CADRENAL THERAPEUTICS

Bridging Important Gaps in Anticoagulation Management for High-Risk Patients

December 2025

Corporate Presentation
NASDAQ: CVKD

Forward-looking Statements

This document contains forward-looking statements. In addition, from time to time, we or our representatives may make forward-looking statements orally or in writing. We base these forward-looking statements on our expectations and projections about future events, which we derive from the information currently available to us. Such forward-looking statements relate to future events or our future performance, including: our financial performance and projections; our growth in revenue and earnings; and our business prospects and opportunities. You can identify forward-looking statements by those that are not historical in nature, particularly those that use terminology such as "may," "should," "expects," "anticipates," "contemplates," "estimates," "believes," "plans," "projected," "predicts," "potential," or "hopes" or the negative of these or similar terms.

In evaluating these forward-looking statements, you should consider various factors, including: our ability to successfully develop and commercialize product candidates, our ability to raise capital when needed, and the competitive environment of our business. These and other factors may cause our actual results to differ materially from any forward-looking statement, including those risk factors disclosed in our Annual Report on Form 10-K for the year ended December 31, 2024, filed with the Securities and Exchange Commission on March 13, 2025, and our Quarterly Reports for the periods ended March 31, 2025, June 30, 2025, and September 30, 2025. Forward-looking statements are only predictions. The forward-looking events discussed in this document and other statements made from time to time by us or our representatives may not occur, and actual events and results may differ materially and are subject to risks, uncertainties, and assumptions about us. We are not obligated to publicly update or revise any forward-looking statement, whether as a result of uncertainties and assumptions, the forward-looking events discussed in this document, and other statements made from time to time by us or our representatives might not occur.

CADRENAL THERAPEUTICS

Our Mission

Develop novel, differentiated products that bridge critical gaps in current acute and chronic anticoagulation management for rare and high-risk patient populations.

Addressing gaps in the $40B anticoagulation market

VLX-1005 expands our portfolio with a novel immune-targeted approach - 12-LOX inhibition

Existing Programs

Tecarfarin

- An oral Vitamin K antagonist (VKA) with a proven mechanism of action (MoA) – same as warfarin
- Completely different – and desirable – metabolic pathway than warfarin
- ODD for end-stage kidney disease (ESKD) patients with atrial fibrillation (AFib)
- ODD for LVAD patients: collaboration with Abbott

Frunexian

- An acute parenteral Factor XIa (FXIa) inhibitor
- Only parenteral FXIa with fast-on / fast-off profile for acute care use
- For complex cardiac surgery (CABG) and continuous renal replacement therapy (CRRT) patients
- Acquired September 2025

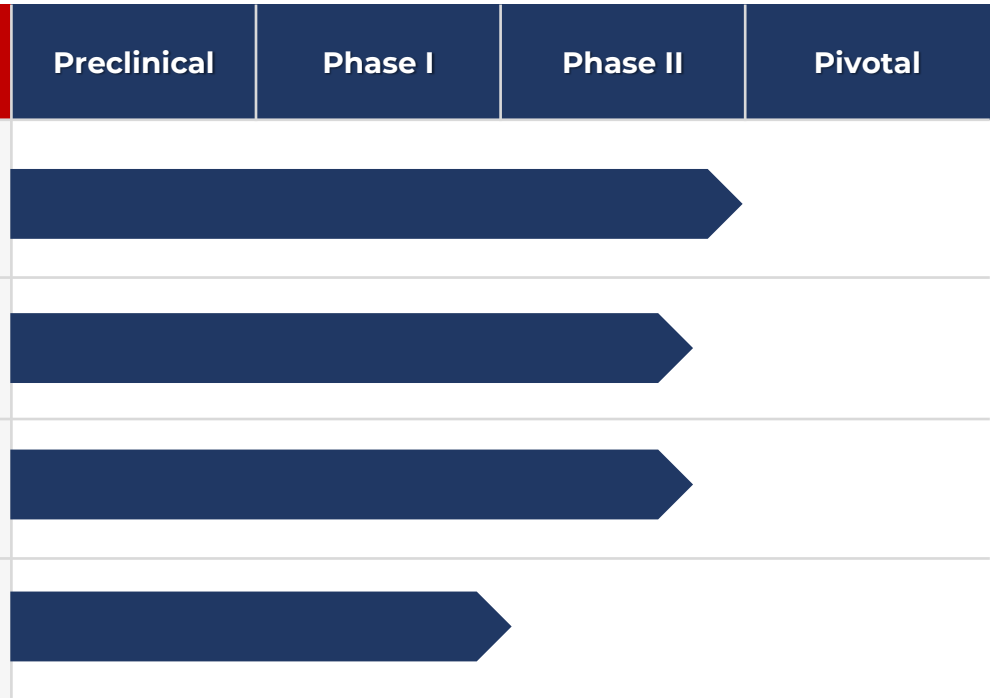
Recent Acquisition

VLX-1005

- A parenteral (intravenous) 12-Lipoxygenase (12-LOX) inhibitor designed to block key pathways in immune-mediated platelet activation
- Blocks platelet activation and inhibits thrombus formation
- Orphan Drug Designation (ODD) for patients with heparin induced thrombocytopenia (HIT)
- Acquired December 2025

Our development pipeline is focused on rare and high-risk indications

Program	MOA	Indication	Form	Regulatory
VLX-1005	12-LOX Inhibitor	HIT	IV	• FDA Orphan Drug Designation • EMA Orphan Drug Designation • FDA Fast Track Designation
Tecarfarin	Vitamin K Antagonist	ESKD+AFib	Oral	• FDA Orphan Drug Designation • FDA Fast Track Designation
		LVAD	Oral	• FDA Orphan Drug Designation
Frunexian	Factor XIa Inhibitor	CABG	IV	• Phase 2 ready



The indications targeted in our pipeline represent a $3 billion+ peak annual revenue potential

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Target:

Immune-mediated Platelet Activation

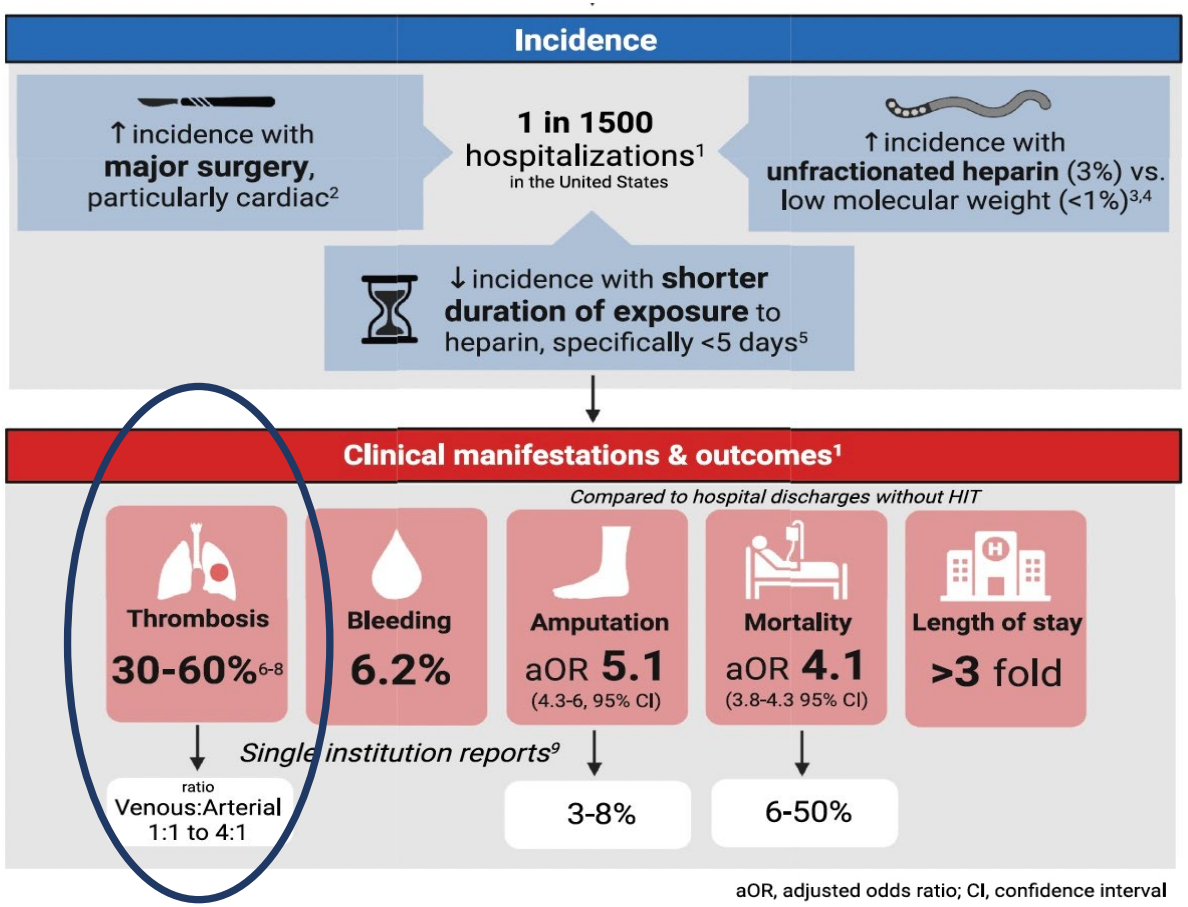
VLX-1005

Dire Outcomes of HIT Require a New Approach to Treatment

Epidemiology (US):

~300,000 suspected patients/year

~50,000 confirmed acute diagnoses/year



Adapted from May J, Westbrook B, Cuker A. Heparin-induced thrombocytopenia: An illustrated review. Res Pract Thromb Haemost. 2023 Jun 22;7(5):100283. doi: 10.1016/j.rpth.2023.100283. PMID: 37601013; PMCID: PMC10439402.

Heparin Induced Thrombocytopenia (HIT)

A Rare but Serious Immune Reaction to Heparin

CVD Interventions Use Heparin

- Cardiovascular disease (CVD) is on the rise across the world due to aging populations
- Complications from CVD also expected to increase

Heparin Usage Leads to HIT

- SoC for CVD interventions is heparin
- HIT results in platelet activation, causing thrombocytopenia and results in thrombosis

HIT has High Morbidity & Mortality

- HIT patients have dire outcomes including death, pulmonary embolism, stroke

Current Treatments

- SoC does not treat disease pathogenesis and can cause severe bleeding

Our Solution

VLX-1005 has a unique MoA that directly addresses the **pathogenesis** of HIT at its source

Current therapies only treat the thrombotic complications of HIT and convey additional bleeding risk

VLX-1005: The only clinical stage 12-LOX inhibitor

VLX-1005 is uniquely positioned to address an underserved indication with a unique mechanism of action (MoA) and expected meaningful impact on thrombotic events beyond that achievable with current anticoagulant therapy

Heparin-Induced Thrombocytopenia (HIT)	
Epidemiology	• ~150K – 300K patients
Est. Peak Sales[1]	• $800M+
Competitive Outlook	• Limited set of commercial non-heparin anticoagulants, led by argatroban • No other products in clinical development
Key Opportunity	• Potential to demonstrate clear value in an orphan indication with high unmet need • Monotherapy and combination possibilities to integrate with current treatment paradigm

12-LOX Inhibition	
Validation	• Validated with human ex vivo and animal in vivo data supporting role in platelet activity
Competitive Outlook	• No other 12-LOX inhibitors have successfully progressed to in-human clinical trials
Key Opportunity	• First-in-class (FIC) molecule with Phase 2 PoC secondary endpoint data validating impact on key thrombotic event rate • ODD and Fast Track Designation from the FDA

VLX-1005 is a first-in-class asset with a unique MoA progressing in an indication ripe for a new therapy

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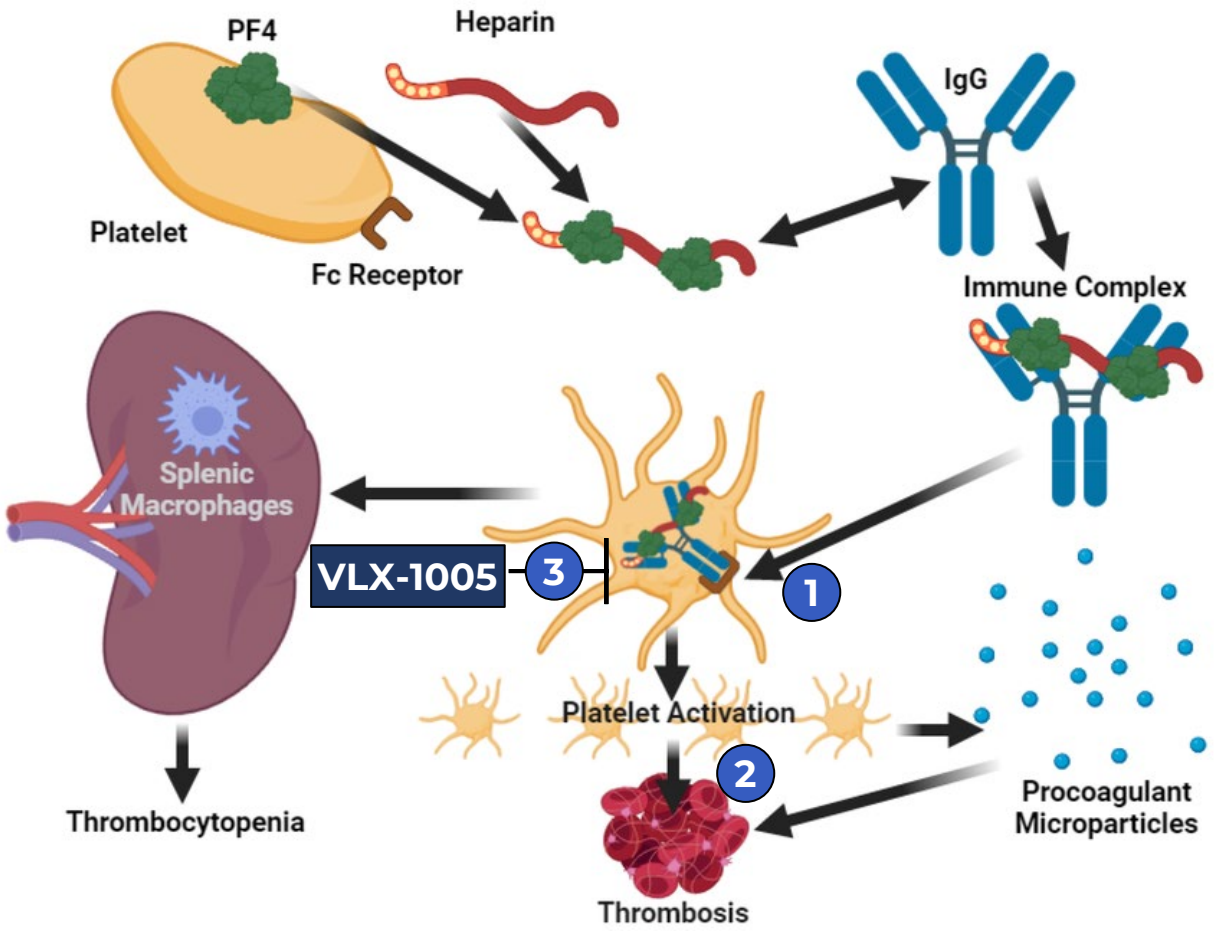
[1]LEK Consulting Analysis, 2022.

VLX-1005 – A Unique Mechanism of Action

12-LOX inhibition treats both platelet activation and thrombus formation in HIT

- HIT is a rare, potentially life-threatening reaction to heparin driven by an immune response to heparin-PF4 complexes
- 12-LOX is a key immune signaling enzyme, essential for the activation of multiple steps in platelet activation after immune complex binding to the FcγRIIa receptor
- Platelet activation drives thrombus formation and the subsequent clinical manifestations of HIT - thrombocytopenia and thrombosis
- VLX-1005 inhibits 12-LOX and blocks associated downstream pathways to reduce the stimuli driving both thrombus formation and thrombocytopenia
- The current standard of care (anticoagulants) only attenuate thrombus formation, without any effect on the underlying cause of HIT - immune complex platelet activation



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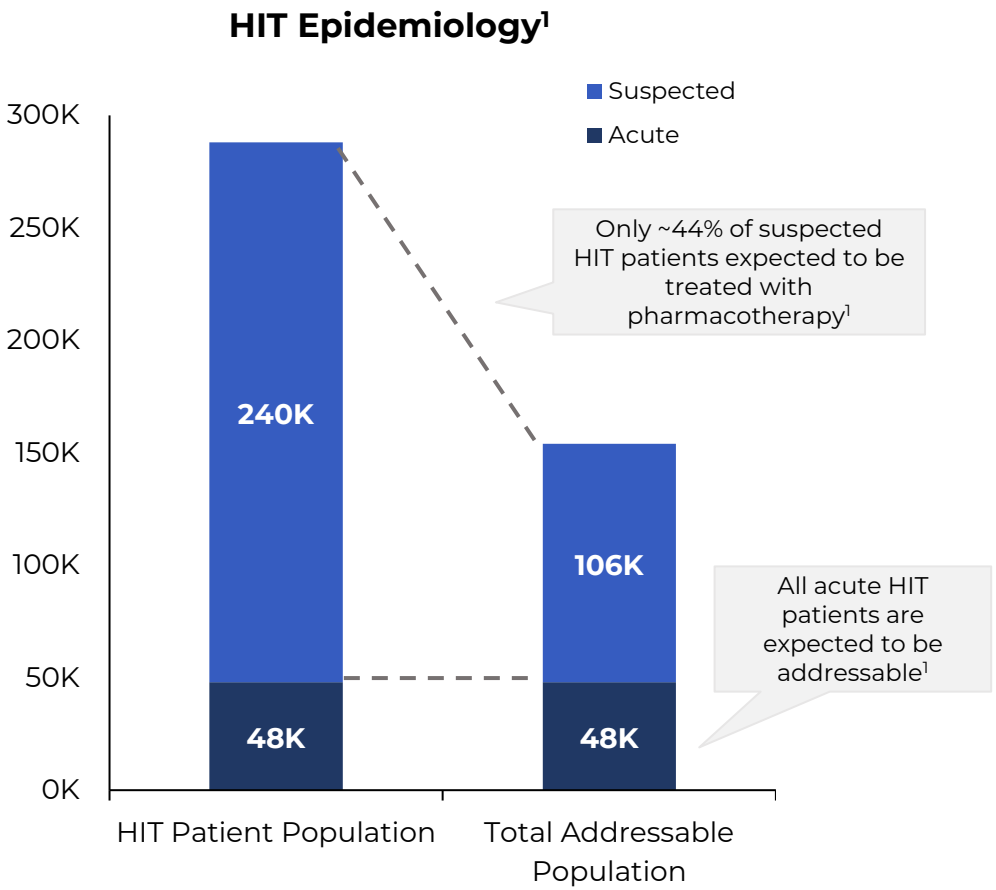
High Unmet Need in HIT

HIT patients face significant risk of severe complications and death; legacy anticoagulation pharmacotherapy is marginally effective in this population and pose further major bleeding risks



While heparin use is declining, it is still a key cornerstone of care, particularly for cardiovascular surgeries

Population dynamics (e.g., aging population, increasing prevalence of comorbidities) point to a growing risk of HIT

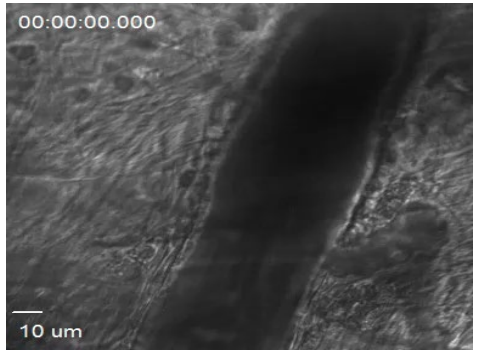
Patients with HIT still have high unmet need, with critical complications of thrombosis, limb gangrene, and death

Current anticoagulation treatment for HIT causes or exacerbates bleeding, and can even result in fatal hemorrhage

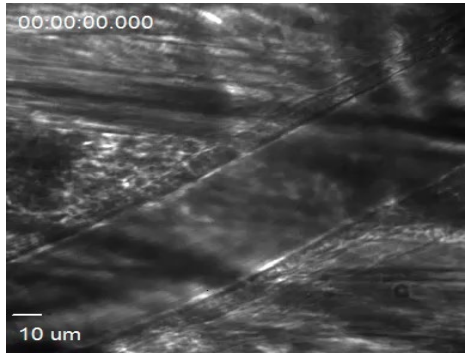
Notes: LEK Analysis, 2022.
Sources: Chong. Blood. 2025; Company Materials.

VLX-1005 Inhibits Thrombus Formation without Altering Normal Hemostasis – Unlike Conventional Anticoagulants

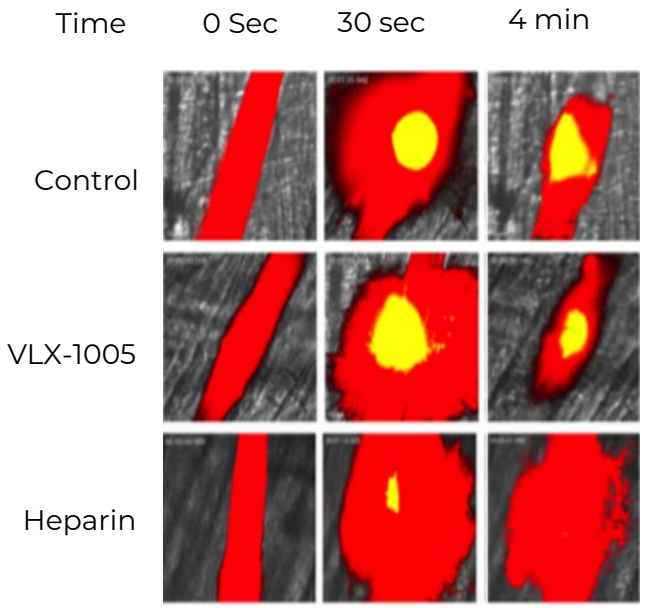
Vehicle Control



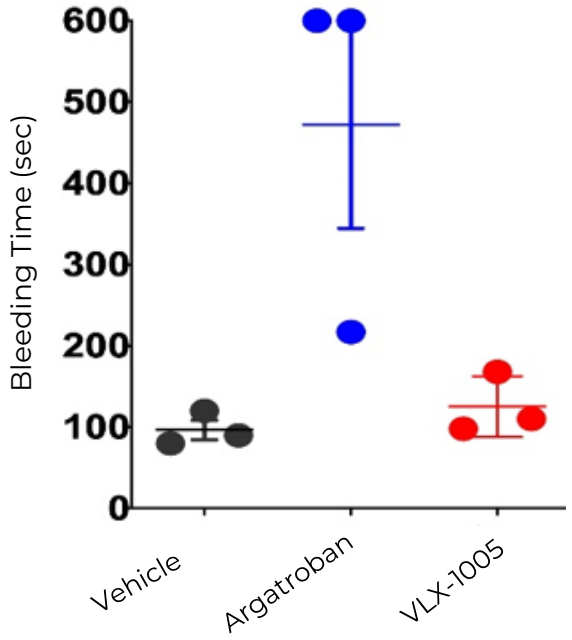
VLX-1005 (7.5 mg/kg, PO)[2]



VLX-1005 shows dose dependent prevention of platelet aggregation in a murine thrombosis model [1]



VLX-1005 does <u>not</u> inhibit hemostatic plug formation



VLX-1005 does <u>not</u> increase bleeding times, in contrast to argatroban

Inhibition of platelet aggregation <u>without</u> increasing bleeding is an important leap forward in safety over existing therapies

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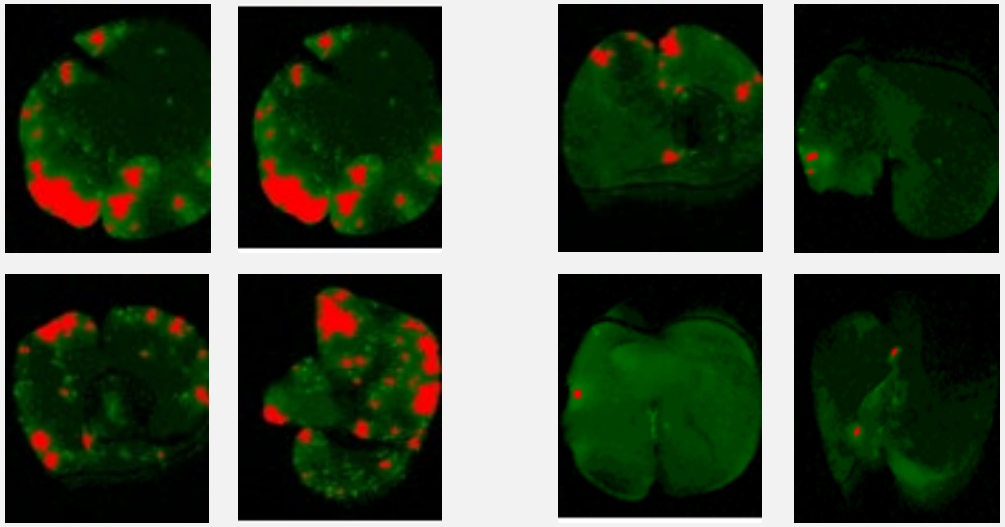
[1] *Arterioscl Throm Vasc Biol* **2017**;37:1828
All images are mouse data
[2]VLX-1005 was dosed 15h prior to imaging

VLX-1005 Effective in an Animal Model of HIT

VLX-1005



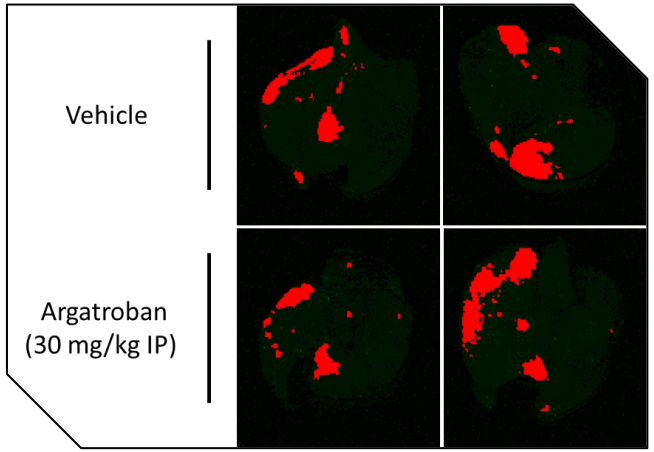
Vehicle **VLX-1005**

Pulmonary Embolism

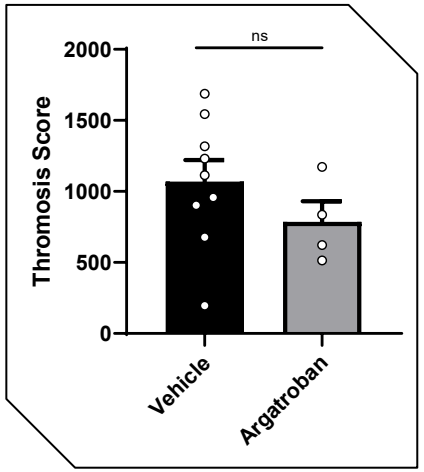
- Lung images from mice
- VLX-1005 treatment significantly inhibits PE

Standard of Care (argatroban) has no impact on:

- Prevention of pulmonary embolism
- Thrombosis



Pulmonary Embolism



Thrombosis
(30 mg/kg IP)

Renna SA *et al. Arterioscler Thromb Vasc Biol.* 2023;43:1808–1817
DOI: 10.1161/ATVBAHA.123.319434

Phase 1 Studies Demonstrate VLX-1005 Well-Tolerated

VLX-1005 well-tolerated in Phase 1 SAD/MAD and DDI studies

- Over 100 subjects dosed
- Adverse events were uncommon and mild in severity
- Orphan Drug Designation in US and EMA
- Fast Track Designation in US

Composite SAD/MAD results:

- No treatment-based discontinuations
- 69 treatment emergent adverse events were observed
  - 30% in VLX-1005 treated subjects vs. 36% in placebo
- **No SAEs reported, no dose limiting toxicities**
- PK well behaved & dose proportional
- **No drug-drug interaction between VLX-1005 & argatroban**

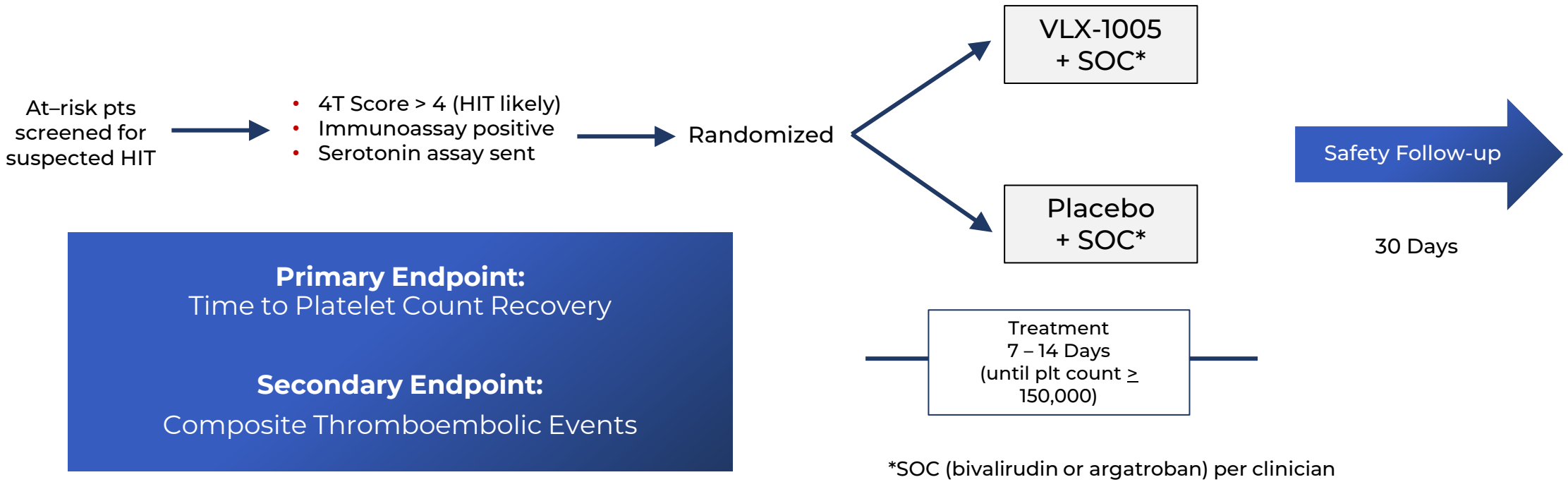
VLX-1005 Phase 2 PoC Study (ALATHEA)

Interim analysis complete. Database analysis 1Q26.

Primary Study Goals

Identify a **clinically meaningful endpoint** to demonstrate **efficacy over SoC**

Evaluate safety in the indicated population

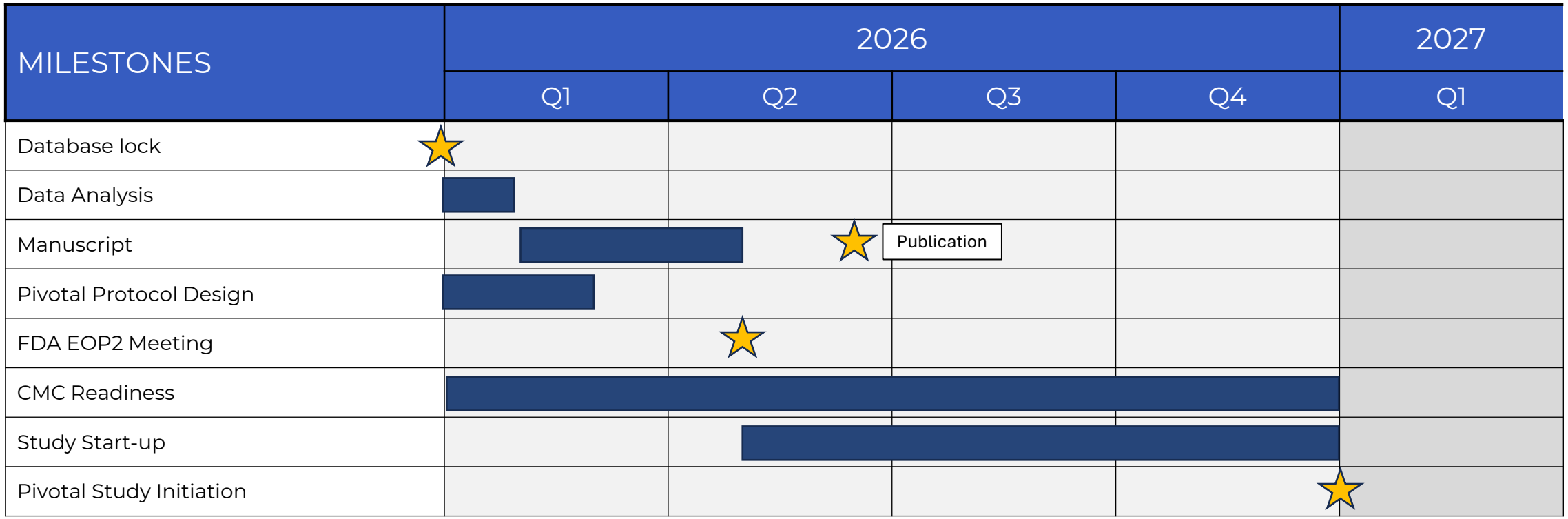


Primary Endpoint:
Time to Platelet Count Recovery

Secondary Endpoint:
Composite Thromboembolic Events

*SOC (bivalirudin or argatroban) per clinician

Near-Term Plan and Estimated Timeline for VLX-1005



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Target:

Coagulation

Tecarfarin and Frunexian

Tecarfarin Overview

A Novel VKA with Multiple Orphan Drug Designations by FDA

Unmet Need

- Poor warfarin control
  - Thrombosis
  - Bleeding
- DOAC failures
- ~30% of people can have genetic variants adversely impacting warfarin metabolism

Differentiation

- Only VKA in late-stage development worldwide
- Not metabolized by CYP450; not affected by renal impairment
- ODD and Fast Track Approval in ESKD patients with AFib
- ODD in LVAD patients

Path Forward

- Extensive safety data (n=1,000+)
- CMC ready
- Potential Phase 2 study in LVAD patients and/or ESKD+AFib patients

Opportunity

- Only new VKA in the last 70 years
- Targeting areas where DOACS are contra-indicated, unapproved, or not recommended

Tecarfarin
A novel VKA

- Addresses important limitations of warfarin
  - Predictable metabolism, fewer interactions
- Suitable for patients with complex medication regimens
- Positive safety and efficacy data
- Consistent anticoagulation control
- Levels not affected in patients with poor kidney function
- Fills critical gaps unaddressed by DOACs

Frunexian Overview

An Acute Parenteral Factor XIa inhibitor

Unmet Need

- Breakthrough Factor XIa asset(s)
- Bridging opportunity (IV to Oral)

Differentiation

- Fast-on / fast-off potent, dose-proportionate parenteral FXIa inhibition
- Only acute-care parenteral XIa in development

Path Forward

- Solidify CMC
- Phase 2 dosing in preparation for potential Phase 3 program(s)
  - Complex cardiac surgery
  - Continuous Renal Replacement Therapy

Opportunity

- Acute-care settings where there are opportunities to address treatment gaps with heparin

Frunexian

Acute parenteral FXIa inhibitor

- Potent (> 95%) XIa inhibition
- Effective blockade of contact activation
- Predictable PK/PD
- Parenteral administration
- Rapid onset, short half-life
- Phase 2 ready

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Leadership & Financial Summary

Experienced Leadership

Across clinical to commercial drug development

Quang X. Pham
CEO & Founder, Chairman

James Ferguson, MD, FACC, FAHA
Chief Medical Officer

Matthew Szot, CPA
Chief Financial Officer

Jeff Cole
Chief Operating Officer

John R. Murphy
Board Member

Steven Zelenkofske, DO
Board Member

Glynn Wilson, PhD
Board Member

Lee Golden, MD
Board Member

Financials, Capitalization & Insider Alignment

Cap Table	
Cash (at 9/30/2025)	$3.9 million
Debt	NONE
Common Shares Outstanding (at 11/20/2025)	2,075,845
Warrants – Investors (avg. $17.82)	615,940
Stock Options Outstanding (avg. $17.61)	426,333

2025 Financial Results – Nine Months Ended Sept. 30	
Operating Expenses (excluding non-cash items)	$8.8 million
Cash used in operating activities	$10.0 million

Market Capitalization	
As of 12/11/2025	$23 million

Insider Ownership (Common Stock)	
Insider Ownership as Percent of Shares Outstanding	22%

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