

CORPORATE PRESENTATION

June 29, 2022

Forward Looking Statements

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INTRODUCTION & PIPELINE

VTX958 | TYK2 Inhibitor | Phase 1

VTX002 | S1P1R Modulator | Phase 2

VTX2735 | Peripheral NLRP3 Inhibitor | Phase 1 Complete

VTX3232 | CNS-penetrant NLRP3 Inhibitor | Pre-clinical

Summary | Milestones & highlights

Our Leadership Team

MANAGEMENT



Raju Mohan, PhD CHIEF EXECUTIVE OFFICER, FOUNDER



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Chris Krueger, JD CHIEF BUSINESS OFFICER



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William White CHIEF FINANCIAL OFFICER, AKERO THERAPEUTICS Raju Mohan, PhD CHIEF EXECUTIVE OFFICER, VENTYX



Our Mission: To become a Leading Immunology Company

Underpinned by Strong Drug Discovery and Development Capabilities

WITH THREE, DIFFERENTIATED, CLINICAL-STAGE CANDIDATES

and a deep pipeline of preclinical programs that target immune-mediated and inflammatory disease indications

OUR INTERNALLY-DISCOVERED SMALL MOLECULE DRUGS

allow us to own 100% commercial rights to our entire portfolio with long patent lives for all product candidates

OUR EXPERIENCED TEAM AND OUR INTERNAL R&D ENGINE

continue to generate candidates with potential to address diseases with high unmet need



Broad Pipeline of Candidates With Multiple Near-Term Catalysts

Addressing Established Inflammatory and Immunology Markets with a Wholly Owned Product Portfolio

TARGET	PROGRAM	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	NEXT ANTICIPATED MILESTONES
ТҮК2	VTX958	Potential indications inc	lude psoriasis, psoriatic art	hritis, Crohn's disease and o	thers	Report topline Phase 1 data Q3 2022 Initiate Phase 2 POC trials H2 2022
S1P1R	VTX002	Ulcerative Colitis				Report topline Phase 2 data 2023
NLRP3 Peripheral	VTX2735	Potential indications inc	lude cardiovascular, hepat	ic, renal, and rheumatologic c	liseases	Initiate POC trial in CAPS Q4 2022
NLRP3 CNS-penetrant	VTX3232	Neuroinflammatory dise	ases			File IND Q4 2022 Initiate Phase 1 trial Q1 2023



Pipeline Targeting Large Well-Established Markets

Sources: Evaluate Pharma, Company Estimates, Wall Street Research

INDICATION*	PATIENTS IN THE U.S.	GLOBAL DRUG REVENUE* (2020)	TARGET POPULATION
Psoriasis Dermatology	~8M	~ \$20B	25-30% MODERATE-TO-SEVERE
Crohn's disease <i>IBD</i>	~700K	~ \$13B	30-40% MODERATE-TO-SEVERE
Ulcerative colitis IBD	~1M	~ \$7B	30-40% MODERATE-TO-SEVERE
Psoriatic arthritis Rheumatology	~1M	~ \$4B	40-60% MODERATE-TO-SEVERE
<mark>SLE</mark> Rheumatology	Up to 500K	~ \$1B	





ORALLY BIOAVAILABLE selective allosteric inhibitor of TYK2



VTX958 Program Summary

Allosteric, Selective TYK2 Inhibitor

Potentially Differentiated TYK2 Inhibitor

- Selective, allosteric TYK2 inhibitor
- TYK2 functional selectivity can potentially differentiate clinical profile vs. less selective TYK2 inhibitors

Clinically Validated Target

- Well established clinical efficacy in psoriasis, IBD and psoriatic arthritis with biologics targeting IL-12/IL-23 and IL-23* pathways
- These pathways also the target of allosteric TYK2 inhibitors
- Phase 3 PoC in psoriasis has been demonstrated** by BMS' allosteric TYK2 inhibitor deucravacitinib

Deucravacitinib in Phase 2/3 for Crohn's disease, psoriatic arthritis, lupus

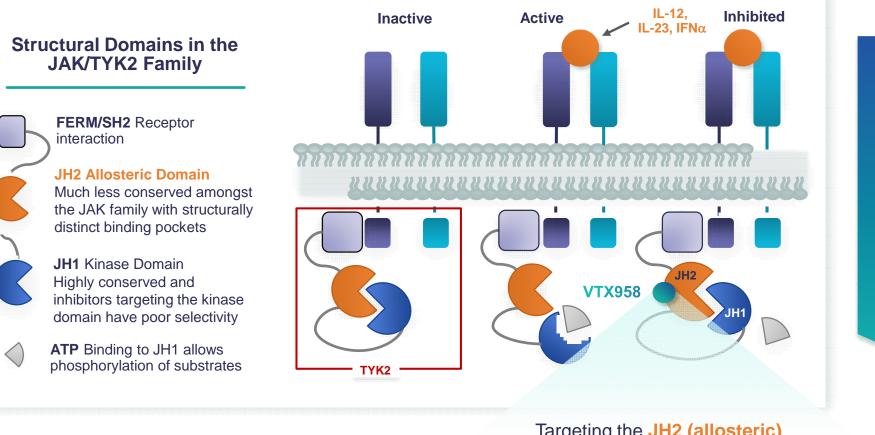
Large Addressable Markets

 Multiple autoimmune disorders in dermatology, IBD, renal and rheumatology total \$45B WW sales*



*Includes approved drugs Stelara[™] (JNJ), Tremfya[®] (JNJ), Skyrizi[™] (ABBV), Ilumya[™] (Sun Pharma) and others in late-stage development (mirikizumab (LLY), brazikumab (AZN) **Deucravacitinib efficacy reported on 16-week primary endpoint of PASI-75 (75% reduction of psoriasis affected area and severity) at AAD '21; p<0.0001 vs placebo and Otezla[®] in POETYK-1; p=0.0003 vs. Otezla in POETYK-2; See slide 14 for more detail on \$45B worldwide market

Allosteric Inhibitor VTX958 Binds Selectively to the TYK2 JH2 Domain



Features of VTX958 JH2 Allosteric Inhibition

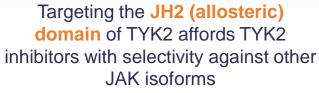
Selectivity for TYK2 JH2 vs. JAK1 JH2 domain (>4,000 X)

No binding to JAK2/3 JH2 domains

No binding to TYK2 kinase JH1 and

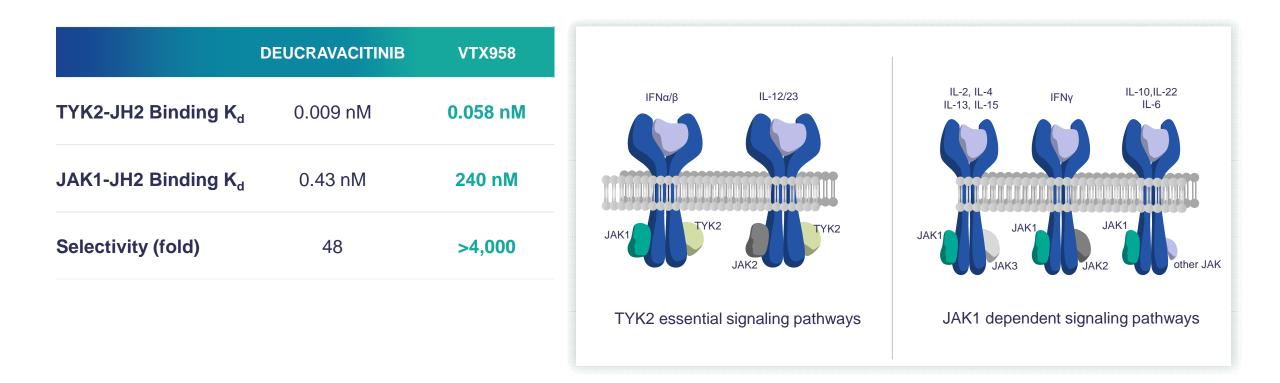
No kinase enzyme inhibition of any JAK family member

Selective TYK2 Inhibitor



VTX958 More Selective than Deucravacitinib for TYK2 JH2 Domain

Inhibits TYK2 Pathways (IL-12, IL-23, IFNα) while Avoiding the JAK1/2/3 Pathways





VTX958 Selectively Targets IL-12, IL-23 and IFN α

VTX958 Potently Inhibits TYK2 Pathways

Selective and potent inhibition of IL-12/23 and Type I interferon axis allows targeting pathways driving immune-mediated diseases

VTX958 Has No Measurable Inhibition of JAK1-Mediated Pathways

Lack of inhibition of IL-6, IL-10 and other protective cytokines may avoid potential AEs associated with less selective inhibitors

PROINFLAMMATORY INNATE & TH1/TH17 CYTOKINES				PLEIOTROPIC CYTOKINES WITH PROTECTIVE FUNCTIONS					
	Р	soriasis Patient PBM	IC						
DRUG	IL-12 IC ₅₀ (nM)	IL-23 IC ₅₀ (nM)	IFNα IC ₅₀ (nM)	DRUG	IL-22 IC ₅₀ (nM)	IL-10 IC ₅₀ (nM)	IFNγ IC ₅₀ (nM)	IL-4 IC ₅₀ (nM)	IL-6 IC ₅₀ (nM)
VTX958	35	5	12	VTX958	>10,000	>10,000	>10,000	>10,000	>10,000
deucravacitinib	10	10	5	deucravacitinib	114	20	350	249	464

KEY TAKEAWAYS

- Potent activity against IL-23, a key cytokine implicated in psoriasis and other indications
- Broad therapeutic window with VTX958 may allow for higher exposures in Phase 2/Phase 3 studies



VTX958 Phase 1 SAD Results Support Clinical Advancement

Safety

Well-tolerated across all cohorts; all AEs observed were mild and not dose- or time-of-dose dependent

Pharmacokinetics

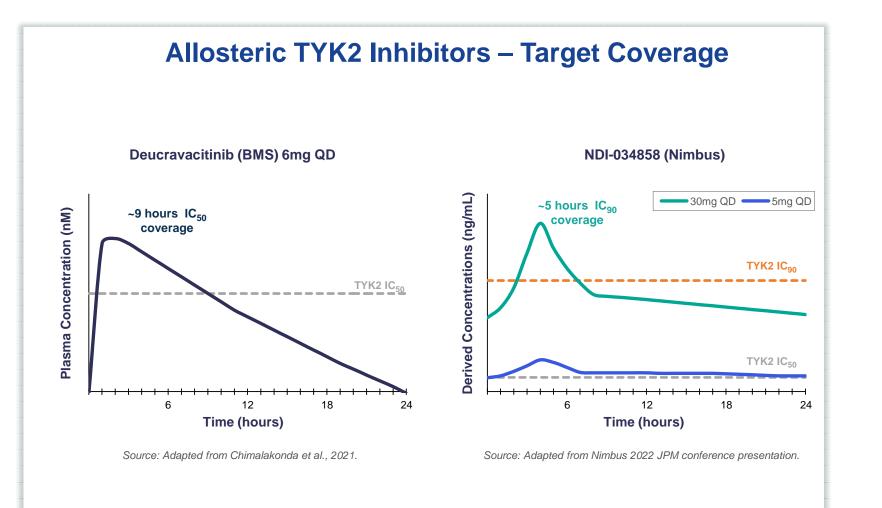
No dose-saturation observed; PK and absorption profiles suggest continued absorption throughout GI tract

Pharmacodynamics

Dose-dependent VTX958-mediated effect on TYK2 signaling observed in both *in vivo* gene expression studies and *ex vivo* stimulation assays



Targeting a Best-in-Class Exposure Profile With VTX958

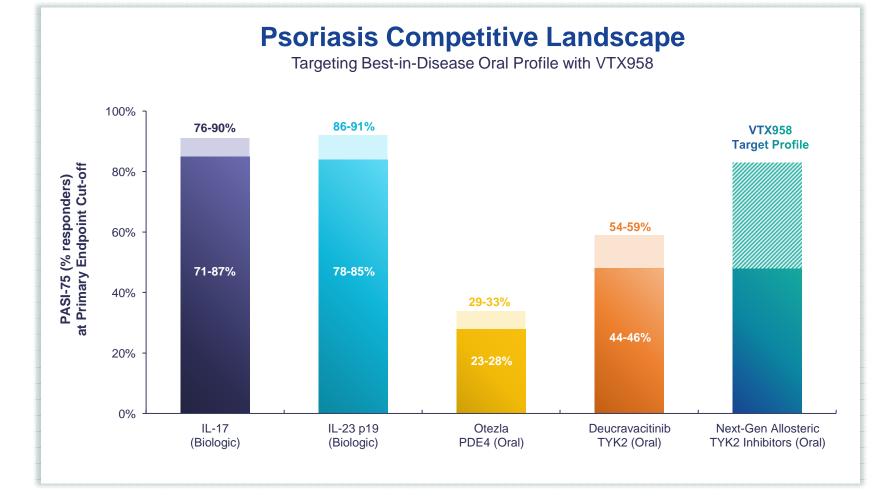


VTX958 Target Profile

- Maximize TYK2 pathway suppression (IC₅₀ and IC₉₀)
- Wide safety margin enabling higher doses and exposures:
 - Potential for improved efficacy in PsO + PsA with greater TYK2 inhibition
 - Higher exposures may be necessary to achieve efficacy in Crohn's disease



VTX958 Profile Expected to Drive Clinical Differentiation



KEY TAKEAWAYS

- Current oral options in PsO are substantially less efficacious than biologics
- Greater TYK2 suppression may produce improved efficacy compared to other oral agents, with potential to approach leading biologics
- Significant opportunity for a best-in-disease oral agent in psoriasis, a >\$20B global market



Note: Solid area represents pbo-adjusted response rate; dashed area indicates total observed response rate; primary endpoint cut-off ranges from Week 10 to Week 16 Sources: Company reports and FDA labels for approved anti-IL-17 and anti-IL-23 biologics

Unlocking the Opportunity in Crohn's Disease

Several-fold Higher Doses Required in Crohn's*

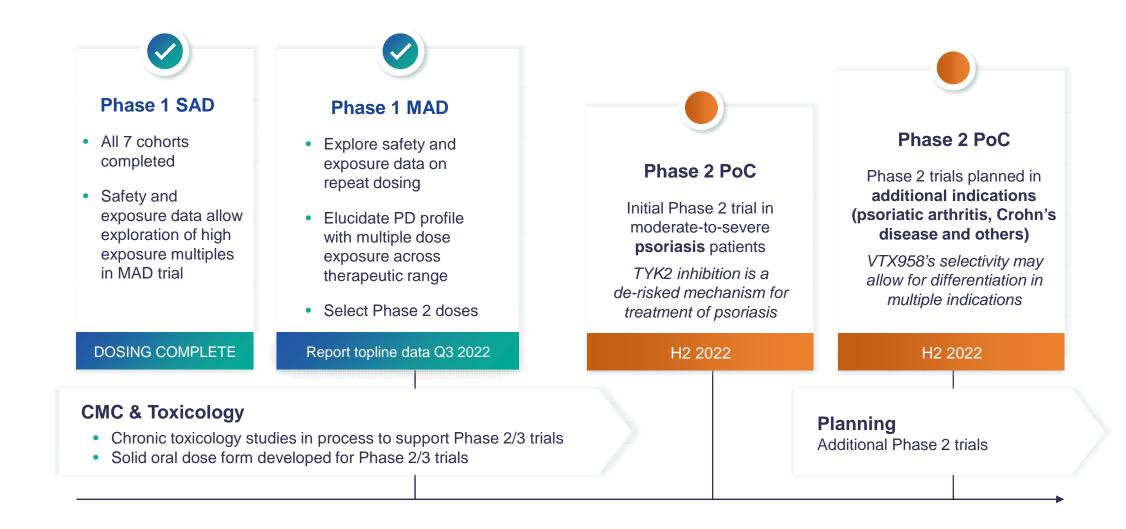
PsO Dose	Crohn's Dose
150mg Q12W	600mg IV Q4W (induction)
Subcutaneous	360mg SC (maintenance)
100mg Q8W	200mg / 600mg / 1200mg**
Subcutaneous	IV Q4W induction
40mg / 90mg Q12W	260mg / 390mg / 520mg
Subcutaneous	IV induction dose
80mg (SC induction)	160mg (SC induction)
40mg Q2W maintenance	40mg Q2W maintenance
	150mg Q12W Subcutaneous 100mg Q8W Subcutaneous 40mg / 90mg Q12W Subcutaneous 80mg (SC induction)

Greater Exposures Needed for TYK2 Inhibitor Efficacy in Crohn's

- Biologics data suggest substantially higher exposures are required for efficacy in Crohn's vs. PsO
- Maximizing TYK2 target coverage expected to differentiate VTX958 from other TYK2 inhibitors
- Selectivity, safety and tolerability considerations may limit the Crohn's opportunity for other TYK2 inhibitors
- Optimized profile of VTX958 may unlock a major market opportunity in Crohn's, a >\$13B global market



VTX958 Clinical Development Plan







PERIPHERALLY RESTRICTED S1P1R MODULATOR with potential for treatment of ulcerative colitis

VTX002 Program Summary

Phase 2 S1P1R Modulator for Ulcerative Colitis

Potentially Differentiated S1P1R Modulator

- Selective S1P1R modulator
- Differentiated on key parameters
- Demonstrated pharmacodynamic activity in Phase 1 trial
- Pursuing clinical development plan in both treatment-naïve and biologic-experienced patients

Clinically Validated Target

- S1P1R modulators approved for MS and UC with clinical trials ongoing in other indications
- BMS' ozanimod approved for UC in May 2021

Large Addressable Markets

 Ulcerative colitis is lead indication totaling up to \$7B in worldwide revenue



VTX002 Differentiates on Multiple Key Parameters vs. Competitors

Potential for Differentiated Clinical Profile in UC Patients	Safety Profile	No Drug-Drug Interactions
Sustained lymphocyte reduction up to 65% across multiple doses in MAD trial	No SAEs, elevated LFTs, abnormal PFTs or macular edema	No CYP inhibition; no food effect; favorable profile for patients with co-morbidities
Fast Onset of Action Faster Lymphocyte Recovery	Ability to Dose Titrate	Peripherally Restricted



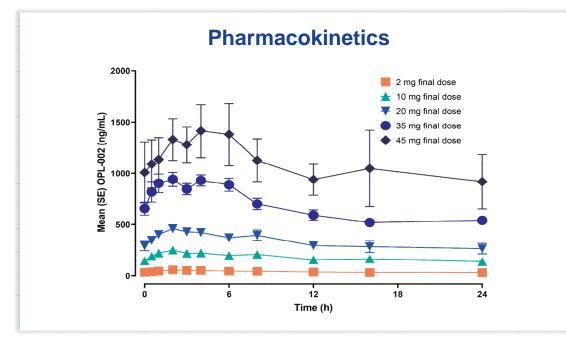
VTX002 Differentiates on Multiple Key Parameters vs. Competitors

Differentiating Parameter	Ozanimod	Etrasimod	VTX002
Receptor Selectivity	S1P 1,5	S1P 1,4,5	S1P 1,5
Lymphocyte Suppression in Healthy Volunteers	1 mg, ~60%	2 mg, 69%	40 mg, ~65%
Lymphocyte Suppression in UC Patients	1 mg, 49%	2 mg, 40%	TBD
CYP450 Interactions	Yes	No	No
Liver Enzyme Elevations	Yes	No	No
Active Metabolites	Yes	No	No
Half-life	19 h, Met 10-13 d	33 h	~20 h
Fast Lymphocyte Recovery Time	No	Yes	Yes
First Dose Heart Rate Reduction	Yes	Yes	Yes
Dose Titration	Yes	Νο	Yes
First Dose Monitoring	No	TBD	TBD

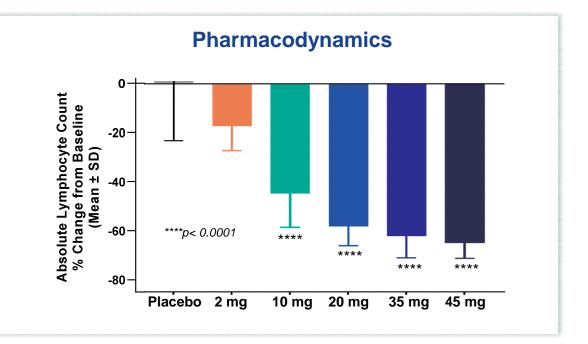


Phase 1 MAD Results: Dose-Dependent Exposure and Lymphocyte Reduction

Absolute Lymphocyte Count (ALC) Reductions of 40-50% Correlated with Clinical Efficacy Observed in UC*



- T_{1/2} of ~20 hours
- Dose-proportionate exposure after single and multiple doses of VTX002 with steady-state reached after 4 to 7 days of targetdose exposure



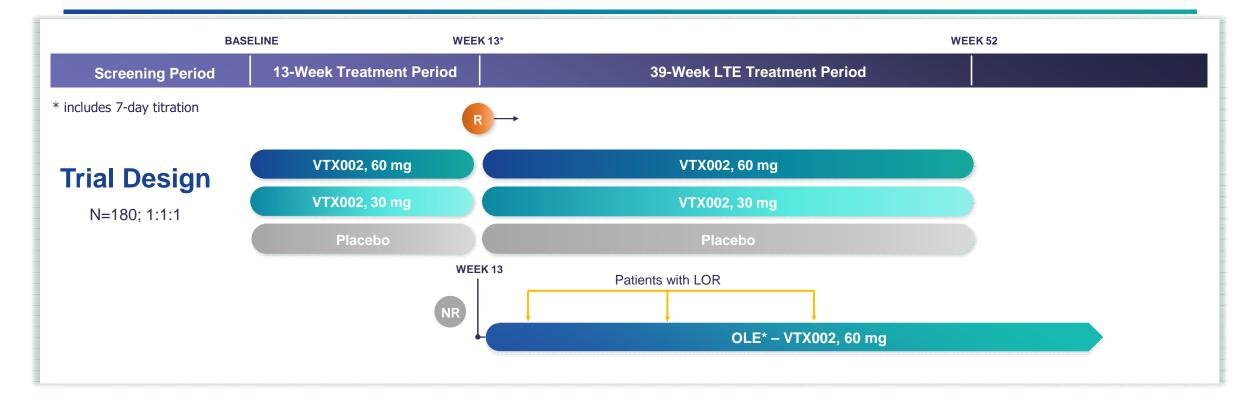
• Demonstrated consistent, sustained reduction of lymphocytes up to 65% across multiple dose groups



Phase 2 Induction Trial in Moderate-to-Severe Ulcerative Colitis

KEY TAKEAWAYS

- Powered for primary endpoint of clinical remission
- Trial may serve as the first of two pivotal trials required for registration

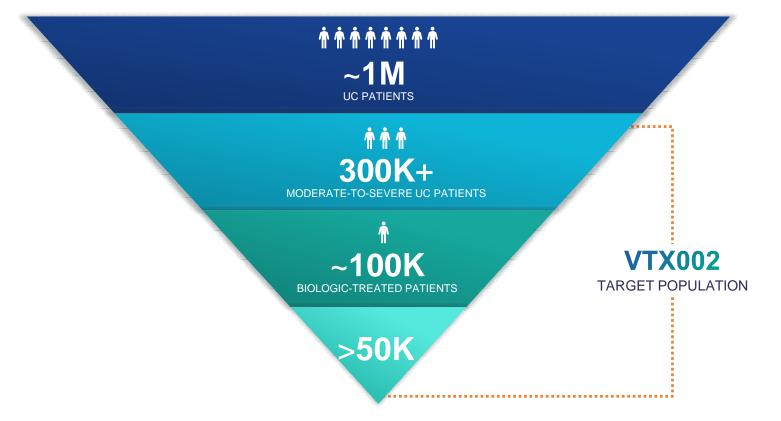




Note: Phase 2 tablet doses of 30mg and 60mg provide comparable VTX002 exposure as Phase 1 suspension doses of 20mg and 40mg, respectively *Induction and OLE non-responder dosing includes 7-day titration period followed by 12 weeks of placebo or VTX002 dose

Underpenetrated Market for Biologic Refractory Patients

Addressable UC Patient Population in US

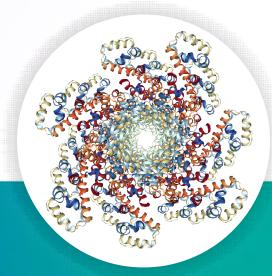


- Existing agents leave room for new treatments
- Novel oral agents may expand penetrance of treated moderate-tosevere UC population beyond current ~25-30%
- S1P well positioned to emerge as leading oral therapeutic class based on its attractive class efficacy/safety profile





SELECTIVE NLRP3 INFLAMMASOME INHIBITORS for systemic and CNS indications



Rationale for Targeting the NLRP3 Inflammasome

NLRP3 Inflammasome Inhibitors Target IL-1β, a Key Driver of Inflammatory Disease

<i>In viv</i> o Evidence	Genetic Evidence	Clinical Validation of Downstream Target
 The NLRP3 inflammasome can become overactive in the presence of persistent tissue damage or crystal deposits Inflammasome activation results in release of IL-1β & IL-18 recruiting neutrophils and driving Th17 response This leads to pyroptosis and further tissue damage 	 Gain-of-function mutations in the NLRP3 gene, associated with certain severe orphan inflammatory diseases, are classified as cyropyrin-associated periodic syndromes (CAPS) 	 IL-1β signaling, downstream of inflammasome activation, is a clinically-validated, anti-inflammatory target with biologics Ilaris® (\$873M sales in 2020) approved for CAPS and other orphan periodic fever syndromes



NLRP3 Inhibitor Program Summary

Peripheral NLRP3 Inhibitor: VTX2735

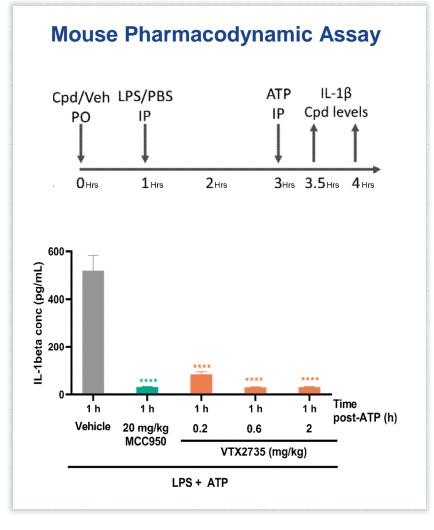
- Selective NLRP3 inhibitor
- Well tolerated in GLP safety and tox assessment
- Phase 1 completed with attractive safety/tolerability profile and evidence of pharmacodynamic activity
- Phase 2 trial in CAPS planned Q4 2022; additional indications are being evaluated

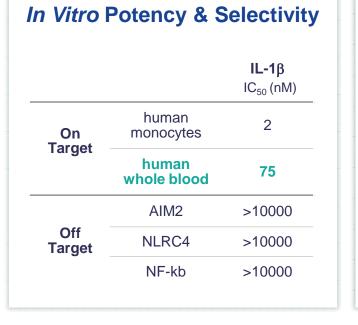
CNS NLRP3 Inhibitor: VTX3232

- Selective compounds generated with high CNS bioavailability
- Novel and proprietary lead series
- Plan to file IND in Q4 2022
- Potential to be first, truly CNS-directed NLRP3 inhibitor to enter clinic



VTX2735 is a Selective & Orally Bioavailable NLRP3 Inhibitor





Non-Human Primate PK IV Clearance: 1.6 mL/min/kg; Oral Bioavailability: 80% 100000-Concentration (ng/mL) 5 mg/kg PO 10000 1000 100-10-1-12 18 24 0 Time (h)

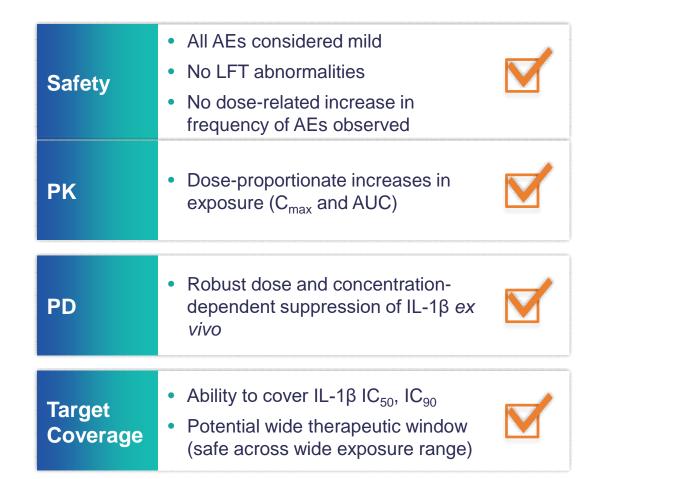
KEY TAKEAWAYS

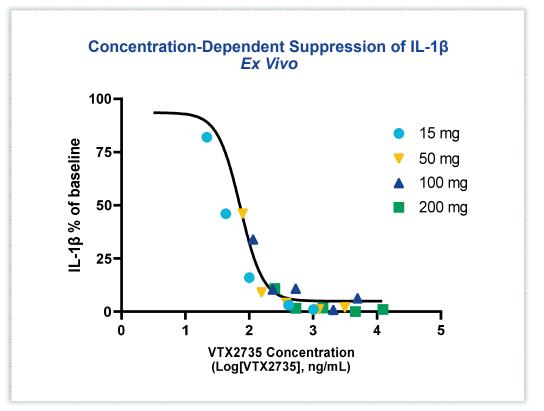
- Well-tolerated preclinically in IND-enabling GLP studies
- Oral bioavailability (80%) in NHP and dose-proportional exposure that predicts potential for wide safety margins based on PK/PD modeling



Summary of VTX2735 Phase 1 Results

Excellent Safety and Pharmacodynamic Activity





Data from Day 10 of Phase 1 MAD, 1 to 8h post-dose $\textit{Ex vivo}\,\text{LPS}$ plus ATP-mediated IL-1 β release assay



VTX2735 Has Broad Activity Against Multiple NLRP3 Mutations

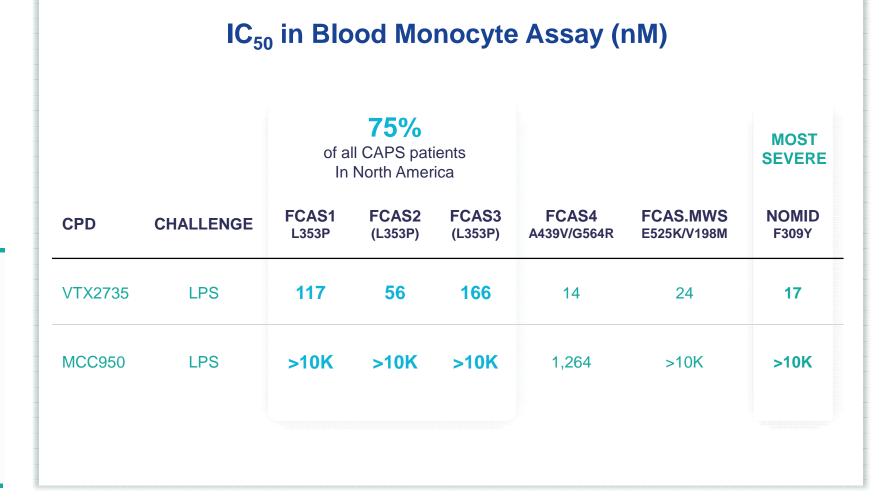
Potential for Differentiation in CAPS Setting*

What is CAPS?

An ultra-orphan auto-inflammatory disease caused by various mutations in NLRP3 and characterized by inappropriate release of IL-1 β and symptoms of recurrent systemic inflammation

KEY TAKEAWAY

VTX2735 blood assay data from CAPS patients suggest inhibitory activity across several mutations: FCAS, MWS and NOMID subset of CAPS patients.

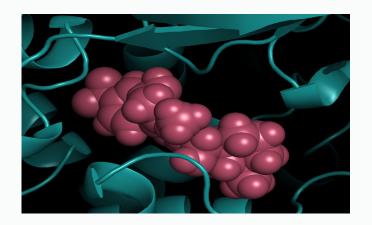


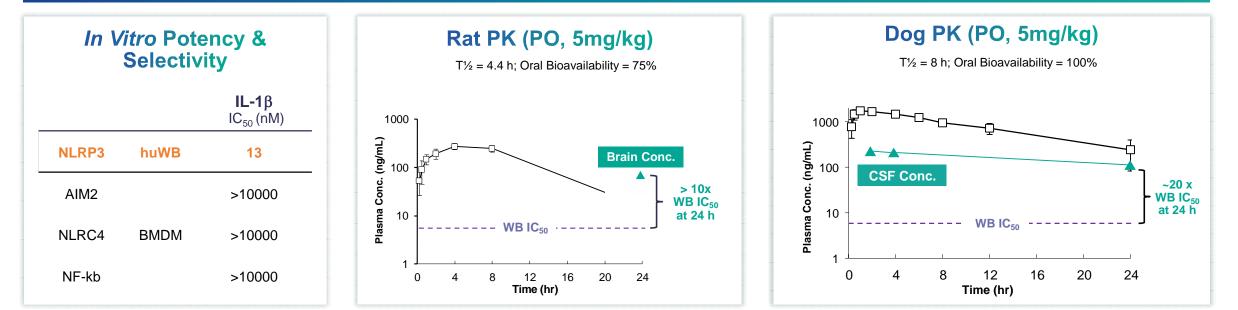


CNS-Penetrant NLRP3 Inhibitor VTX3232

KEY TAKEAWAYS

- Novel, potent, brain-penetrant inhibitor of NLRP3
- 13 nM IC₅₀ in human whole blood IL-1 β release assay
- Unique structural chemotype vs. peripheral NLRP3 inhibitors
- Provisional application filed June 2021
- IND filing in Q4 2022; Phase 1 start in Q1 2023





NLRP3 Program Clinical Development Plan







Our Comprehensive NLRP3 Portfolio Targets a Broad Range of Major Inflammatory Diseases

NLRP3

Systemic Diseases

Peripheral NLRP3 inhibitors are designed to treat cardiovascular, rheumatic, fibrotic and rare genetic diseases

Neuroinflammatory Diseases

CNS-directed NLRP3 inhibitors are designed to treat a range of neurodegenerative disorders, such as Alzheimer's and Parkinson's disease



- Cardiovascular
- Rheumatic
- Fibrotic Diseases
- Rare Genetic Diseases

Our solution: VTX2735

₹3 •

- Alzheimer's Disease
- Parkinson's Disease
- ALS
- Multiple Sclerosis

Our solution: VTX3232





PROJECTED PIPELINE CATALYSTS AND SUMMARY

Projected Catalysts Over Next 24 Months





Investment Highlights

EFFICIENT & PRODUCTIVE IMMUNOLOGY PLATFORM

- Internal R&D engine designed to generate candidates to address autoimmune and inflammatory diseases with high unmet need
- **100% commercial rights** to entire portfolio; long patent life for all product candidates

POTENTIALLY DIFFERENTIATED MEDICINES

- Multiple selective, oral, small molecule product candidate portfolio:
 - VTX958: allosteric TYK2 inhibitor for multiple autoimmune indications
 - VTX002: peripherally-restricted S1P1R modulator for ulcerative colitis
 - VTX2735: peripheral NLRP3 inhibitor for multiple autoimmune indications
 - VTX3232: CNS-targeted NLRP3 inhibitor for multiple neurodegenerative indications

TARGET MAJOR INFLAMMATORY & IMMUNOLOGY DISEASE MARKETS

- Our portfolio can address I&I markets, such as psoriasis, IBD, and other indications
- Opportunity to disrupt existing markets dominated by biologics with varying degrees of efficacy and safety in order to:
 - Capture refractory patients
 - Expand market share of moderate-to-severe patient populations with patient-friendly oral therapy

CAPITAL-EFFICIENT BUSINESS MODEL

- Over \$339 million raised from dedicated biotech investors
- Cash & equivalents and marketable securities balance of \$273.1M as of March 31, 2022; Runway into H1 2024





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