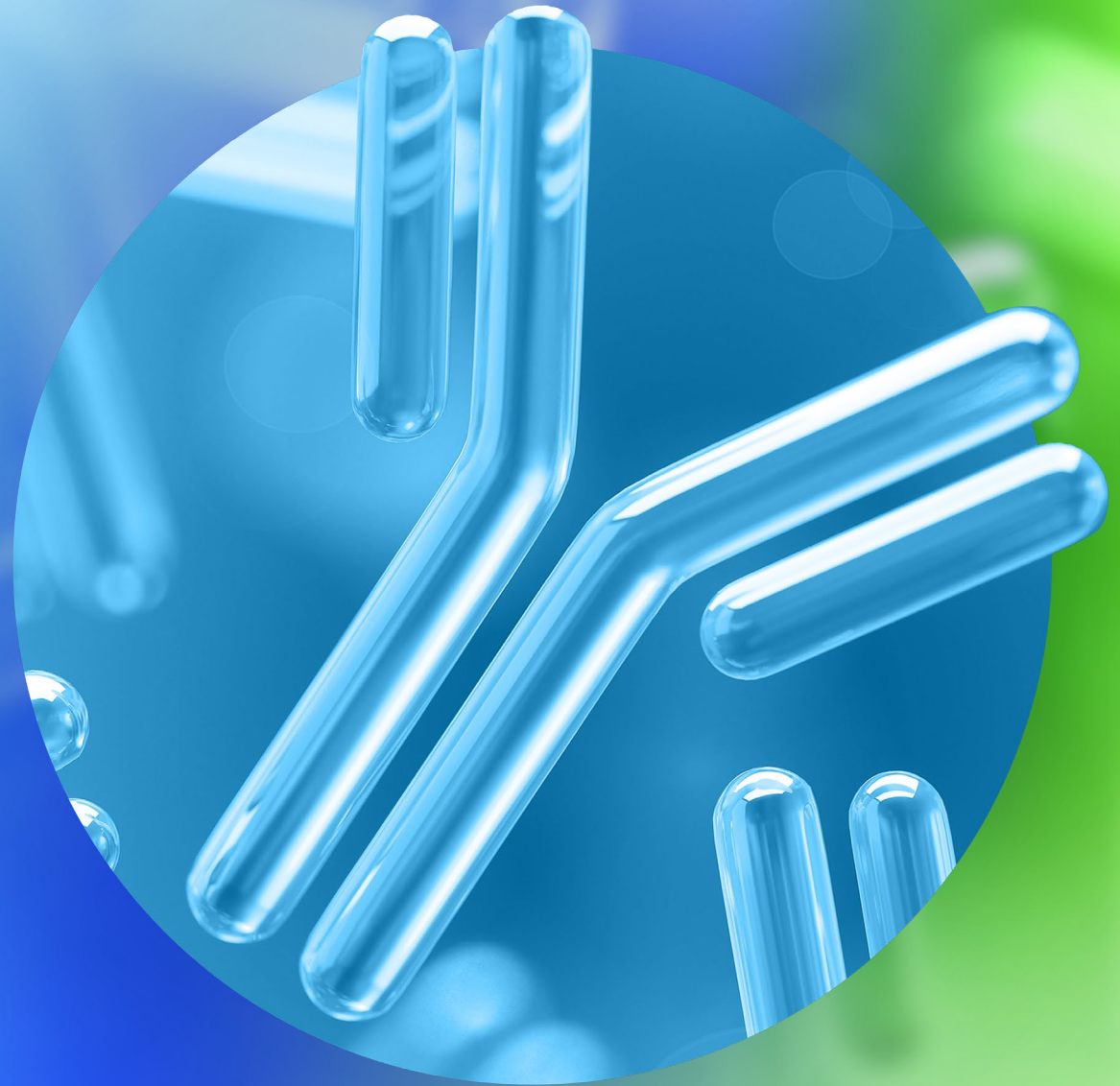




Restoring Vision Through the Science of Renewal



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Designing Best-in-Disease Multi-Functional Antibodies for Retinal Vascular Disease



Retinal Vascular Disease Represents Large Markets with Ongoing Unmet Need

- Large, chronic patient populations with sustained commercial demand
- Current standard-of-care therapies primarily inhibit VEGF, leaving residual disease activity
- Recent launches (e.g., Vabysmo) validate the benefit of addressing multiple disease drivers

Retinal Vascular Disease is Driven by Multiple Pathways

- Vascular leakage, barrier dysfunction, and inflammation each contribute to disease progression
- VEGF inhibition alone does not fully address this biology
- Wnt signaling plays a distinct, complementary role in maintaining vascular integrity

Our Platform Enables Rational Combinations of These Pathways

- Deep experience in FZD/LRP biology and antibody engineering
- Capability to design antibodies that combine Wnt activation with inhibition of other relevant pathways
- Broad and growing IP portfolio covering antibody formats, mechanisms, and disease targets

Our Pipeline has Multiple Candidates Designed to Deliver Best Anatomic Outcomes

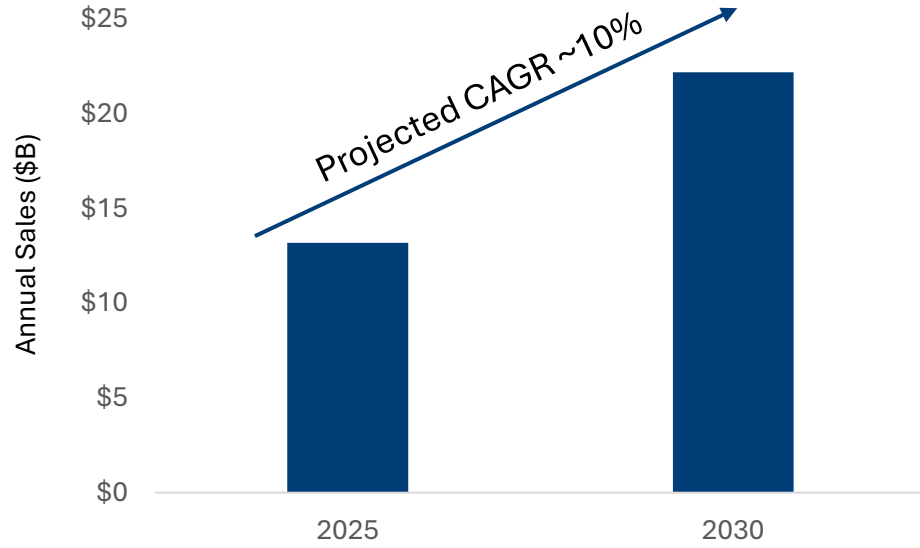
- SZN-8141 (Wnt/VEGF) and SZN-8143 (Wnt/VEGF/IL-6) are designed for best retinal drying
- Preclinical results demonstrate superiority to standard of care and MOA synergy
- IND submission anticipated in the second half of 2026 for SZN-8141



Retinal Vascular Diseases (DME, wet AMD) are Large and Growing Markets with Significant Unmet Need

Retinal Vascular Diseases	
US Prevalence ¹	2.3M
Global Prevalence	>40M ^{2,3}
Anti-VEGF Market ⁴⁻⁶	\$13B in 2025 -> >\$20B in 2030 CAGR: ~10%
Morbidity ⁷	- DME patients incur healthcare costs 2-3x higher vs having diabetes alone ⁴ - AMD is major cause of sight loss in the population >65
Key Products ¹	Anti-VEGF therapies: Aflibercept, Ranibizumab, Faricimab
Key Unmet Needs ⁸	Need for better drying agents Need for longer lasting therapies Need for new therapies beyond VEGF inhibition

Global Anti-VEGF Agents Ophthalmic Drug Market



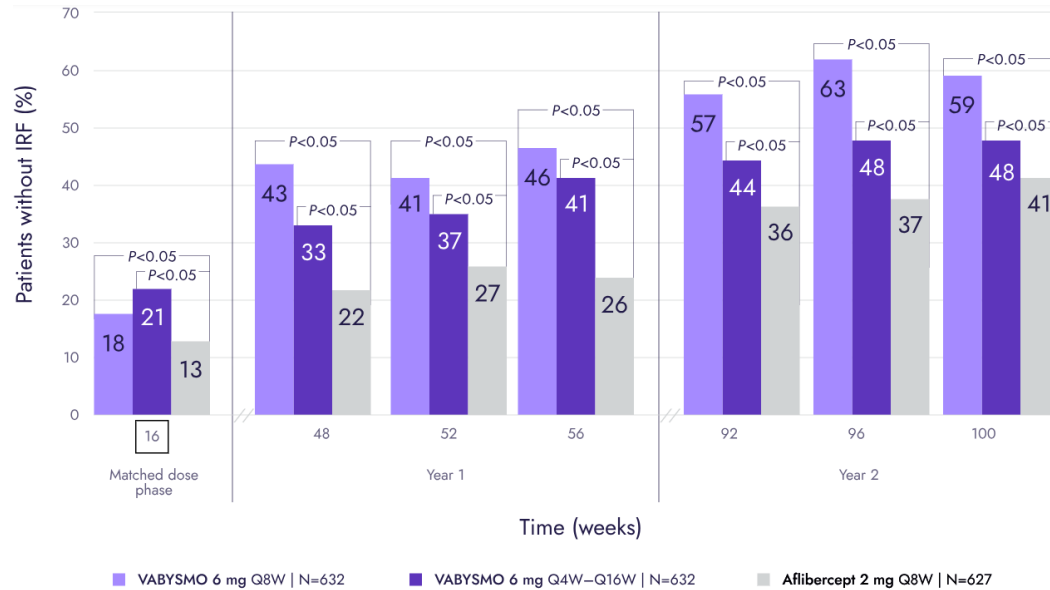
Source: 1. Health Advances DME and wet AMD primary market research for Surrozen - Nov 2024; 2. Im et al, Survey of Ophthalmology, July-Aug 2022; 3. Wong et al, Lancet Global Health Feb 2014; 4. Surrozen estimates from range of research report analyses: Datamonitor – Anti-VEGF wet AMD Grandview Research – Global Ophthalmic Drugs Market Size Outlook 2024, Global Market Insights - Ophthalmic Anti-VEGF Therapeutics; 5. Roche 2025 Financial Report; 6. Regeneron 2025 10-K; 7. Choi et al, 2024 Oct 8;10:e56741 doi: 10.2196/56741; 8. ASRS PATS Survey 2025.



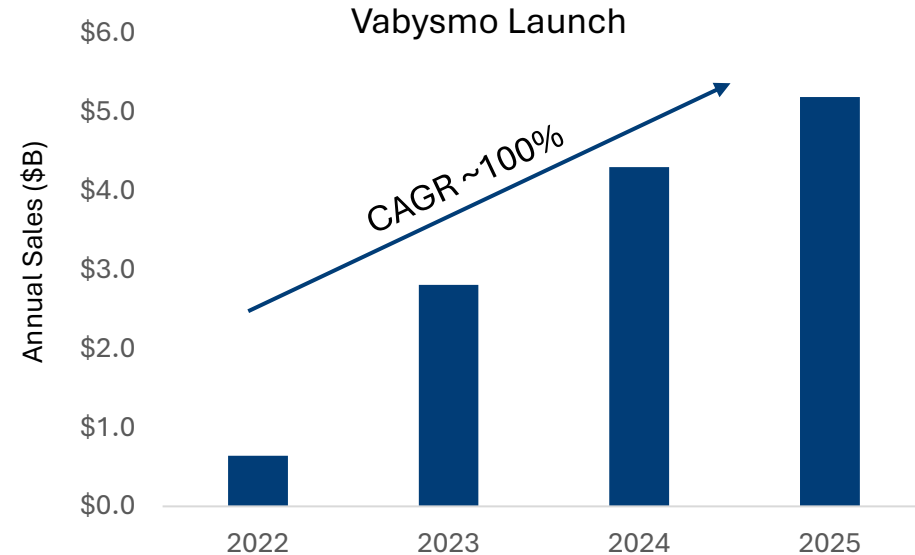
Vabysmo (VEGF + Ang2): Blueprint for VEGF Combinations

Multiple targets drive additional clinical benefit
Incremental dosing leads to rapid adoption

Vabysmo Clinical Outcomes in DME



Global Vabysmo Revenue



Source: Vabysmo Website; Roche Financial Reports 2022 – 2025.

Evidence that Adding to VEGF Inhibition Confers Additive Clinical Benefit



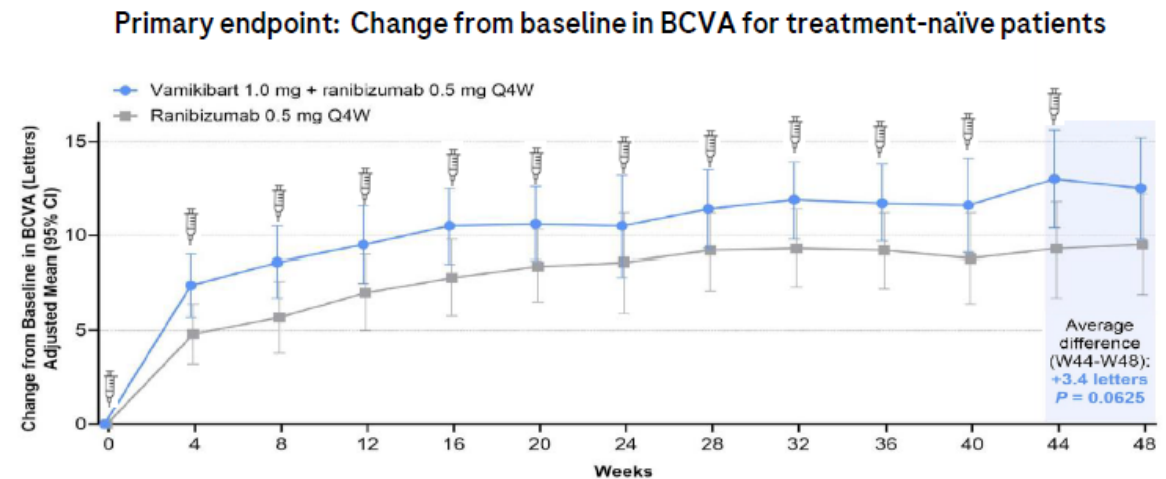
Roche demonstrated IL-6 in combination with VEGF inhibition superior to VEGF alone

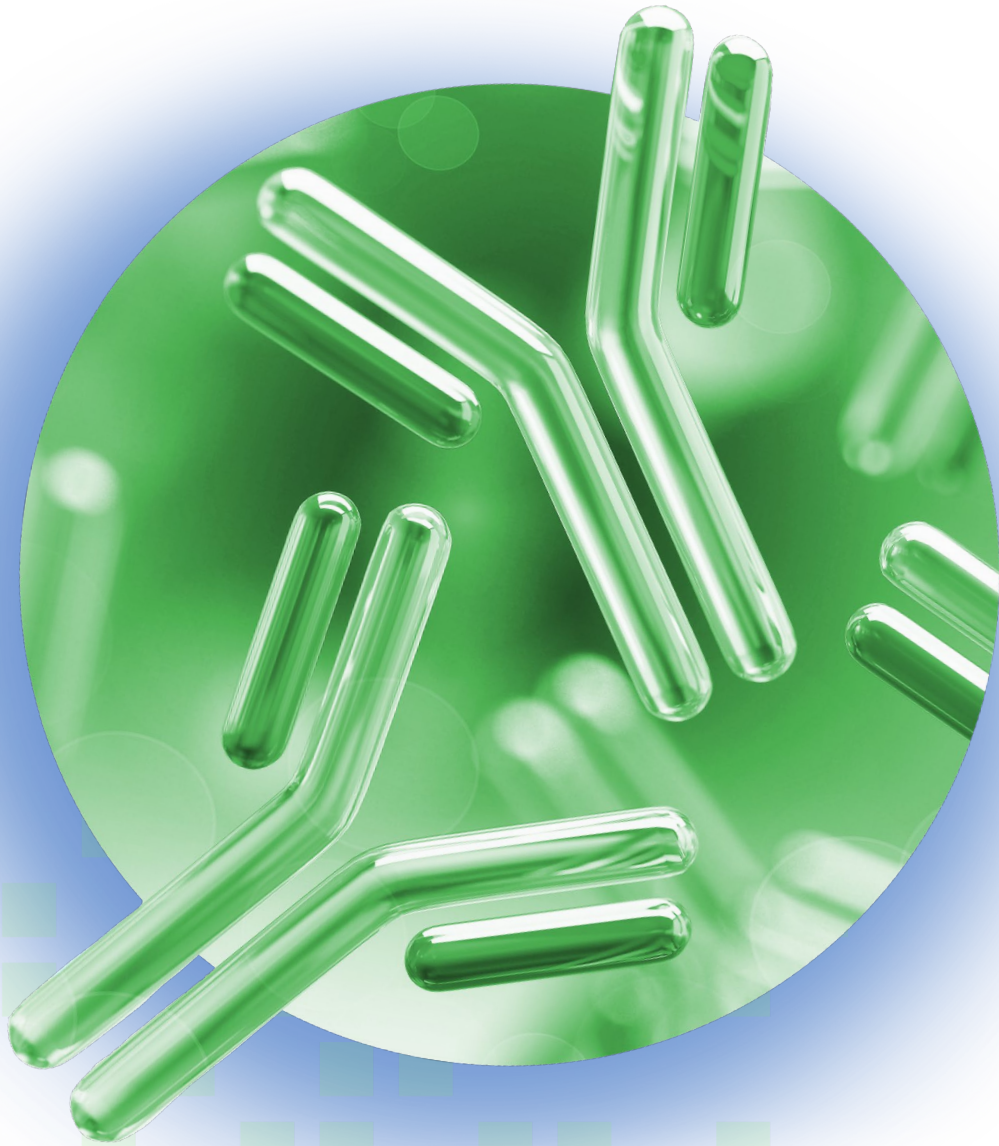
Consistent BCVA gains for combination throughout the study

Large increase in percent of patients achieving greatest visual acuity gains: 44.7% of patients receiving combination gained ≥ 15 BCVA letters vs 28.6% with VEGF alone

Roche pursuing IL6 x VEGF bi-specific

Ph II (BARDENAS): Anti-IL-6 (vamikibart) + anti-VEGF (ranibizumab) showed superior efficacy in DME





Wnt Signaling

A Key Regulator of Vascular
Biology



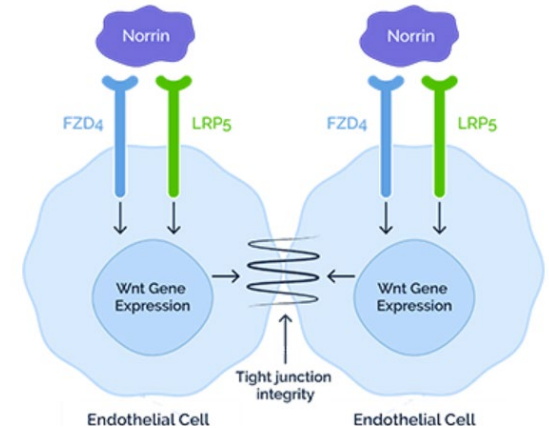
Wnt is A Key Regulator of Retinal Vascular Function

A complementary mechanism to VEGF inhibition

- **Biology:** Wnt signaling is essential for maintaining retinal vascular integrity and barrier function
- **Genetic Validation:** Mutations in FZD4 mediated Wnt signaling cause Norrie disease and FEVR — both marked by severe vascular defects
- **Preclinical Evidence:** Wnt mimetics restore vascular integrity, reduce leakage, and regenerate vasculature in models of retinal injury
- **Clinical Evidence:** Proof of concept established in DME patients with outcomes comparable to anti-VEGF therapy

Wnt Biology in the Retina

Norrin, a Wnt ligand, binds to receptors FZD4 and LRP5, activating Wnt signaling to maintain retinal vascular integrity



Restoret (Wnt agonist) Ph1b/2a Study

- 26 patient DME study
- BCVA gain of ~11 letters at 12 weeks
- Mean reduction in excess retinal thickness of 80%

Wnt Biology is Driving Strategic Investment



SURROZEN'S SZN-413 | 2022

- Potential best-in-class FZD4 /LRP bi-specific antibody for retinal diseases like neovascular AMD and DME
- Licensed to Boehringer Ingelheim in October 2022
- Surrozen is a first-mover, building on the seminal work of our founders and scientific advisors who discovered the Wnt gene and key regulators of the Wnt pathway
- Surrozen received \$12.5M upfront; potential milestones of up to \$586.5M; mid-single to low double-digit royalties
- License scope enables Surrozen to pursue other FZD4 targeted antibodies on its own

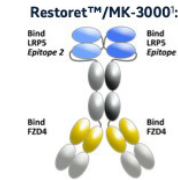


MERCK | 2024

Merck acquired EyeBio in 2024 for **\$1.3B for Restoret (EYE103, MK-3000)**

Ophthalmology

- Completed acquisition of EyeBio
- Restoret™/MK-3000 is an investigational, potentially first-in-class tetravalent tri-specific Wnt antibody for treatment of diabetic macular edema and neovascular age-related macular degeneration



Roche | 2024

Roche acquired AntlerA Therapeutics in 2024 for **a library of anti-FZD/LRP drug candidates**

Investigating Wnt agonism with ANT-Pharm
Acquisition of AntlerA, a leader in Wnt signaling, added a library of anti-FZD/LRP drug candidates

Wnt pathway

ANT-Pharm: A combinatorial Wnt-mimetic library platform

- Extensive scientific expertise on the Wnt pathway in Roche
- Approaches mimicking the natural ligand Norrin, which agonizes the Wnt pathway, have been shown to be effective in restoring and maintaining a sealed blood retinal barrier in preclinical models
- Clinical proof of concept of agonizing Wnt has been demonstrated in DME
- ANT-Pharm is a library of anti-FZD/LRP molecules capable of activating the Wnt Pathway in various cell types and tissues
- Preclinical lead asset, has the potential to be best-in-class treatment for AMD and DME
- Potential to expand to indications beyond Ophthalmology where Wnt signaling plays an important role

NSA figure modified from Owens, H. Cell (2023). Late-onset age-related macular degeneration: DME-diabetic macular edema.

Surrozen Advantage: Defining the Next Era of Retinal Repair



Architects of Drugging the Pathway:

Built by the world's leading experts who discovered the Wnt pathway, its role in tissue restoration and ligand:receptor biology

Antibody Design Leadership:

Deep expertise in FZD/LRP signal modulation – multiple publications on optimizing antibody formats



Next Generation Multifunctional Biologics:

First-in-class multifunctional biologics which combine Wnt activation with modulation of other drivers of retinal disease (VEGF, IL-6)

Broad IP Portfolio:

Our leadership is protected by a robust patent estate, including 8 issued U.S. patents and over 25 pending families globally



Our Development Pipeline



Second Generation Multi-Functional Wnt Targeted Antibodies

Complementary mechanisms enable potential best-in-disease antibodies

SZN-8141 – WNT Activation + VEGF Inhibition

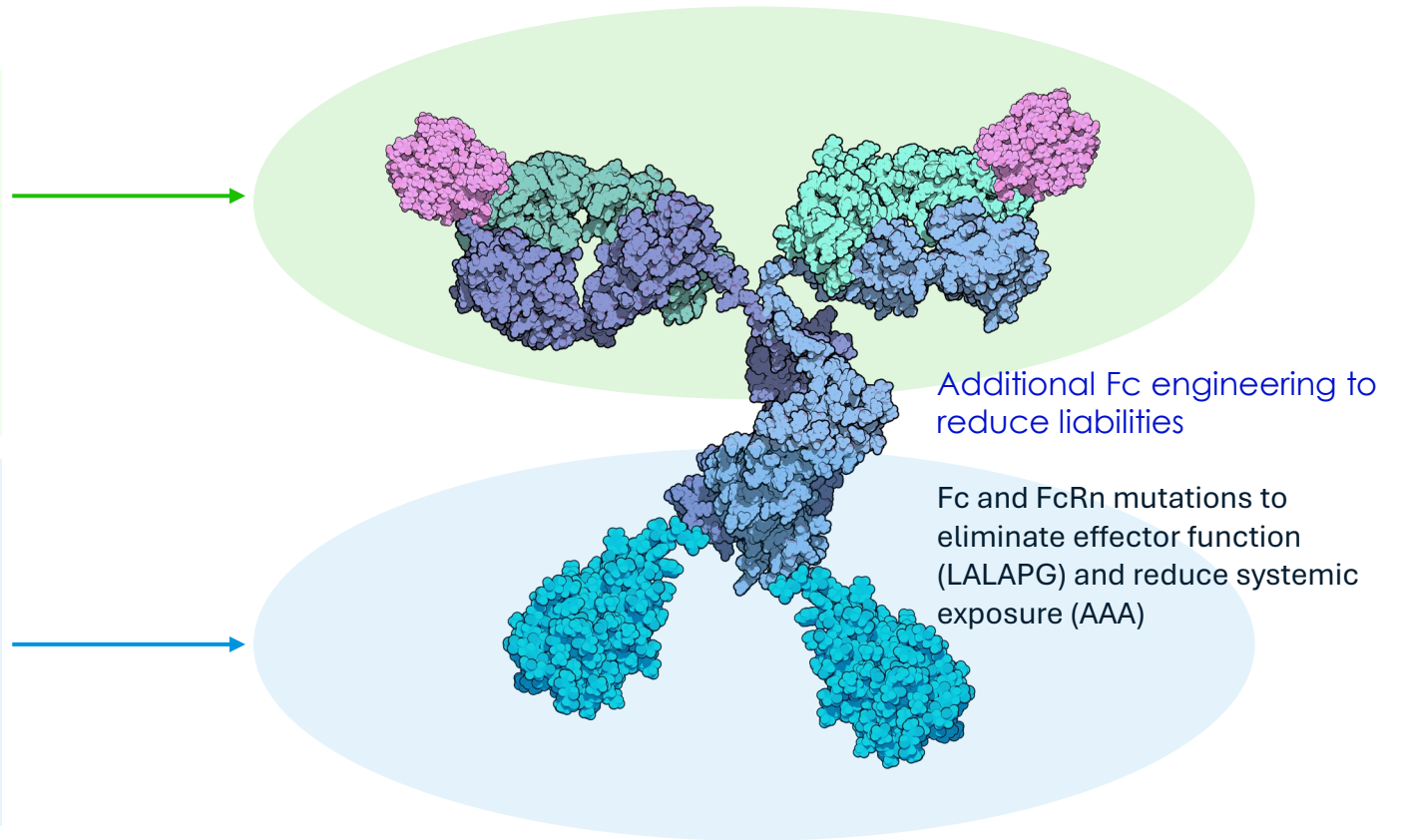
Wnt activation via FZD4/LRP5

- Potential for best-in-class Wnt activation
- Wnt activation upregulates tight junction proteins and restores blood retina barrier function

VEGF inhibition via VEGF decoy receptors

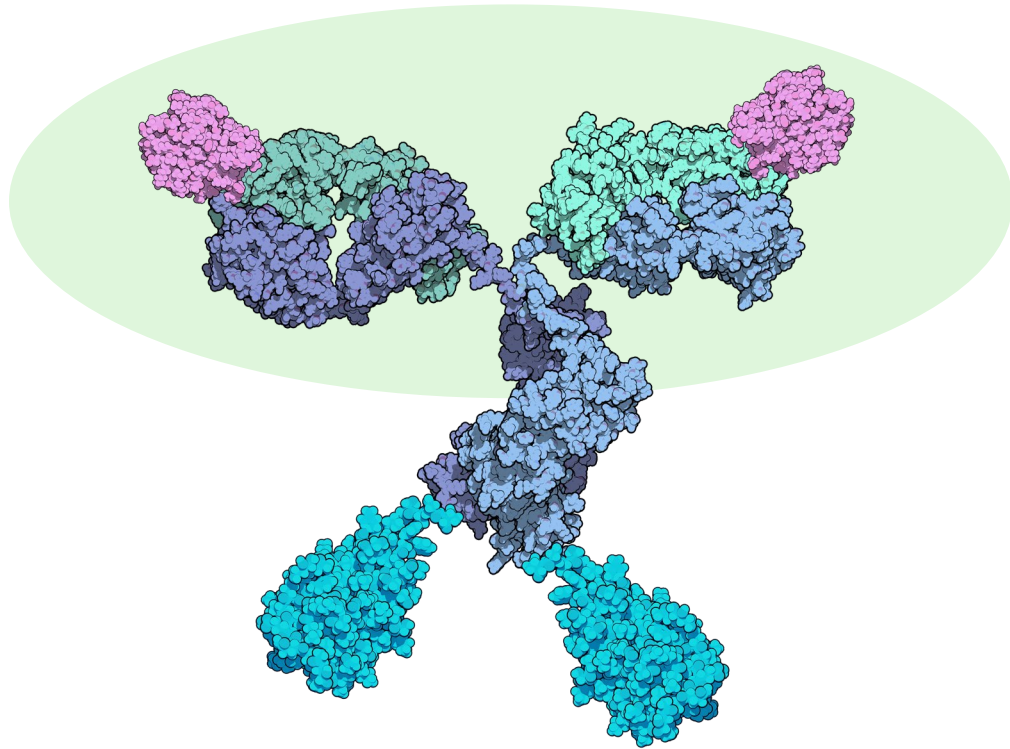
Aflibercept sequences - comparable VEGF-A/PlGF binding affinity

- Reduces neovascularization and vascular leakage



FZD4 IgG1/LRP5VHH/VEGF soluble decoy receptor

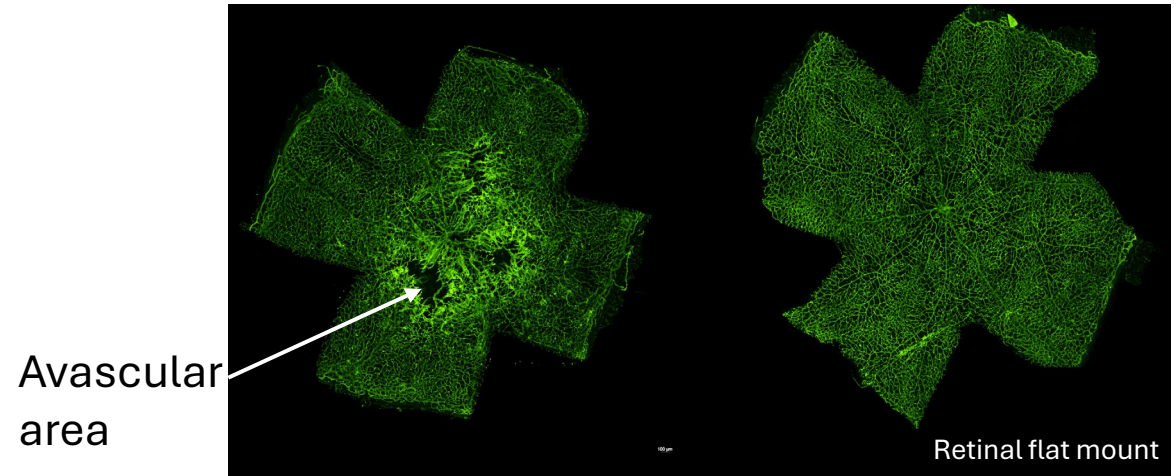
SZN-8141 Restores Retinal Vessel Architecture



Novel mechanism for treatment of retinal vascular diseases that can directly reduce leakage and eliminate pathologic leaky vessels

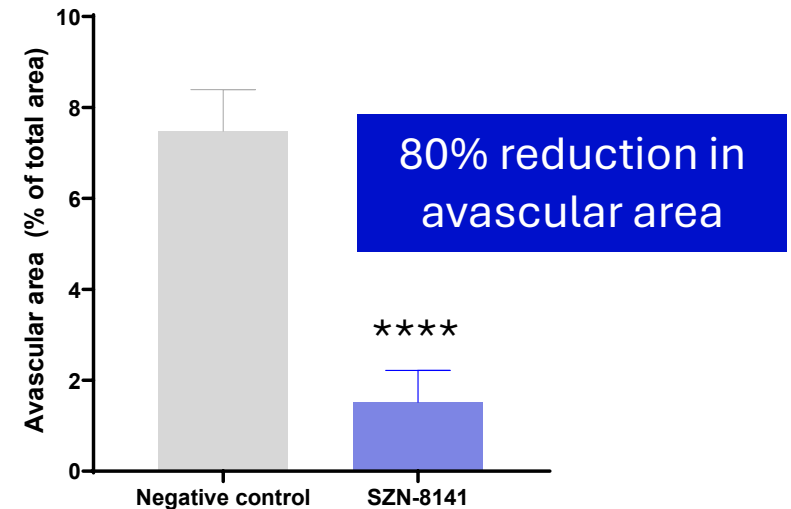
****p<0.0001 v. negative control. N = 12-14 mice per group.

Oxygen Induced Retinopathy Model

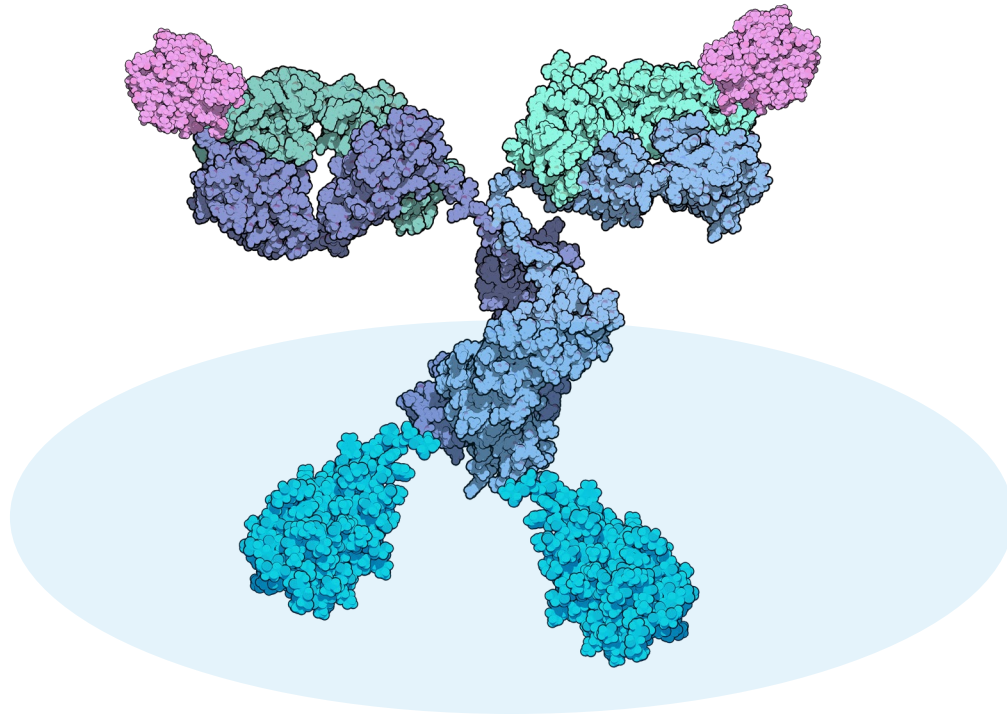


Control

SZN-8141



SZN-8141 VEGF Binding Domains Neutralize Pathologic VEGF-A

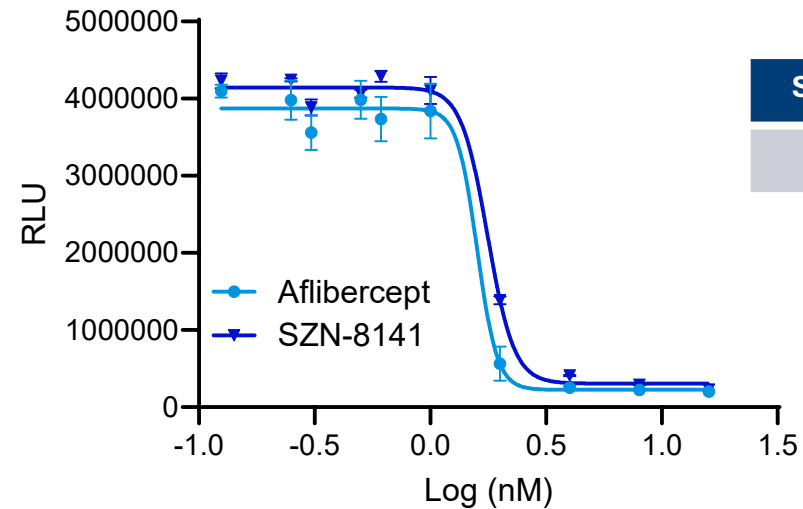


VEGF soluble decoy receptor domains bind soluble VEGF-A with comparable affinity and potency to aflibercept

In vitro assays demonstrate similar potency, affinity and binding to aflibercept

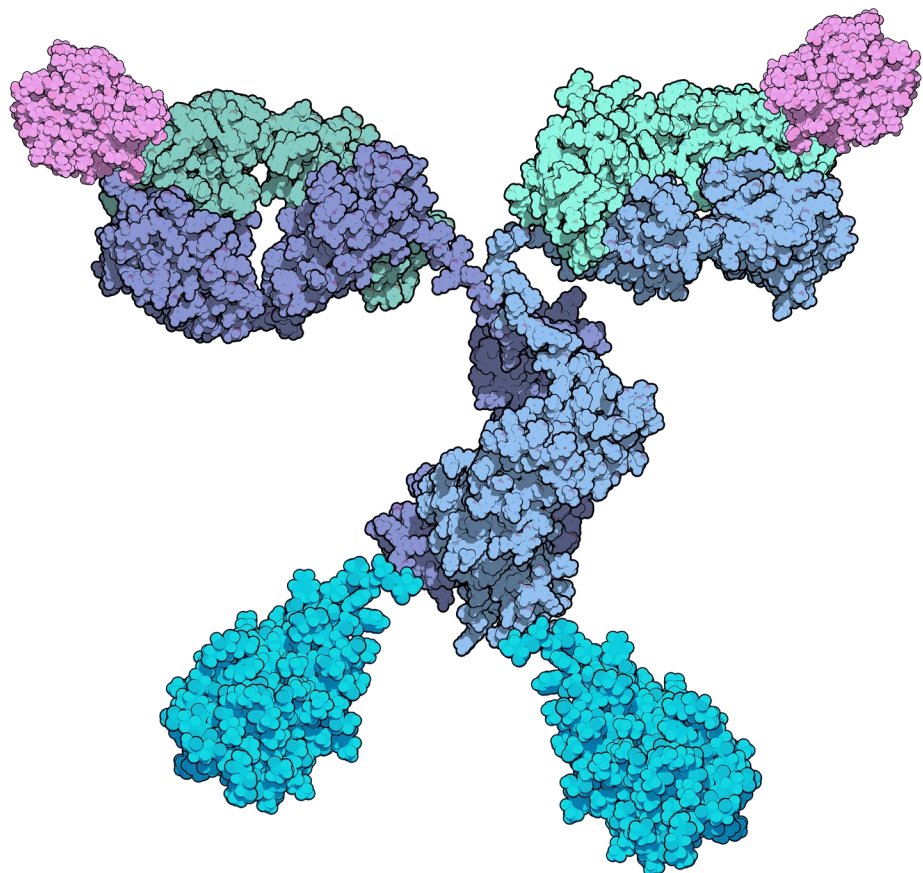
KDR/NFAT-RE HEK293 Cell Potency Assay

Binding Affinity to VEGF (Kd)

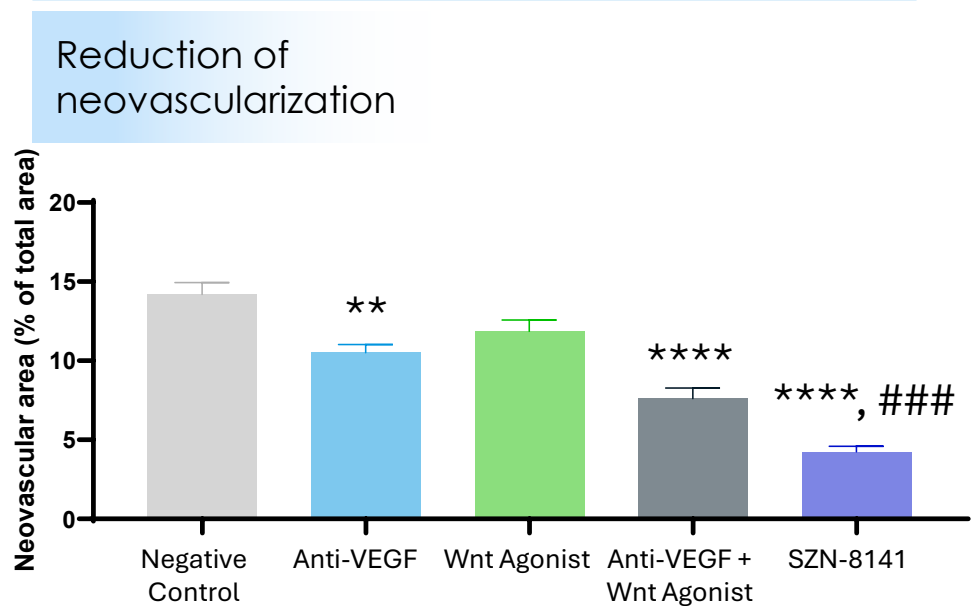
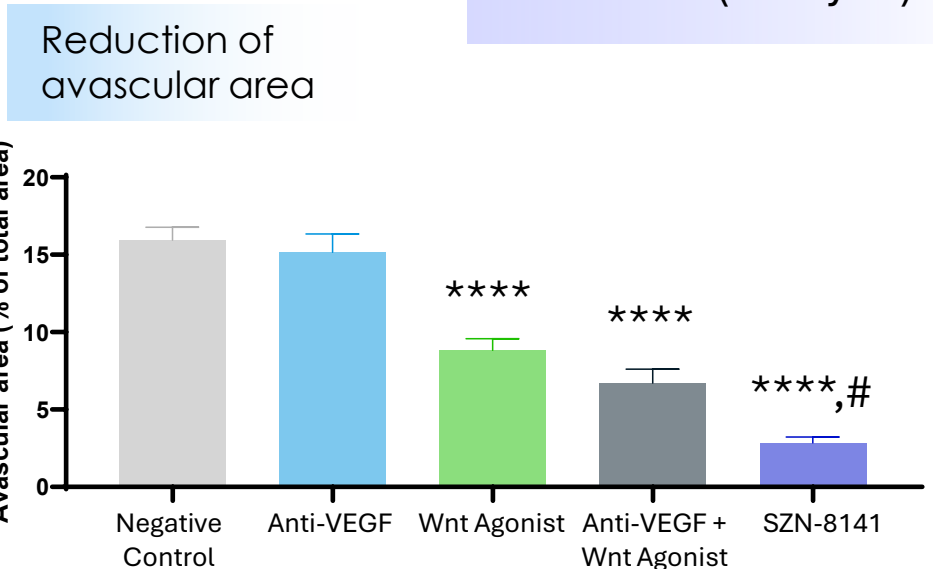


SZN-8141	Aflibercept
<1pM	<1pM

SZN-8141 Synergy Observed with Wnt and VEGF Dual Mechanism



OIR Model (Delayed)

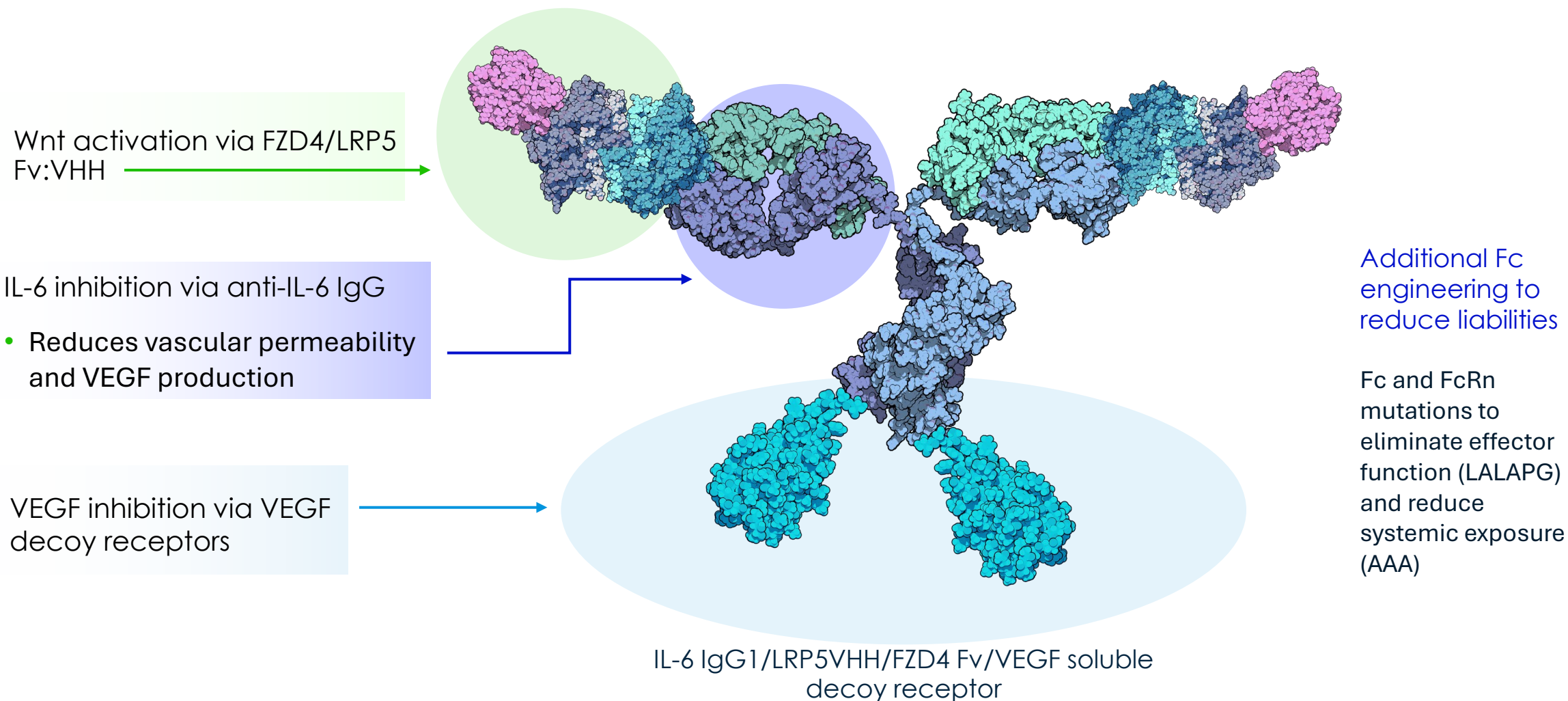


p<0.01, **p<0.0001 v. negative control; #p<0.05, ###p<0.001 v. anti-VEGF + Wnt agonist; n=7 mice for negative control and n=15 mice for active treatment groups. Anti-VEGF is SZN-8141 with mutations to eliminate Wnt activity; Wnt agonist is SZN-8141 with mutations to eliminate anti-VEGF activity.

SZN-8143: Targeting IL-6 Could Provide Additional Clinical Benefit in UME and Other Retinal Vascular Diseases



SZN-8143 – WNT + VEGF + IL-6



Wnt Biology Supports Multiple Therapeutics Opportunities in Ophthalmology Pipeline

Wnt agonists show promising preclinical activity across multiple disease models



Fuchs' Endothelial Dystrophy Program

- Loss of corneal endothelial cells causes corneal swelling, haziness and vision loss, accompanied by ECM deposition (“guttata”)
- Development stage candidate demonstrated preclinical evidence of:
 - Rapid and significant reduction in central corneal thickness
 - Rapid improvement in corneal clarity
 - Stimulates proliferation in human cornea cultures

Geographic Atrophy Program

- Advanced form of macular degeneration that leads to progressive loss of central vision due to the degeneration of retinal cells
- Candidate demonstrated preclinical evidence of:
 - Neuroprotection in acute injury and progressive degeneration models of photoreceptor damage
 - Stimulation of RPE proliferation and differentiation in vitro

Retinitis Pigmentosa Program

- A group of genetic retinal disorders leading to degeneration of photoreceptors
- Candidate demonstrated preclinical evidence of impact on muller glial cells and photoreceptors



Thank You

www.surrozen.com

