Corporate Presentation

Restoring Health, Transforming Lives Through Innovation

Q2-2026 I OTCQB:ZVSA

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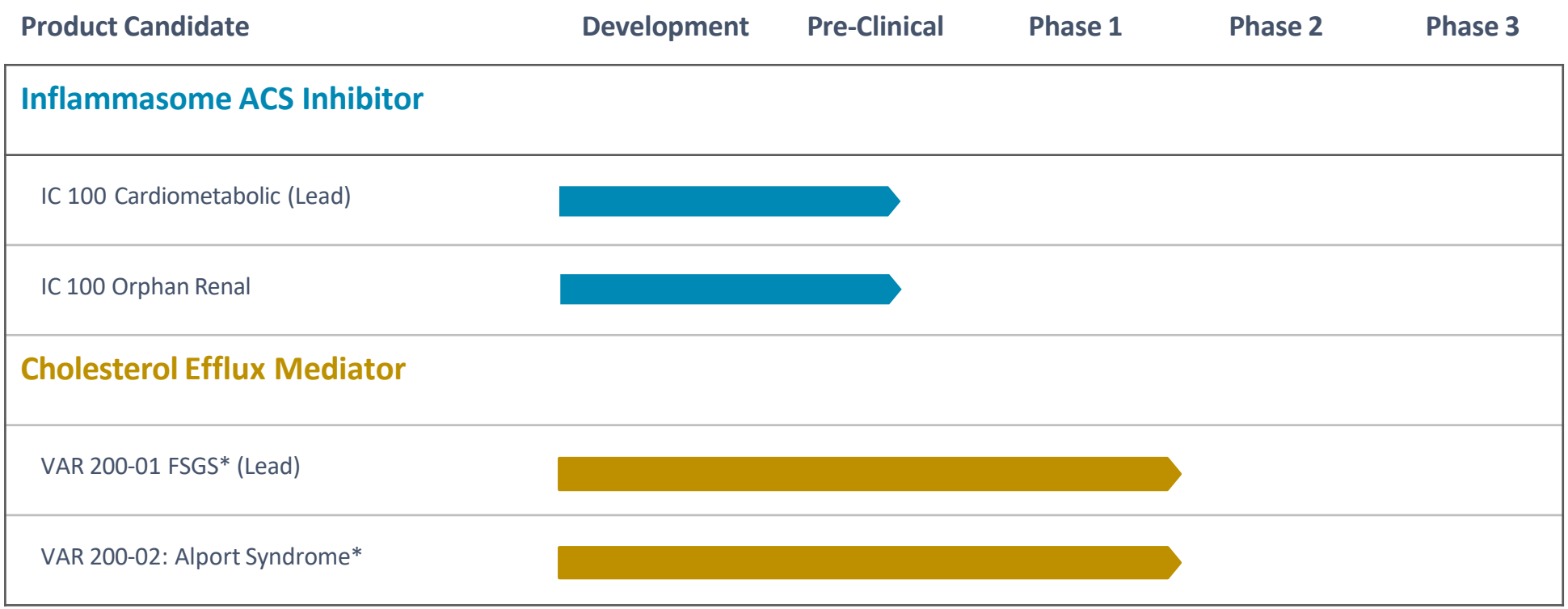
Key Investment Highlights

- **Large market opportunity (> $100B TAM) across inflammatory and renal diseases including high-value orphan indications IC 100 and VAR 200**
- **Differentiated pipeline including a novel approach targeting ASC to inhibit the inflammasome**
  - IC 100: targets inflammasome-driven cardiometabolic conditions associated with obesity and other inflammatory disorders
  - VAR 200: a cholesterol efflux mediator representing a disease-modifying therapy for Focal Segmental Glomerulosclerosis (FSGS), Alport Syndrome and Diabetic Kidney Disease (DKD)
- **Strong preclinical MOA and safety data, recently published competitor data validates inflammasome approach**
- **Near-term value creating milestones including filing IND/advancing Phase 1 for IC 100 and completing FSGS/Alport Phase 2a trials for VAR 200**
- **Strong IP position covering composition of matter, drug/device combinations and potential orphan exclusivity**

Two Proprietary Product Platforms

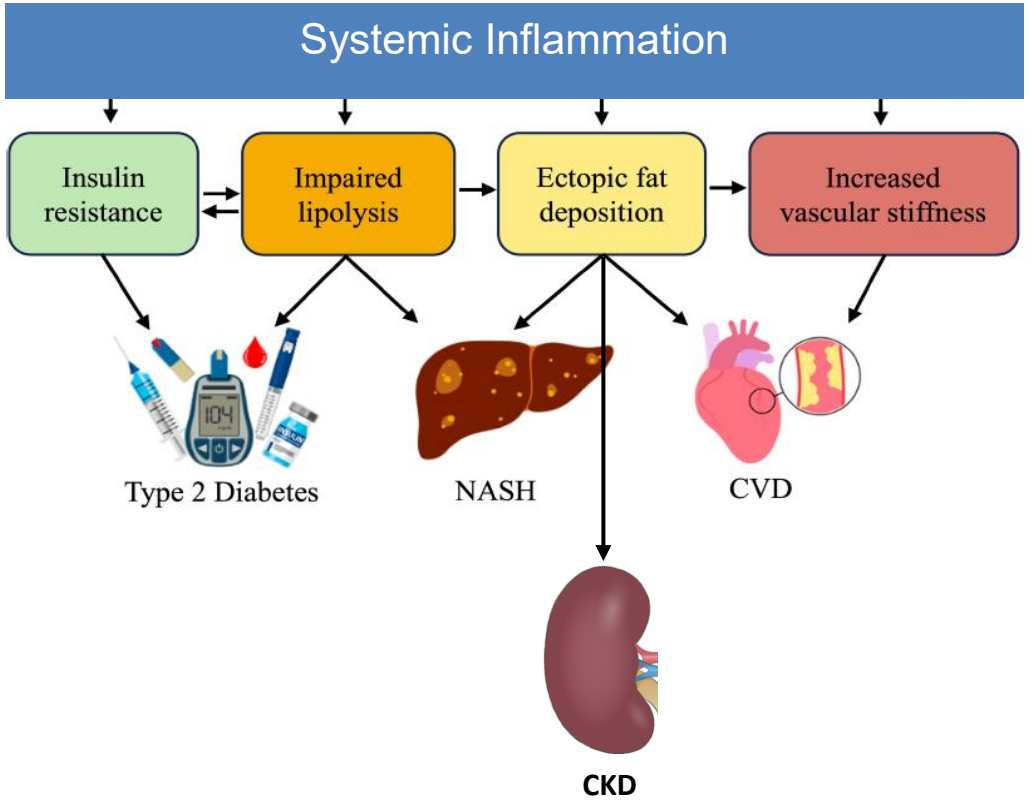
First-in-Class Drugs for Inflammatory and Renal Diseases



*Orphan Disease

Critical Unmet Need to Address Inflammation-Driven Diseases

- Inflammation-driven diseases have become an epidemic with increasing prevalence of obesity
- Systemic inflammation is a common factor in a wide variety of disease processes
- ~38 million people with type 2 diabetes[1], ~15 million people with Nonalcoholic Steatohepatitis (NASH)[2] and ~35 million people with Chronic Kidney Disease (CKD)[3]
- Cardiometabolic diseases (CMDs) are an important and growing subset of inflammatory disorders
  - ~38 million US adults with Cardiovascular Kidney Metabolic Syndrome[4]
- CMDs are interconnected, multi-organ processes and therefore a systemic approach may be required



Adapted from Turner et al. Obes Rev. 2025 Nov;26(11)

1. National Diabetes Statistics Report, May, 15 2024; 2. The Global Epidemiology of Nonalcoholic Fatty Liver Disease (NAFLD) and Nonalcoholic Steatohepatitis (NASH), January 3, 2023; 3. Chronic Kidney Disease in the United States, May 15, 2024; 4. Prevalence of Cardiovascular-Kidney-Metabolic Syndrome Stages in US Adults, May 8, 2024

IC 100 Targets ASC/ASC Specks to Control Perpetuation of Damaging Inflammation

Driving Innovation Across Cardiometabolic and Renal Diseases

- **Total accessible market:** $105B in 2024; $186B in 2034[1]
- **Lead indication:** Cardiometabolic conditions, with indication expansion potential in orphan renal diseases
- **Broad Tissue Penetration**: Brain, spinal cord, heart, lungs, kidney, liver
- **Preclinical MOA proof-of-concept:** 10 cardiometabolic and CNS conditions (including diabetic nephropathy, stroke-related cardiovascular injury, Parkinson's disease)
- **Good preclinical safety:** No immune suppression; low immunogenicity (9%); no drug-related AEs/histopath changes at high doses
- **Strong IP:** Exclusive global license, all indications; 6 US patents; 14 foreign patents/allowed applications, 59 pending applications

1. Anti-inflammatory Biologics Market Market Size and growth 2025 – 2034, July 18, 2025

VAR 200 Targets Renal Lipids to Attenuate Renal Damage and Disease Progression

Advancing Novel Therapies for FSGS and Rare Kidney Diseases

- **Total accessible market:** $18B in 2024; $30B in 2034[1]
- **Lead indication:** FSGS, with indication expansion potential into Alport Syndrome and DKD
- **Rare pediatric disease voucher potential**: FDA-authorized pediatric enrollment in Phase 2 FSGS trial
- **Preclinical proof-of-concept:** FSGS, Alport Syndrome, DKD
- **Strong IP:** Exclusive global license, kidney diseases; potential orphan drug exclusivity (7 years - US; 10 years - EU); drug/device combination

1. Nephrology Drugs Market Size, Share, and Trends 2024 – 2034. Precedence Research, Nov 13, 2024

Inflammatory Opportunity

Inflammasome ASC Inhibitor: IC 100

Market Landscape for the Innate Immune Market: Rapidly Evolving

Driving Innovation Across Cardiometabolic and Renal Diseases

Danger signals / DAMPs / PAMPs → Inflammasome Sensors (NLR) → ASC speck → Caspase-1 → IL-1β / IL-18 → IL-6 → CRP & systemic inflammation → Adaptive cytokines / T-cell/B-cell responses

Adaptive Immune System

- TNF inhibitors (Humira, Enbrel)
- IL-23 / IL-12-23 inhibitors (Skyrizi, Stelara, Tremfya)
- IL-17 inhibitors (Cosentyx, Taltz)
- Th2 inhibitors (Dupixent)
- Crowding + biosimilar erosion

Mature & competitive – large revenues / limited whitespace

IL-6/JAC Bridge

- IL-6R blockers (Actemra, Kevzara)
- JAK inhibitors (Rinvoq, Xeljanz, Olumiant)
- Innate-linked cytokine suppression can reach multi-billion revenues downstream of inflammasomes

Downstream control of inflammation – does not block ASC / NLR sensor activation

Innate Immunity

- ASC inhibition (IC 100) – blocks PYD–PYD interactions, ASC speck formation, caspase-1 activation
- NLRP3 inhibitors: IL-1β / IL-18 small-molecule inhibitors – emerging pipeline
- IL-1β / IL-18 inhibitors (anakinra, canakinumab
- Significant M&A and Blockbuster Potential

Most upstream intervention point – potential to redefine treatment of multiple inflammatory diseases

Critical Unmet Need to Reduce Inflammation-Driven Obesity Comorbidities

4 of 5 Million Annual Deaths Associated with BMI Are Related to Comorbidities[1]

- Standard-of-Care Incretin Drugs Regulate Appetite and Caloric Intake to Reduce Excess Body Weight
- Incretin Drug Discontinuation Rates Are High Leading to Significant Weight Regain in 8 - 12 weeks [2]
- Weight Regain Exacerbates Adipose Tissue & Systemic Inflammation That Drive Comorbidities[3]
- Neither Current Nor Pipeline Weight Loss Drugs Address Systemic Inflammation That Drives Comorbidities

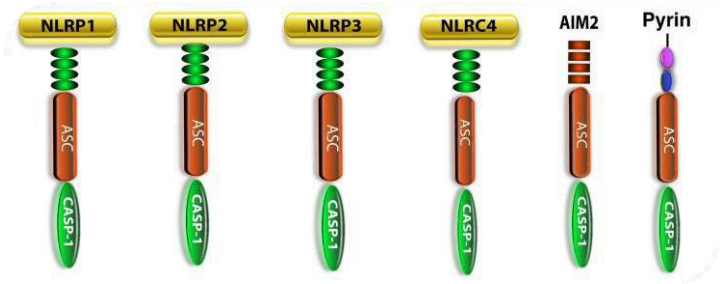
Innovative Drugs Are Needed to Attenuate Systemic Inflammation That Drives Comorbidities

1. World Obesity Atlas, 2024; 2. Wu H, Yang W, Guo T, et al. Trajectory of the body weight after drug discontinuation in the treatment of anti-obesity medications. BMC Med. 2025 Jul 22;23(1):398 ; 3. Li W, Chen W. Weight cycling based on altered immune microenvironment as a result of metaflammation. Nutr Metab (Lond). 2023 Feb 22;20(1):13;

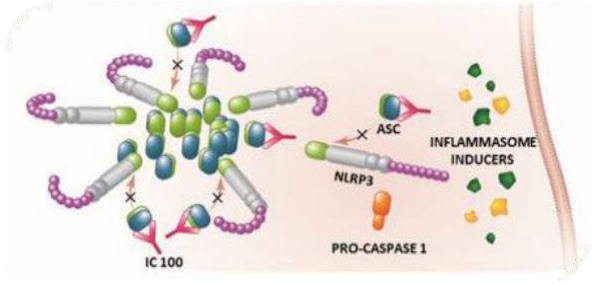
IC 100 Targets the Inflammasome Through ASC Rather Than NLRP3

ASC Inhibition Expected to Better Control Inflammation

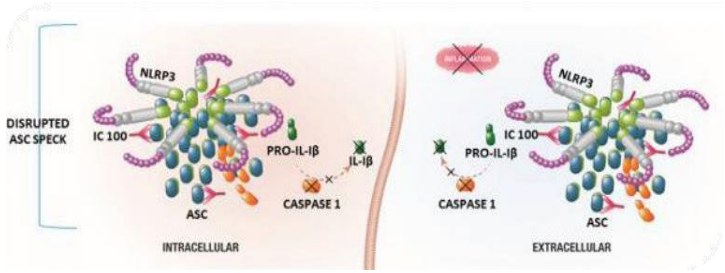
Potentially Inhibits 12 or More Types of Inflammasomes to Control Inflammation Regardless of Its Triggers



Inhibits Intracellular ASC to Block Inflammasome Formation & Initiation of the Inflammatory Cascade



Inhibits Intra- and Extracellular ASC Specks, Disrupting Their Structure & Function to Block Perpetuation of Damaging Inflammation



Multiple Inflammasomes Trigger Many Diseases/Conditions

Disease/Condition	Inflammasomes Implicated
Metabolic Conditions	
Obesity	AIM2, NLRP3
Insulin Resistance	AIM2, NLRP1, NLRP3, NLRC4, NLRP6
Diabetic Nephropathy	AIM2, NLRP3
Neurological Conditions	
Parkinson's Disease	NLRP1, NLRP3, AIM2
Alzheimer's Disease	AIM2, NLRP1, NLRP3
Multiple Sclerosis	AIM2, NLRP1, NLRP2, NLRP3, NLRC4

NLRP3 Inhibitors Block Only One Type Inflammasome
Don't Address ASC Specks To Block Systemic Perpetuation of Inflammation

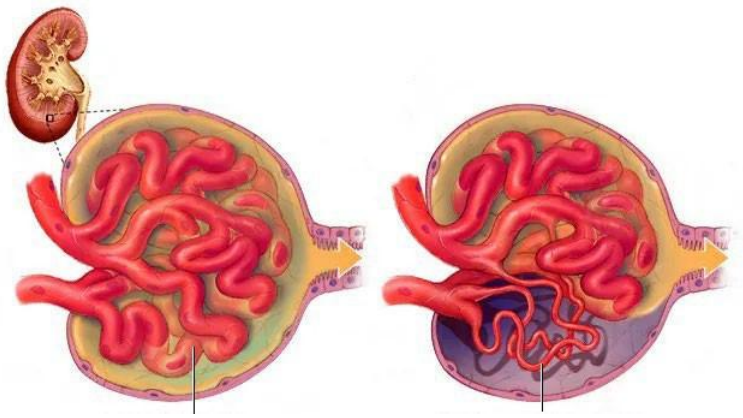
IC 100 Attributes Versus Key Inflammasome Competitors

Attribute	IC 100 (mAb)	VTX 3232 (SM)	VTX 2735 (SM)	NT-0796 (SM)	NT-0249 (SM)	NN6022-0001 (SM)	VENT-02 (SM)	RG6418 (SM)	DFV 890 (SM)
Company	**ZyVersa**	**Ventyx**		**NodThera**		**Novo Nordisk**	**Ventus**	**Roche**	**Novartis**
Molecular Target	ASC/ASC Specks	NLRP3	NLRP3	NLRP3	NLRP3	NLRP3	NLRP3	NLRP3	NLRP3
# Inflammasomes Targeted	Up to 12	1	1	1	1	1	1	1	1
Body System Target	CNS & Peripheral	CNS	Peripheral	CNS & Peripheral	CNS	Peripheral	CNS	Peripheral	Peripheral
Target Specificity	Yes	No	No	No	No	No	No	No	No
Latest Development Stage	Preclinical	P2	P2	P1b/2a	P1	P1	P2	P2	P2
Indications	CMD*/ Renal	Obesity/CMD* Parkinson's	Recurrent Pericarditis	Parkinson's Obesity/CV	TBD	CMD*	Obesity/Osteoarthritis Parkinson's	Obesity/ASCV#; Parkinson's	Cardiovascular Risk Reduction
Expected Dose Frequency	Quarterly to Twice Annually	Once Daily	Once Daily	Once Daily	Once Daily	Once Daily	Once Daily	Once Daily	Once Daily

*Cardiometabolic Diseases; ~Coronary Artery Disease; #Atherosclerotic Cardiovascular Disease

Cholesterol Efflux Mediator VAR 200

FSGS: A Rare Progressive Kidney Disease with No Approved Therapies



Normal Kidney[1] FSGS Kidney[1]

FSGS

- Glomerular podocyte injury resulting in scarring that is focal (affects only some glomerulus) and segmental (affects only part of glomerulus)
- Leads to impaired kidney filtration causing protein to spill into the urine (proteinuria)
- Proteinuria levels directly correlate with disease progression and kidney failure
- Current Standard of care (SOC): Off label drugs to reduce proteinuria
  - Background Therapy: ACEs & ARBs to control blood pressure & glomerular hypertension
  - First-line Immunosuppressive Therapy: High dose glucocorticoids
  - Second-line Immunosuppressive Therapy: Calcineurin inhibitors
- Despite SOC, FSGS is leading cause of kidney failure proteinuria

40,000[2] FSGS Patients in US

60%[2] Become Treatment Resistant

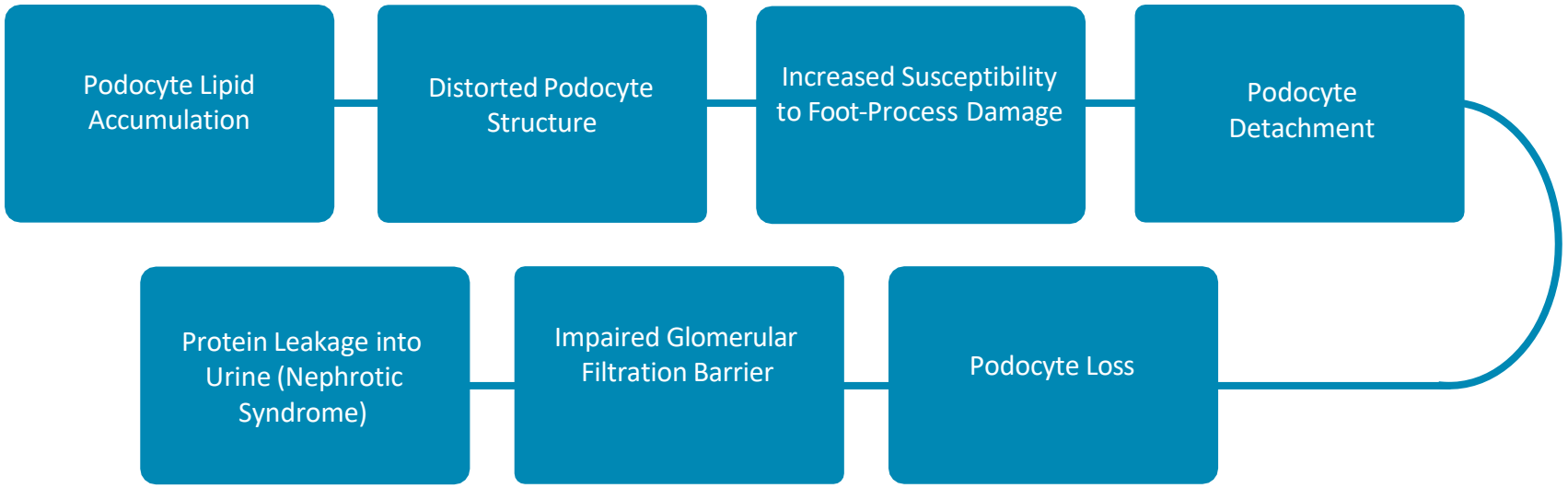
50%[2] Progress to Kidney Failure

1,000/Year[2] Receive Kidney Transplant

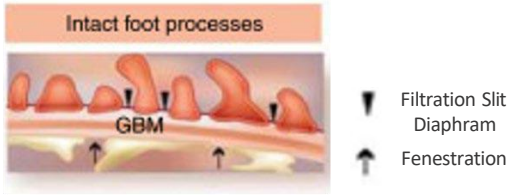
30 – 50%[2] FSGS Recurrence Rate

1. Focal Segmental Glomerulosclerosis (FSGS). Mayo Clinic, January 31, 2025; 2. Nephcure Policy Priorities, 2024

Renal Lipotoxicity Leads to Podocyte Structural Damage, Proteinuria, and Progression of Kidney Disease[1,2]



Normal: *Intact podocyte foot process*



Abnormal: *Flattened podocytes*

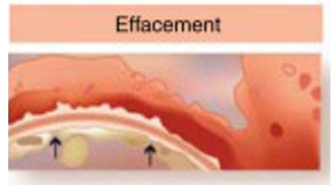
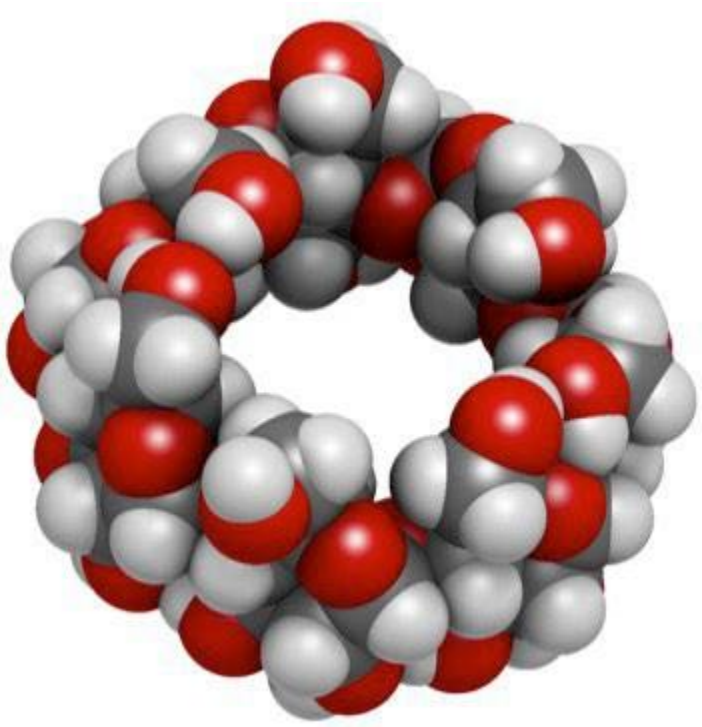


Image Adapted From D'Agati VD: Kidney Int. 2008 Feb;73(4):399-406

1. Mitrofanova, Molina J, Varona Santos J, et al, Hydroxypropyl-β-cyclodextrin protects from kidney disease in experimental Alport syndrome and focal segmental glomerulosclerosis. Kidney Int. 2018 Dec;94(6):1151-1159; 2. Ducasa GM, Mitrofanova A, Mallela SK, et al. ATP-binding cassette A1 deficiency causes cardiolipin-driven mitochondrial dysfunction in podocytes. J Clin Invest. 2019;129(8):3387–3400

Cholesterol Efflux Mediator VAR 200 (2-Hydroxypropyl-Beta Cyclodextrin 2HPβCD) Alleviates Excess Podocyte Cholesterol and Lipids



Space filling model of
β-Cyclodextrin

Comprised of 7 Sugar Molecules Bound Together in a 3-D Ring

- 2HPβCD has a hydrophobic core that forms an inclusion complex with intracellular cholesterol and lipids removing them from the kidney
- 2HPβCD is believed to mediate active cholesterol and lipid removal through upregulation of cholesterol efflux transporters ABCA1 and ABCG1
- Cholesterol and lipid removal protect renal structure and function

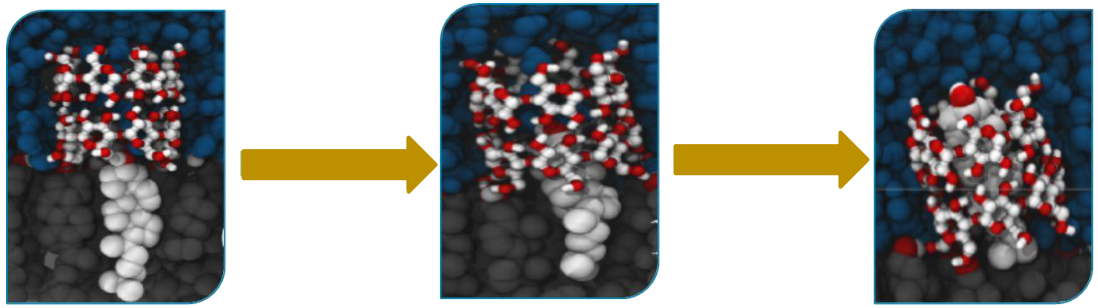


Image of βCD Adapted From Lopez et al: LoS Comput Biol 7(3): e1002020. doi:10.1371/journal.pcbi.1002020

Comparison of FSGS Drugs in Development

Attribute	ZyVersa	Travere	Dimerix
Drug	**VAR 200**	**Sparsentan**	**DMX-200**
Molecule	2HPβCD	Endothelin & Angiotensin II Receptor Antagonist	Chemokine Receptor (CCR2) Antagonist
MOA	Cholesterol/Lipid Efflux	Vasodilation	Anti-inflammatory
Latest Phase	2a	Pending FDA Review	3
Dosing Form	IV, SubQ	Oral	Oral
Positioning	Attenuates key renal pathologies by removal of excess lipids to protect renal structure and function, delay progression	Only non-immunosuppressive oral medication that directly targets podocyte injury by optimally blocking the endothelin A receptor and the angiotensin II subtype 1 receptor	With use in combination with ARBs synergistically disrupts the cycle of damage in FSGS
Treatment Position	In combination with SOC	Replace ACE & ARBs; in combination with immunosuppressants	In combination with ARBs, immunosuppressants
Other Indications	Alport Syndrome, DKD	IgAN	Other Rare Renal Considered

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