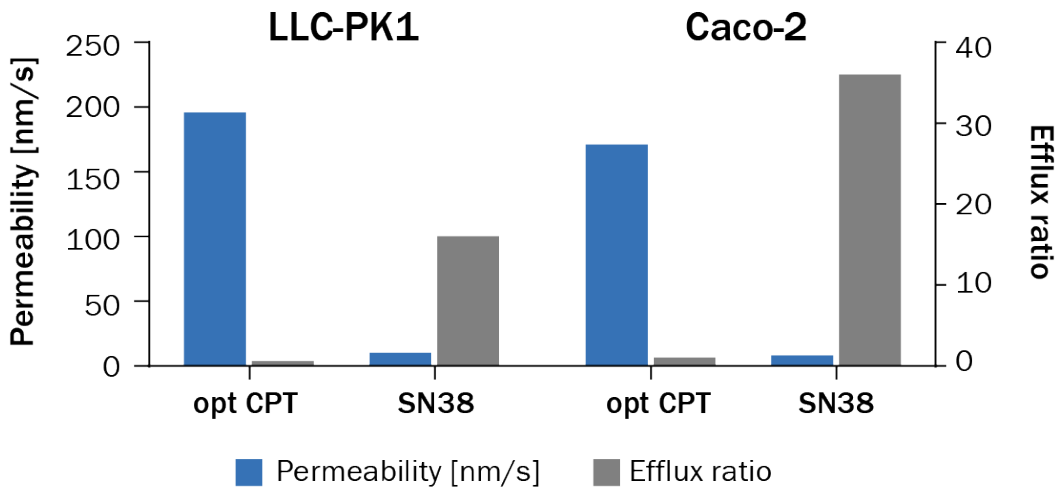


- VIP236 administration leads to higher tumor exposure to the payload (10.8-fold higher tumor plasma ratio) compared to direct payload administration
- Improvement is based on VIP236 targeted delivery to the tumor and selective release of the payload in the tumor microenvironment

IV, intravenous; PK, pharmacokinetics.

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

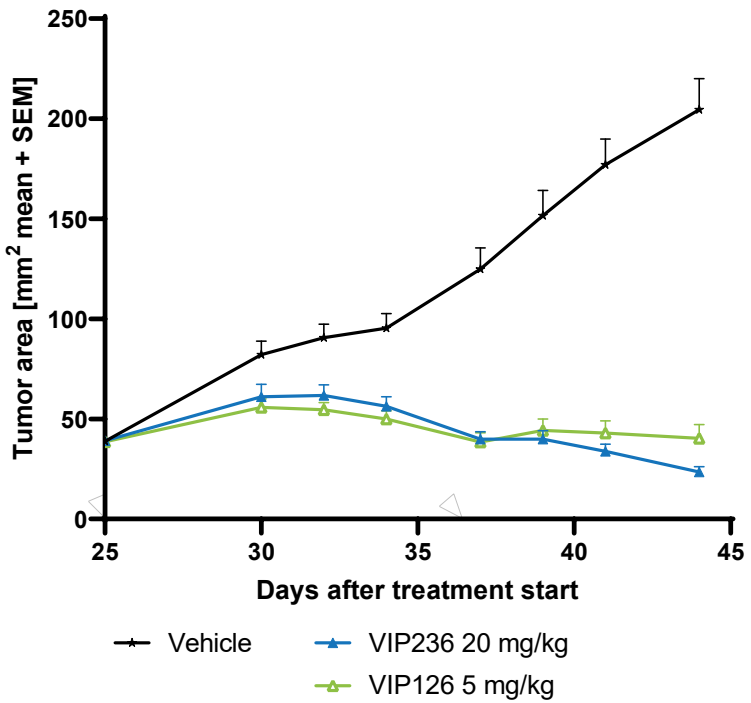
	IC50 (nM)		
Compound	NCI-H1975	NCI-H1975 — P-gp	NCI-H1975 — BCRP
SN38	45	141	512
OptCPT	19	34	27

- Payload: structurally related to the active metabolite of irinotecan known as SN38
- The payload of VIP236 is optimized for high permeability with low 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

BCRP; breast cancer resistance protein; IC50, half maximal inhibitory concentration; OptCPT, optimized camptothecin; P-gp, P-glycoprotein.

Improved Exposure Translates Into Improved In Vivo Efficacy and Safety

LUNG CANCER CDX NCI H69 IN VIVO MOUSE MODEL



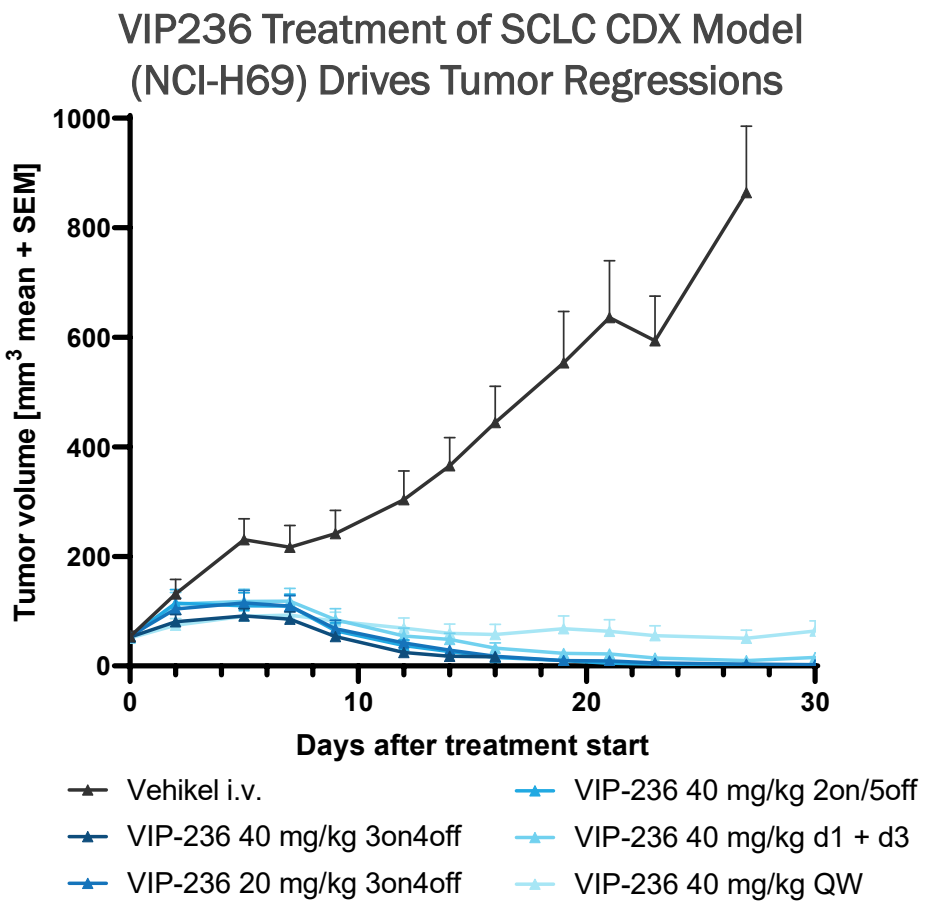
Dose / Schedule	Max Body Weight Loss	CR	PR	SD	PD	T/C Volume	Response Rate
Vehicle PBS i.v.	-1%	0	0	0	10	1.00	0.0%
VIP236 i.v. 20 mg/kg 2on5off	-1.21%	0	8	2	0	0.04	66.7%
VIP126 i.v. 5 mg/kg 2on5off	-5.75%	0	3	3	4	0.09	25.0%

- VIP236 significantly improved response rate (66.7% vs 25%) compared to VIP126 in the NCI H69 (SCLC)mouse model
- Body weight loss as first sign of adverse effects is significantly reduced (about 5-fold lower)
- Both improvements are based on VIP236 targeted delivery to the tumor

CDX, cell line derived; SCLC, small cell lung cancer.

Vincerx PHARMA

Complete Remission Achieved with all Schedules and Doses of VIP236 Tested



Optimized Dose and Schedule Improves Complete Response Rate to VIP236 (2on5off)

Dose/ Route Treatment schedule	Max. Body weight loss	CR	PR	SD	PD	T/C volume
Vehicle	-4.28%	0	0	0	12	1.00
VIP236 40mg/kg QD 3on4off	-10.14%	1	11	0	0	0.04
VIP236 20mg/kg QD 3on4off	-5.42	6	5	0	1	0.00
VIP236 40mg/kg QD 2on5off	-4.79	7	5	0	0	0.00
VIP236 40mg/kg QW 2on5off	-4.59	3	3	2	4	0.06

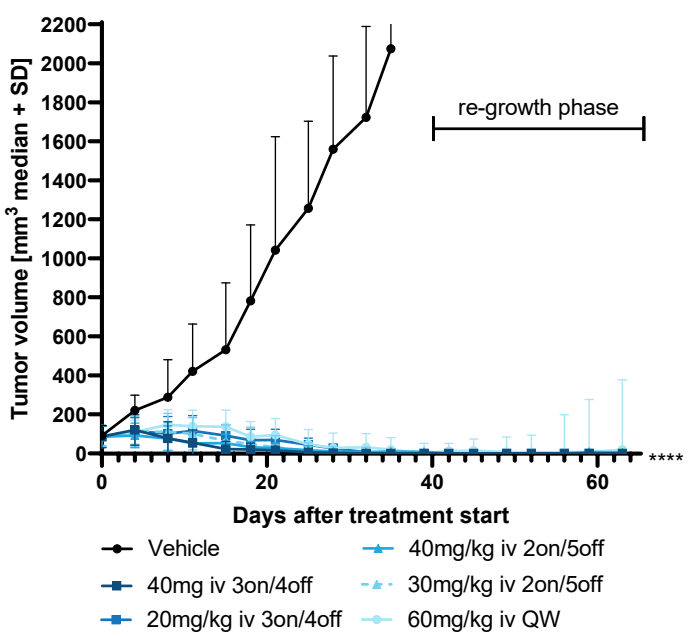
CR = complete response, PR = partial response, SD = stable disease, PD = progressive disease, T/C = treatment vs control,

Vincerx PHARMA

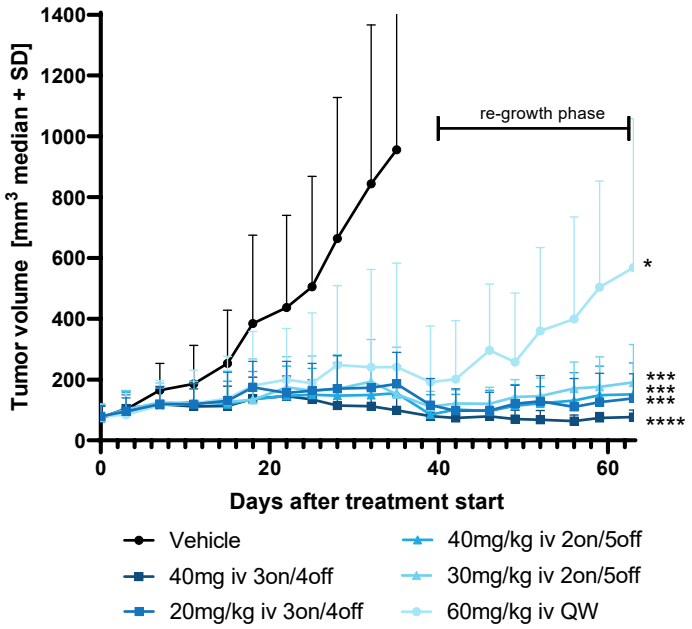
VIP236 Induces Tumor Regression in Patient Derived Xenograft Models Across Indications

DOSE AND SCHEDULE OPTIMIZED IN HARD TO TREAT & INVASIVE MODELS

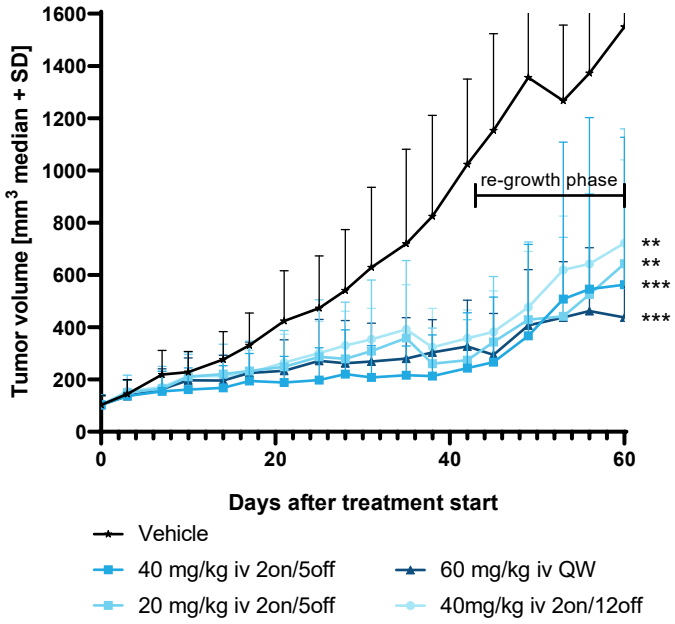
NSCLC



RCC



Liver metastasis from CRC

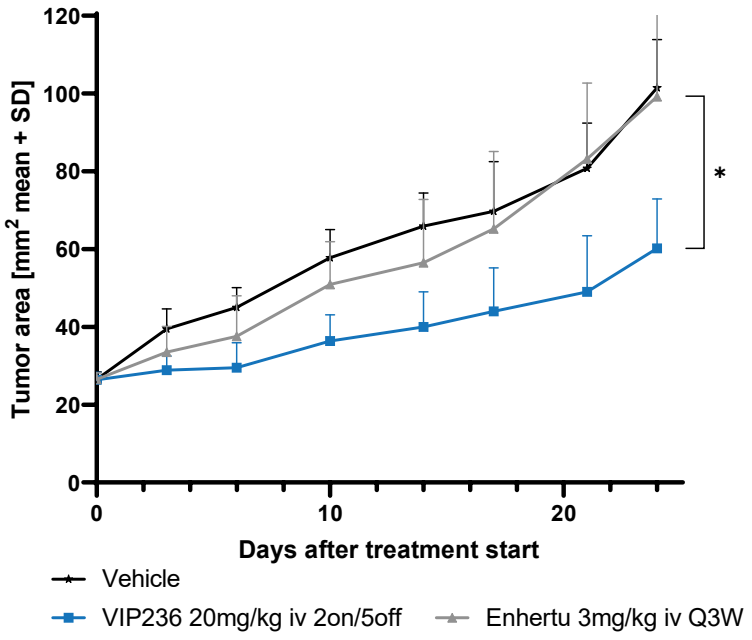


- Durable complete regression in a NSCLC PDX model with all schedules and doses tested
- Partial regression in renal PDX model with durable anti-tumor activity in the 3on/4off and 2on/5off schedules compared to once weekly treatment
- Statistically significant tumor growth inhibition in a liver metastasis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

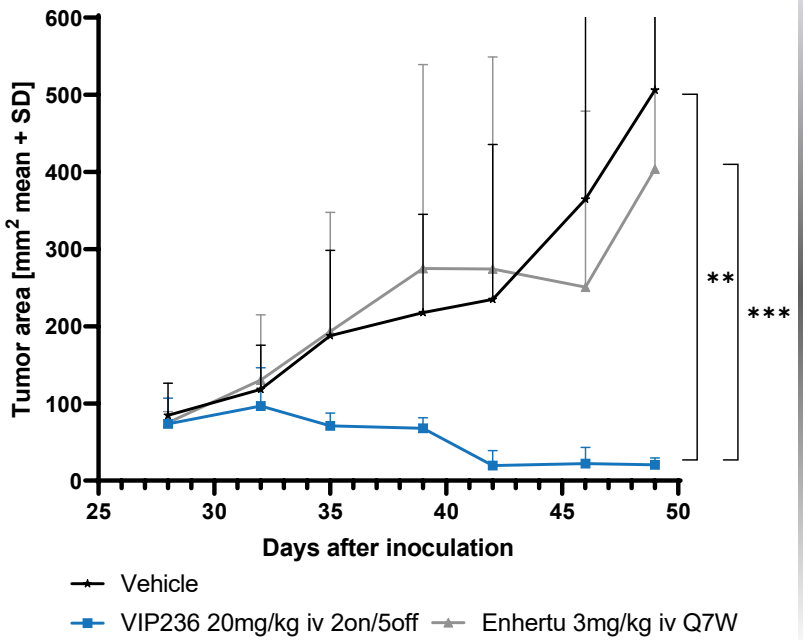
NSCLC, non-small cell lung cancer; CRC, colorectal cancer; iv, intravenous; PDX, patient derived xenograft; QW, once weekly dosing; RCC, renal cell carcinoma; SD, standard deviation; TNBC, triple negative breast cancer;
* $p < 0.05$ compared with vehicle , ** $p < 0.01$ compared with vehicle , *** $p < 0.001$ 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

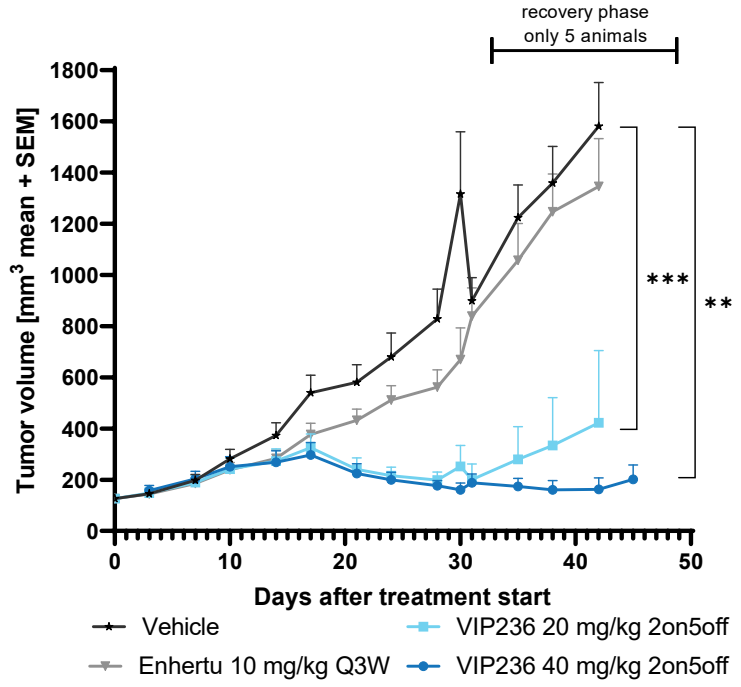
NCIN87 HER2^{high}



SNU16 HER2^{neg}



GXA3040 HER2^{low}



- 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^{neg} , whereas statistically significant tumor growth inhibition is seen in the NCI N87 HER2^{high} CDX model
  - In the GXA3040 HER2^{low} PDX model the 2on/5off schedule with high doses of 40mg/kg shows tumor regression and reduced re-growth

CDX, cell derived xenograft; iv, intravenous; PDX, patient derived xenograft; Q7W, every seven weeks; SD, standard deviation.

VIP236 First-in-Human Dose-Escalation Study

VNC-236-101

An open-label, multicenter Phase 1 study to characterize safety, tolerability, preliminary antitumor activity, pharmacokinetics, and pharmacodynamics of VIP236 monotherapy in subjects with advanced cancer [NCT05712889]

Enitociclib

P-TEFb PROGRAM

P-TEFb, positive transcription elongation factor B.

Enitociclib Overview

Best-in-class highly selective CDK9 inhibitor; partner of choice for novel combinations

Clinical Path

- Diffuse large B-cell lymphoma (DLBCL)
- Peripheral T-cell lymphoma (PTCL)
- MYC-driven solid tumors (eg, ovarian)

Current Trials

NIH sponsored trial in R/R lymphoid malignancies

Market Opportunity

- DLBCL 18K (US)/ 30K (EU)
- PTCL 12K (US)/ 6K (EU)
- Ovarian 26K (US)/ 40K (EU)

Near Term Milestones

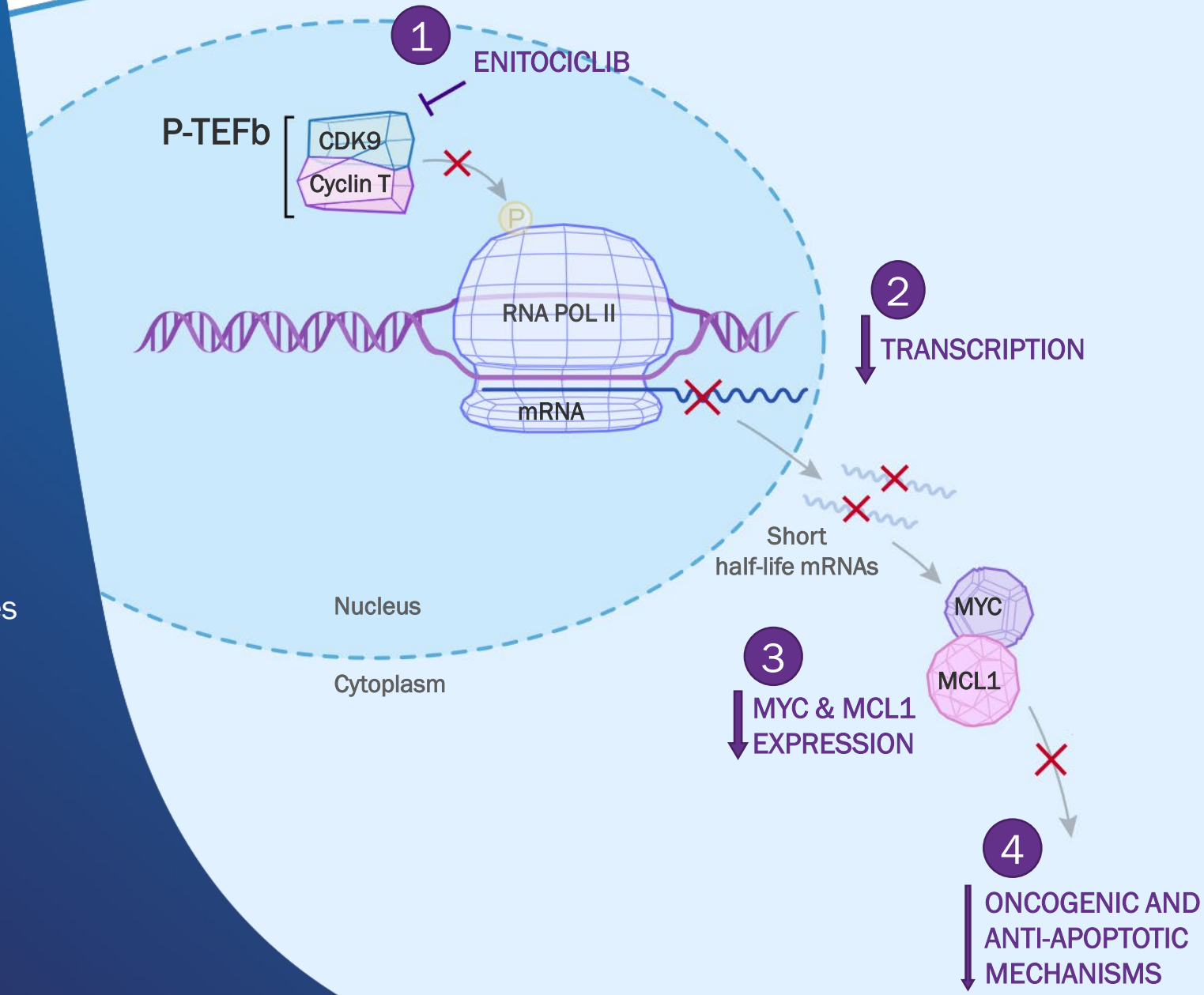
Additional studies pending funding/partnership

Market opportunity in patients / year. Data based on CI5XII, RKI and SEER*

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”



CDK, cyclin-dependent kinase.

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

- 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.

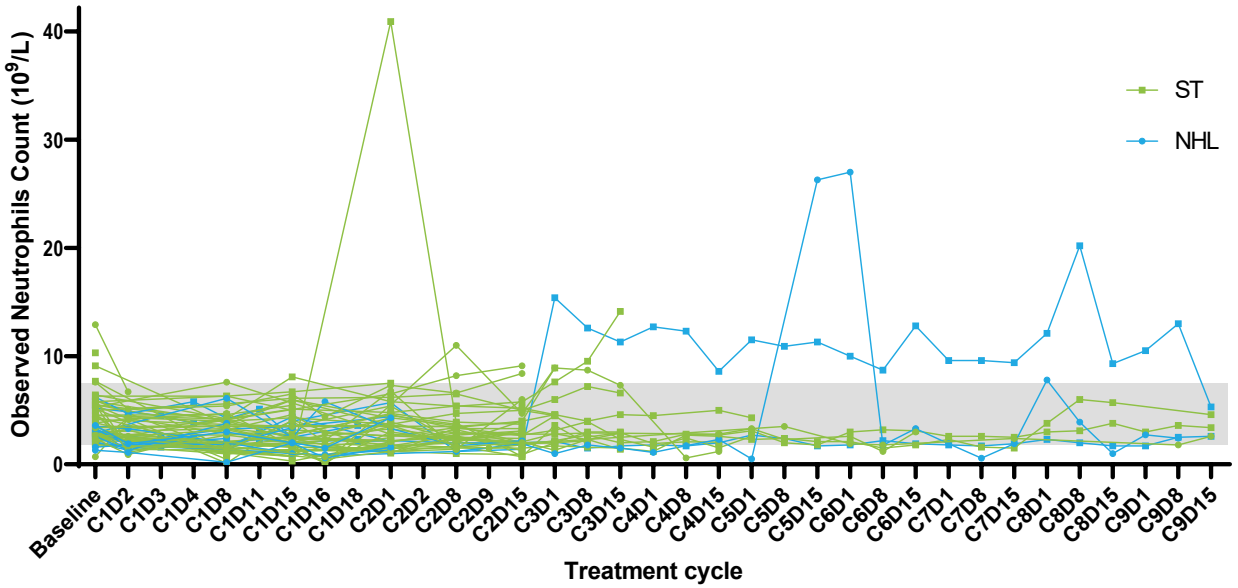
Lücking et al. J. Med. Chem. 2021, Boffo et al. J Exp Clin Cancer Res. 2018, Frigault, et al. EHA 2022.; Diamond, et al. CCR 2022.

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

Treatment-Emergent Adverse Events (n=63)

Adverse Events (>15%)	Any Gr n (%)	Gr 1 n (%)	Gr 2 n (%)	Gr 3 n (%)	Gr 4 n (%)	Gr 5 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

CDK, cyclin-dependent kinase; Gr, grade; PK, pharmacokinetics ; NHL, non-Hodgkin lymphoma; ST, solid tumor; Frigault, et al. EHA 2022.

Vincerx PHARMA

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

Monotherapy Activity

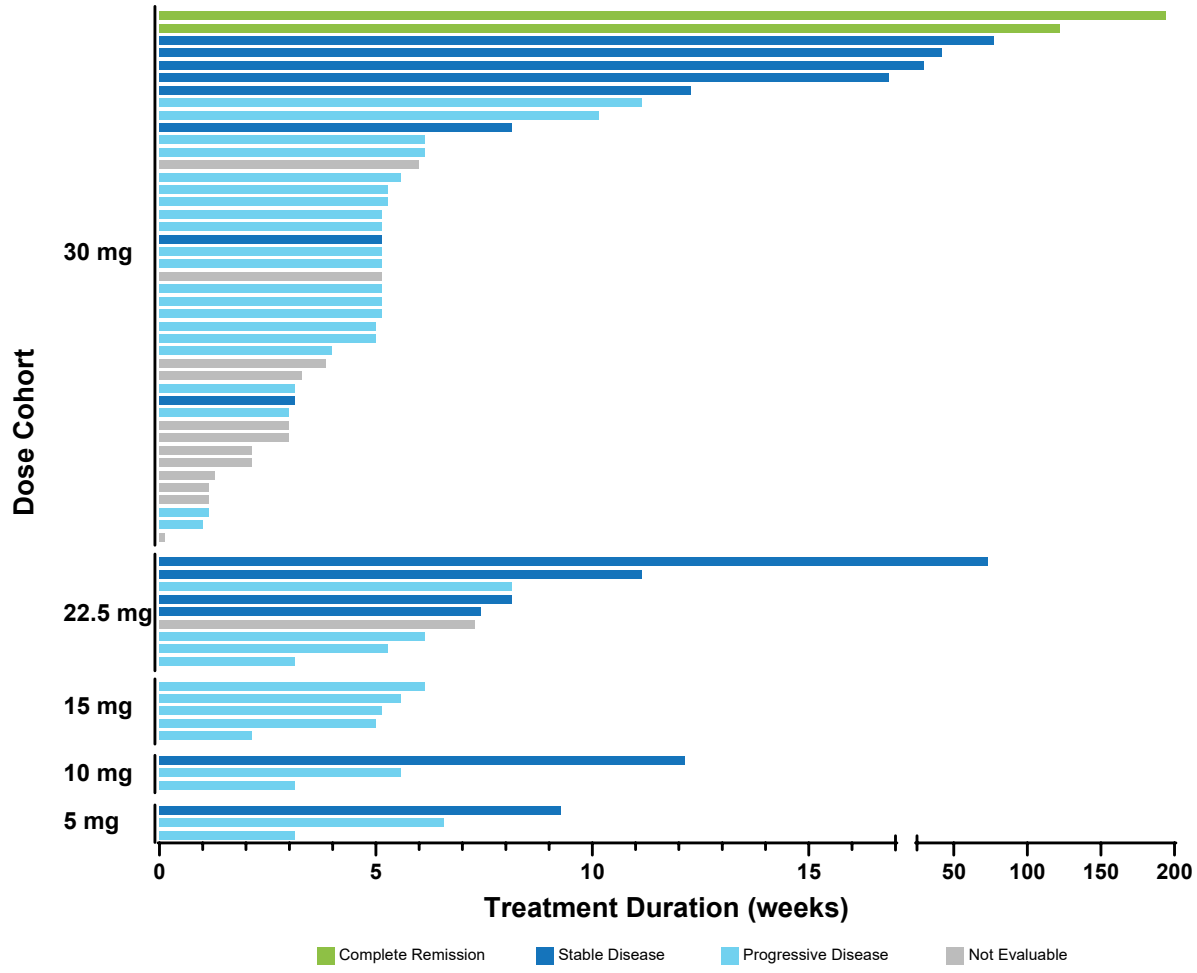
2 CRs of 7 DH-DLBCL (29% CR rate)

- 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, 27 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



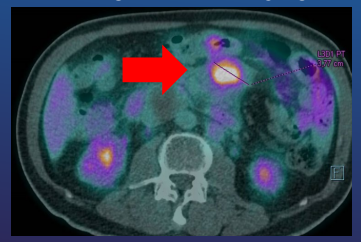
CR, complete remission; DH-DLBCL, double-hit diffuse large B-cell lymphoma.
Data cut off 21-Dec-23

Enitociclib Induces Durable Complete Metabolic Remission and Tumor Regression

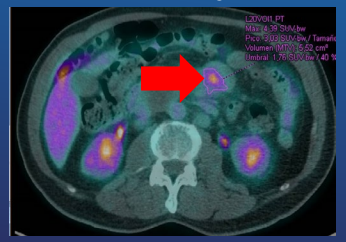
PATIENT 1: Diagnosis DH-DLBCL
With MYC and BCL2 Translocations

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

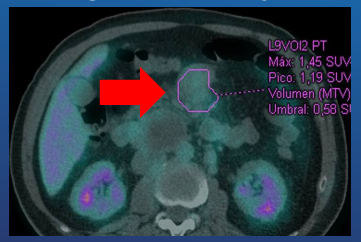
NOVEMBER 2016



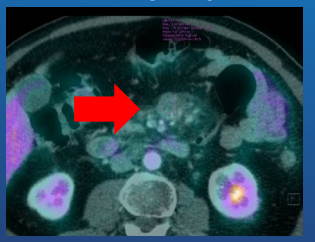
APRIL 2017



SEPTEMBER 2017



MARCH 2021



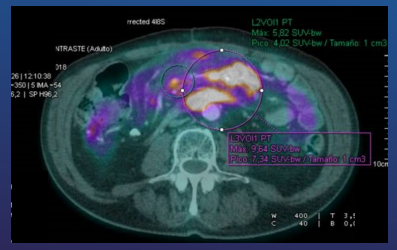
Date of 1st dose: 08 NOV 2016

Date of last dose: 16 JUL 2020

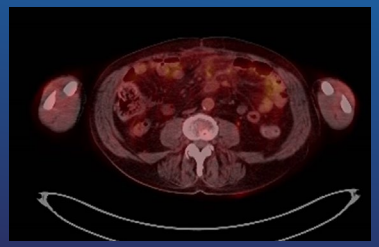
PATIENT 2: Diagnosis DH-DLBCL
With MYC and BCL2 Translocations

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

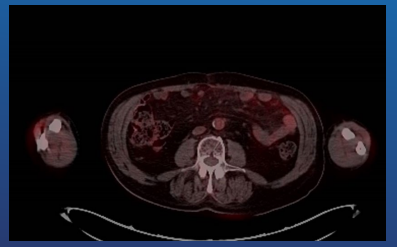
MARCH 2018



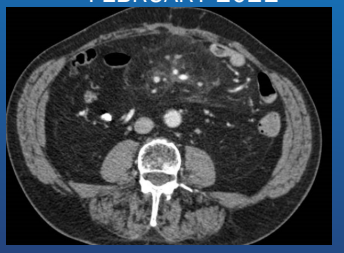
OCTOBER 2018



JUNE 2020



FEBRUARY 2021



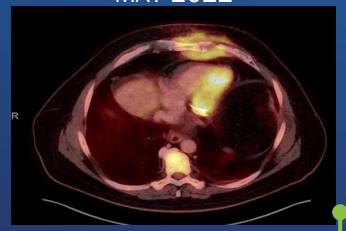
Date of 1st dose: 03 APR 2018

Date of last dose: 23 JUL 2020

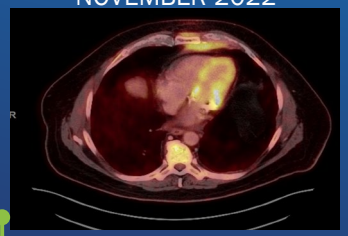
PATIENT: Diagnosis tFL
Age: 72 years
Cell of origin: GCB
Prior therapy (response):
- R-EPOCH (CR for 5 years)

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

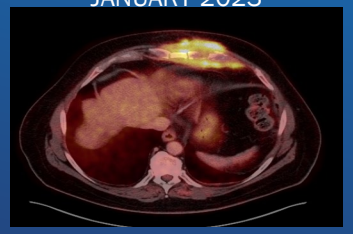
MAY 2022



NOVEMBER 2022

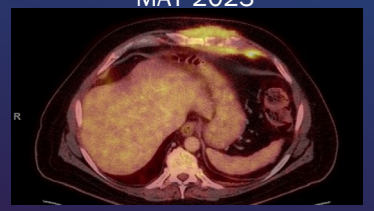


JANUARY 2023

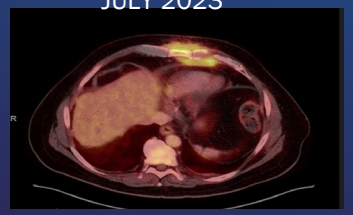


Date of 1st dose: 08 JUNE 2022

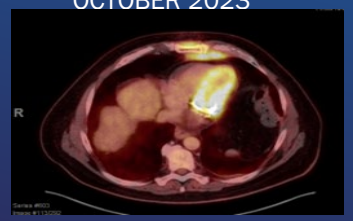
MAY 2023



JULY 2023



OCTOBER 2023



Cycle 23: 51% reduction in 2 TL

NIH Sponsored Trial in R/R Lymphoid Malignancies

VNC-152-801 (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

DLBCL, diffuse large B-cell lymphoma; GCB, germinal center B-cell; MTD, maximum tolerated dose; RP2D, recommended phase 2 dose; R/R relapsed/refractory; VVIP, venetoclax + VIP152, .

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

ENROLLMENT CONTINUES

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

AITL, angioimmunoblastic T-cell lymphoma; EBV+PTCL, Epstein-Barr virus-positive peripheral T-cell lymphoma; HGBCL-DH-BCL2, High-grade B-cell lymphoma with MYC and BCL2 rearrangements; R/R, relapse and refractory.

Vincerx PHARMA

Our Pipeline

PROGRAM	DISCOVERY	PRECLINICAL	PHASE 1	MOST RECENT MILESTONE ACHIEVED	NEXT EXPECTED MILESTONE
VersAptx™ Platform					
VIP236 $\alpha_v\beta_3$ - optCPT SMDC *First in Class*	Multiple Solid Tumors			Dosing Initiated in 1Q23	Preliminary Data Early 2024
VIP943 Anti-CD123 - KSPi ADC *Best in Class*	Leukemias & MDS			First Patient Dosed in September 2023	Preliminary Data Mid-2024
VIP924 Anti-CXCR5 - KSPi ADC *First in Class*	B-cell Malignancies				IND 2025
Anti-CD33 - KSPi ADC	Leukemias & MDS				Partnering Opportunity
Undisclosed Target 1 - KSPi ADC	Solid Tumors				Partnering Opportunity
Undisclosed Target 2 - KSPi ADC	Solid Tumors				Partnering Opportunity
P-TEFb					
ENITOCICLIB* CDK9 inhibitor (IV) *Best in Class*	MYC-rearranged DLBCL, Non-GCB DLBCL, Peripheral T-cell Lymphoma *(in partnership with NIH)*				Partnering Opportunity

*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.

Vincerx PHARMA

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

Vincerx PHARMA