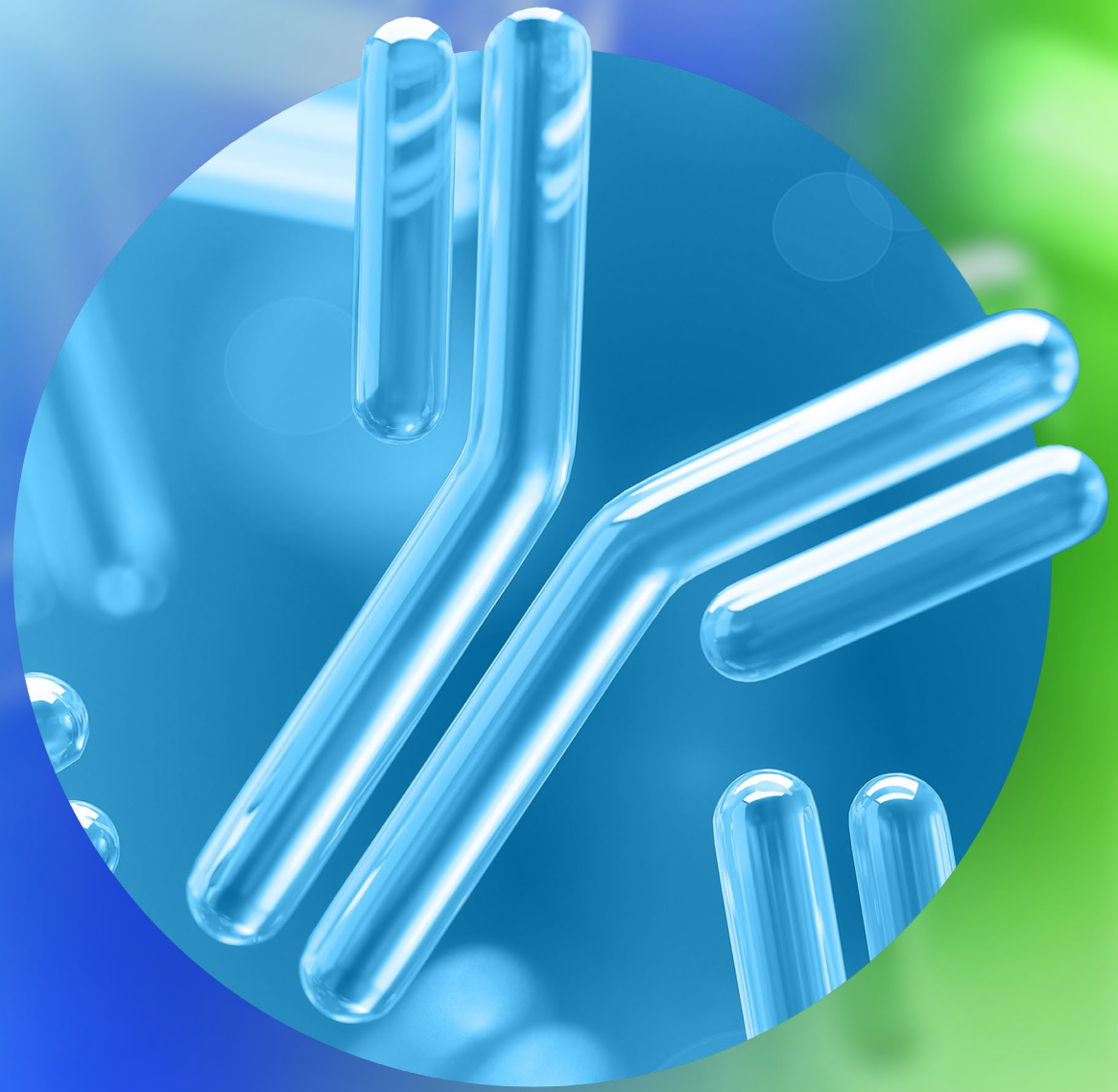
Surrozen

Restoring Vision Through the Science of Renewal



May 2026

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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

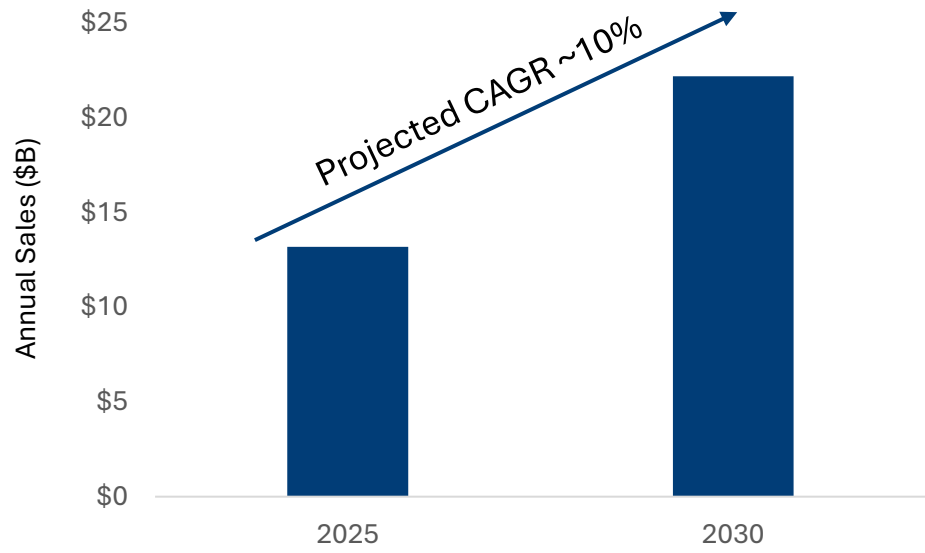
Our Pipeline has Multiple Candidates Designed to Deliver Best Anatomic Outcomes

- 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

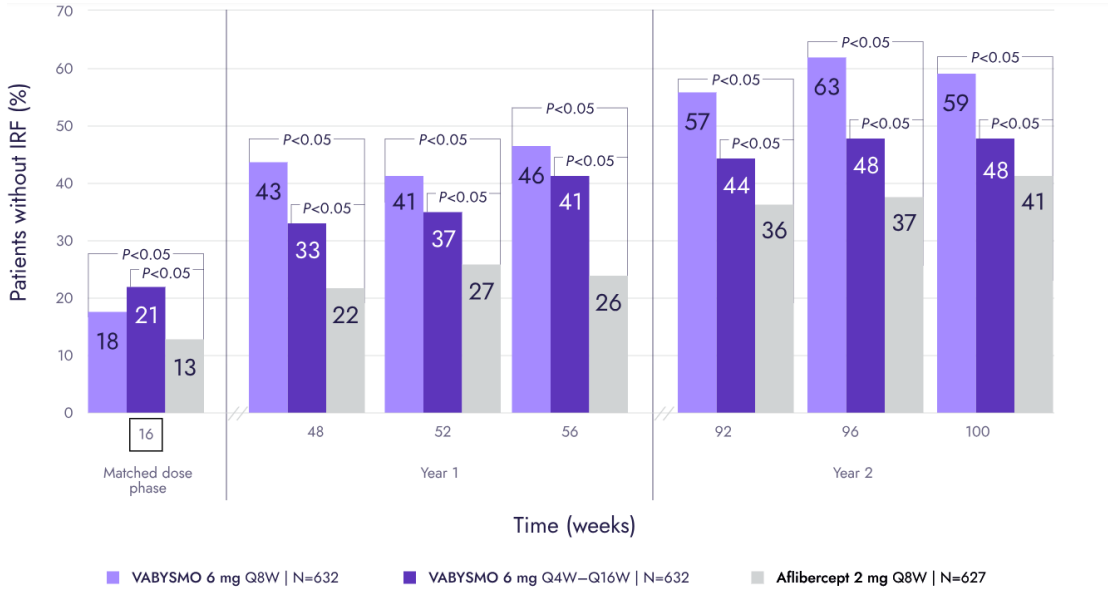


Source: 1. Health Advances DME and wet AMD primary market research for Surrozen - Nov 2024; 2. Im et al, Survey of Ophthalmology, July-Aug 2022; 3. Wong et al, Lancet Global Health Feb 2014; 4. Surrozen estimates from range of research report analyses: Datamonitor – Anti-VEGF wet AMD Grandview Research – 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.

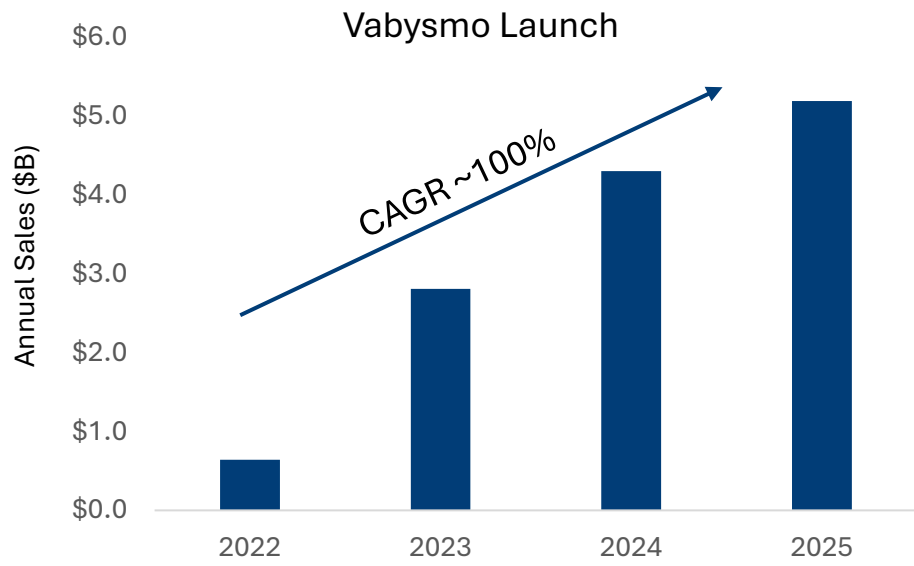
Vabysmo (VEGF + Ang2): Blueprint for VEGF Combinations

Multiple targets drive additional clinical benefit
Incremental drying 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

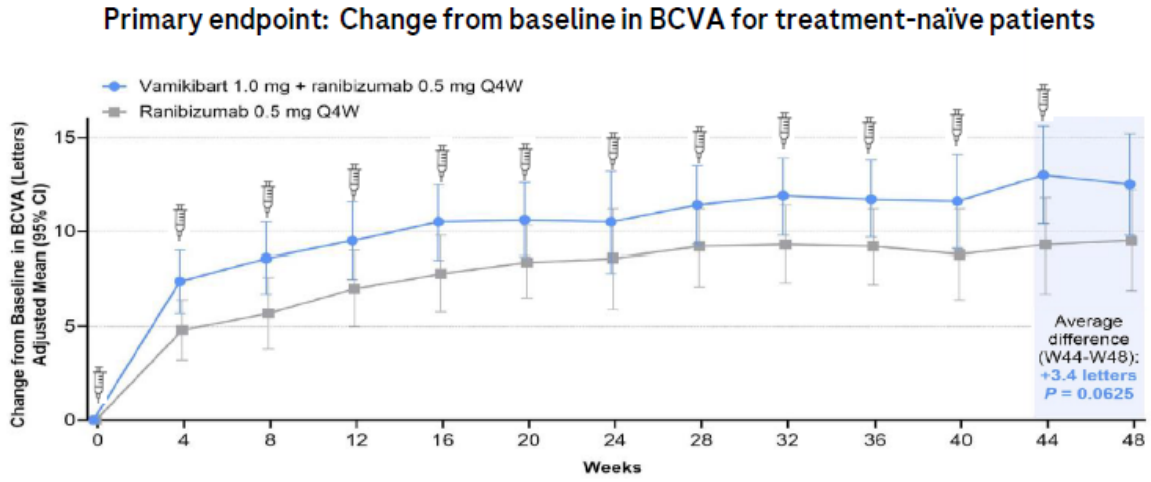
Roche demonstrated IL-6 in combination with VEGF inhibition superior to VEGF alone

Consistent BCVA gains for combination throughout the study

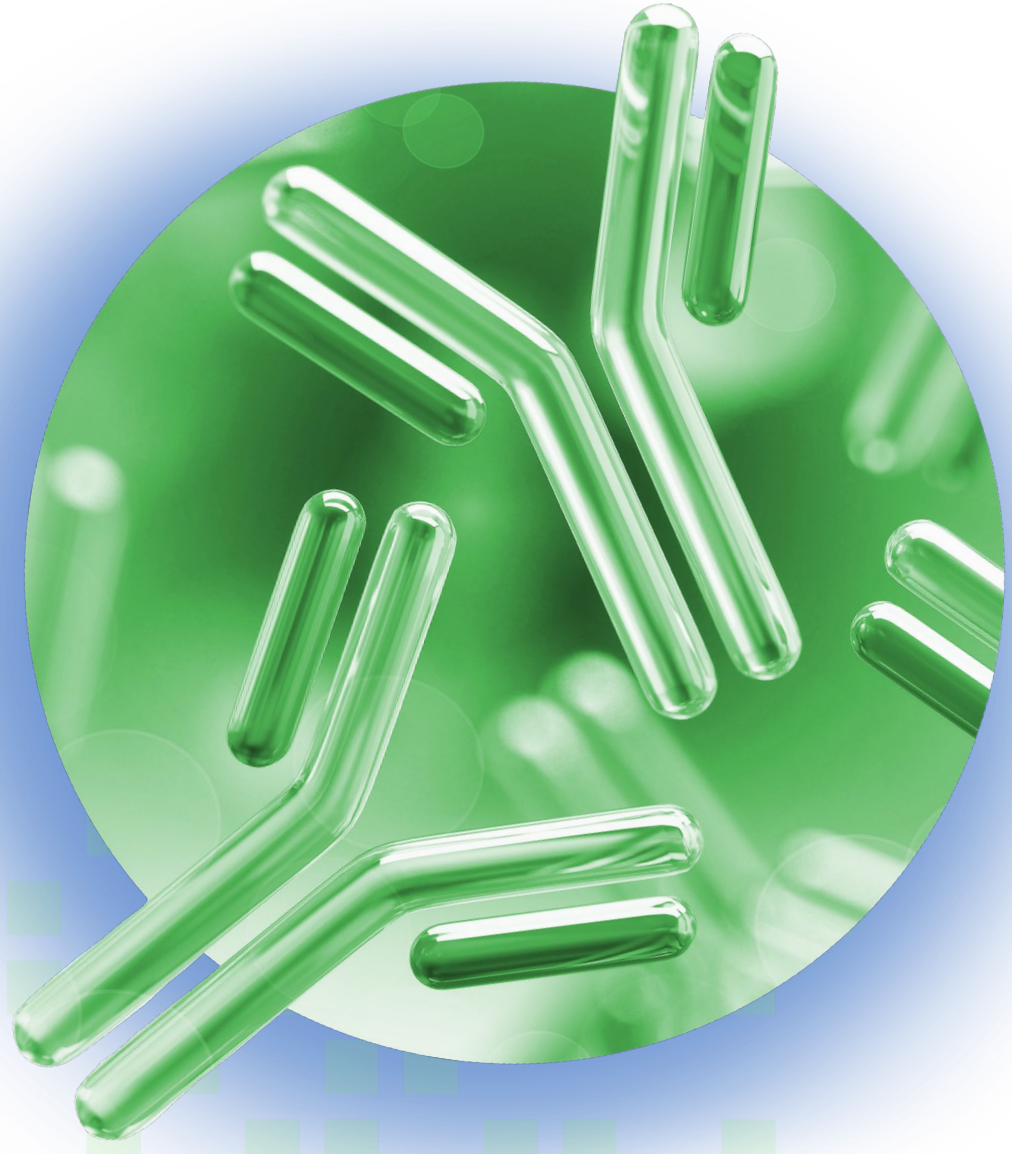
Large increase in percent of patients achieving greatest visual acuity gains: 44.7% of patients receiving combination gained ≥15 BCVA letters vs 28.6% with VEGF alone

Roche pursuing IL6 x VEGF bi-specific

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



Source: Roche ASOPRS/AAO 2025 IR Event Presentation.



Wnt Signaling

A Key Regulator of Vascular Biology

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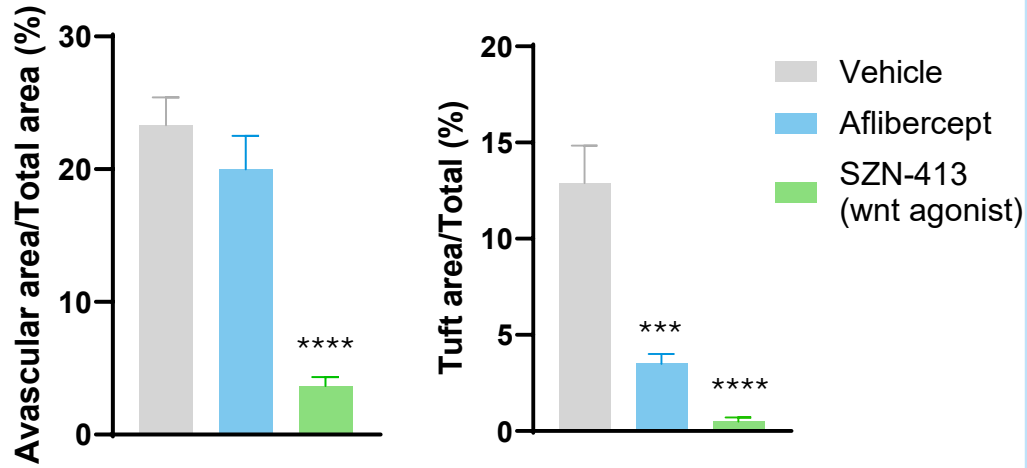
Wnt is A Key Regulator of Retinal Vascular Function

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

Oxygen Induced Retinopathy Model



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%

Source: Data on file; EyeBio Press Release, February 13, 2024.
***p<0.001, ****p<0.0001 v. vehicle. N = 3-4 mice per group.

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 additional next-generation FZD4 targeted antibodies on its own

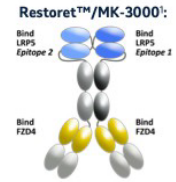
Boehringer Ingelheim

MERCK | 2024

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

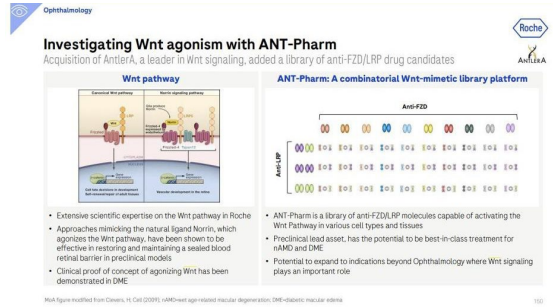
Ophthalmology

- 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**



Source: Merck Press Release, July 12, 2024; Roche Pharma Day 2024 Presentation.

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

Next Generation Multifunctional Biologics:

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

Antibody Design Leadership:

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

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

Surrozen

SZN-413: First FZD4/LRP5 Antibody Targeting Wnt Activation

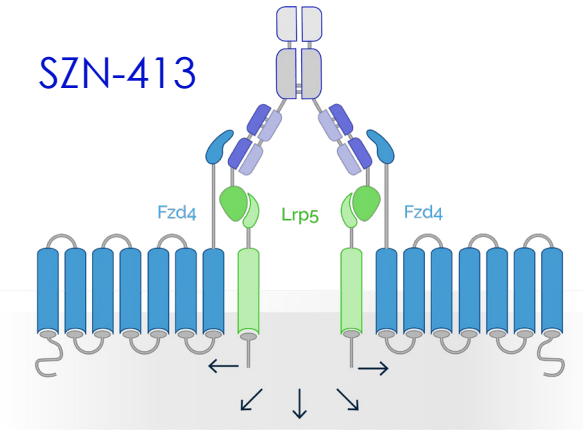
Wnt Pathway:
Distinct Mechanism of Vascular Repair

Novel mechanism for treatment of retinal vascular diseases that can directly reduce leakage and restore blood-retina barrier function

Multiple preclinical models of retinal injury demonstrated that SZN-413 rapidly reduces vascular leakage and avascular areas

Licensed to Boehringer Ingelheim
- $12.5M upfront
- Potential total milestones up to $586.5M
- Mid-single to low double-digit royalties

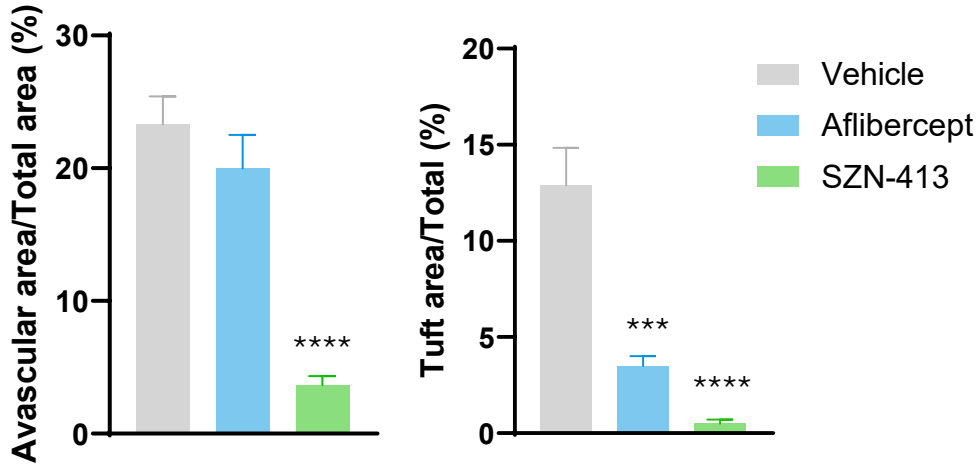
Clustering FZD4 and LRP5 receptors on retinal vascular endothelial cells via a multi-valent, bi-specific antibody activates the Wnt signal transduction cascade. Downstream biological effects in the eye include up-regulation of tight-junction proteins, reduction in vessel leakage and re-establishment of normal vessel architecture



Wnt activation

Oxygen Induced Retinopathy Model

SZN-413 demonstrated superiority of reduction in total avascular area as well as comparable reduction in neovascular tufts compared to aflibercept



***p<0.001, ****p<0.0001 v. vehicle. n = 3-4 mice per group.

Second Generation Multi-Functional Wnt Targeted Antibodies

Complementary mechanisms enable potential best-in-disease antibodies

SZN-8141 – WNT Activation + VEGF Inhibition

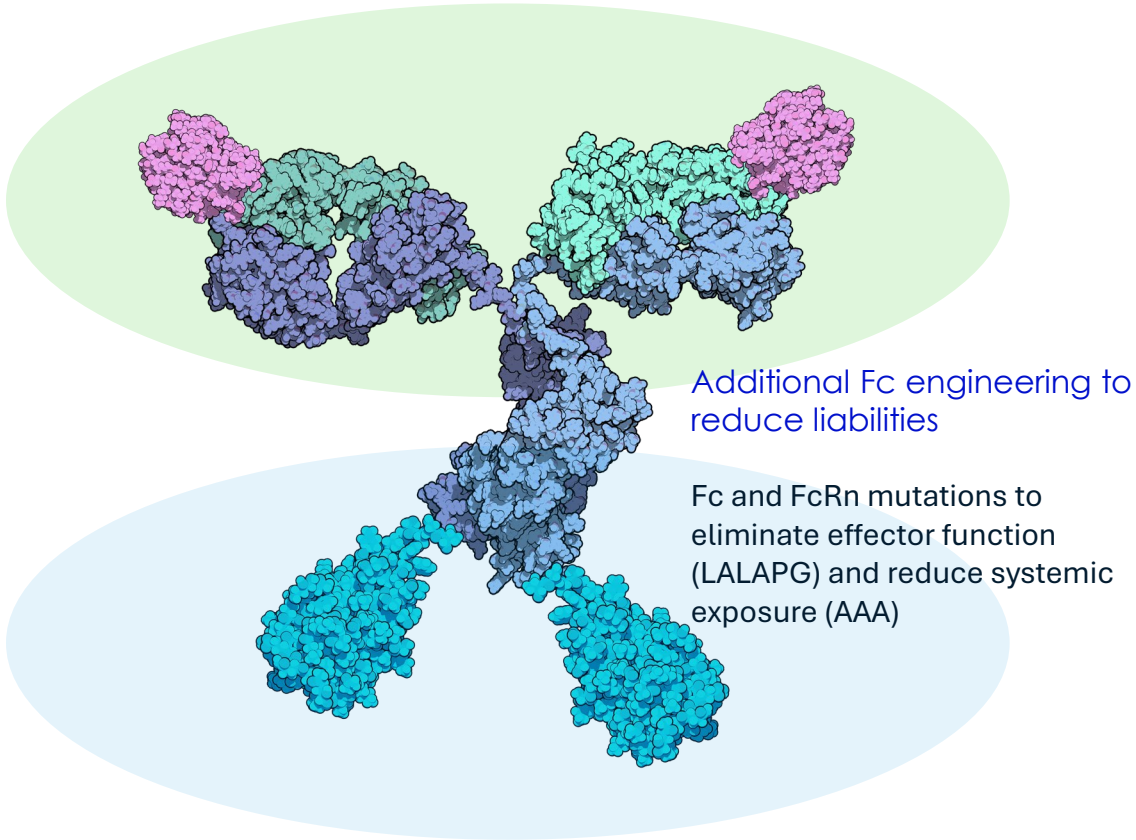
Wnt activation via FZD4/LRP5

- 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

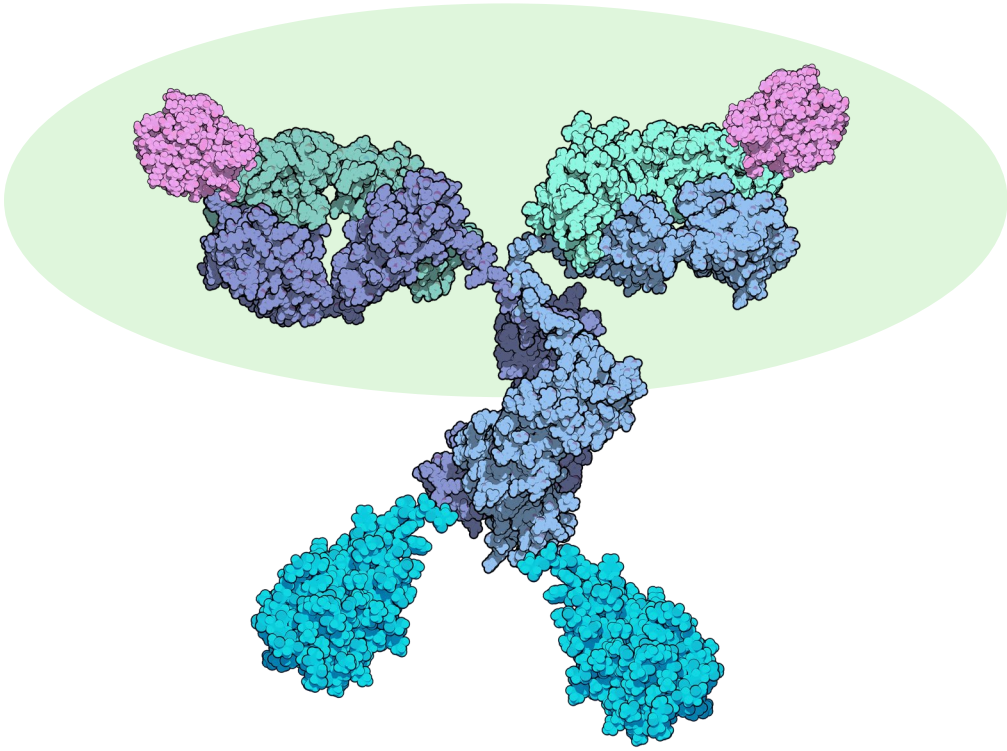
Aflibercept sequences - comparable VEGF-A/PlGF binding affinity

- Reduces neovascularization and vascular leakage



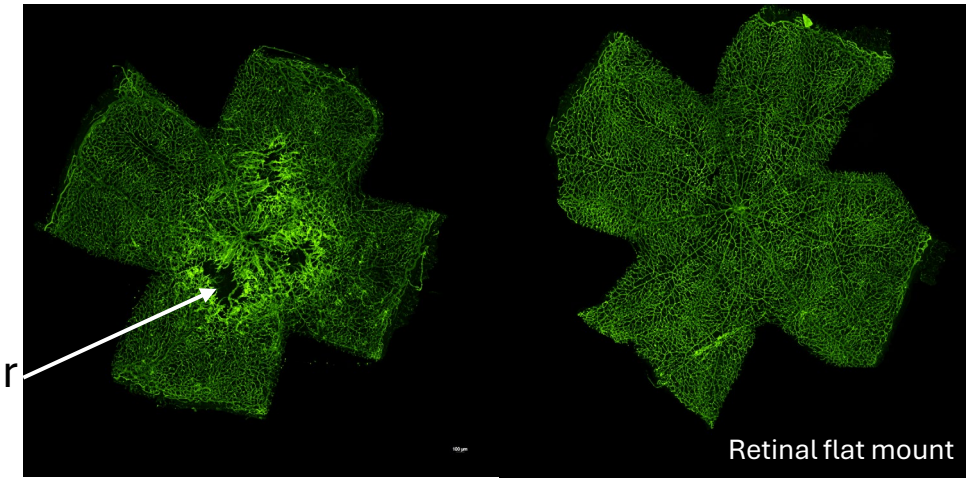
FZD4 IgG1/LRP5VHH/VEGF soluble decoy receptor

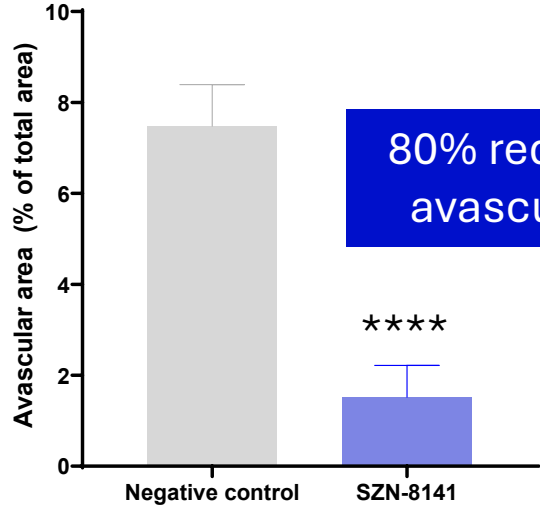
SZN-8141 Restores Retinal Vessel Architecture



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

Oxygen Induced Retinopathy Model

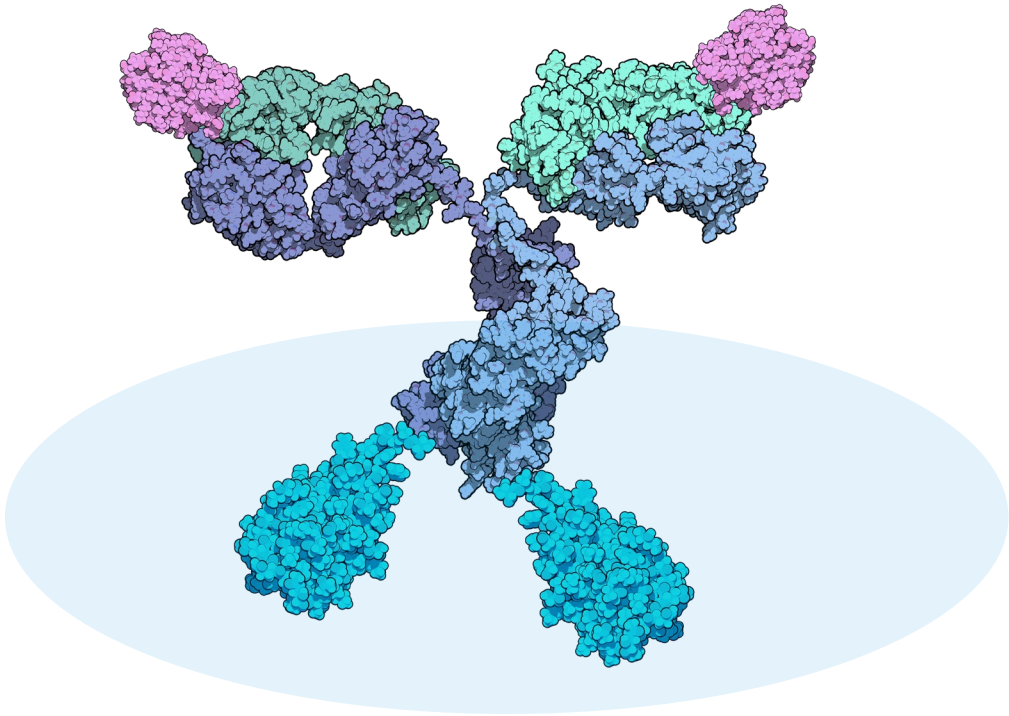




80% reduction in avascular area

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

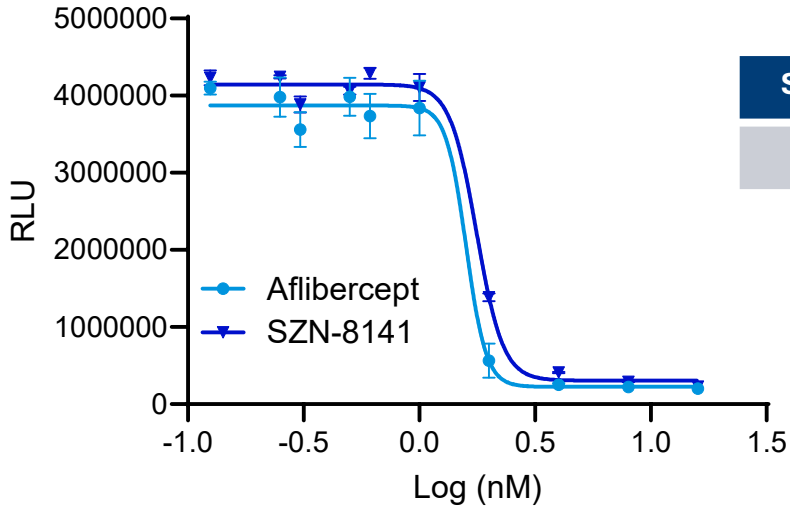
SZN-8141 VEGF Binding Domains Neutralize Pathologic VEGF-A



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

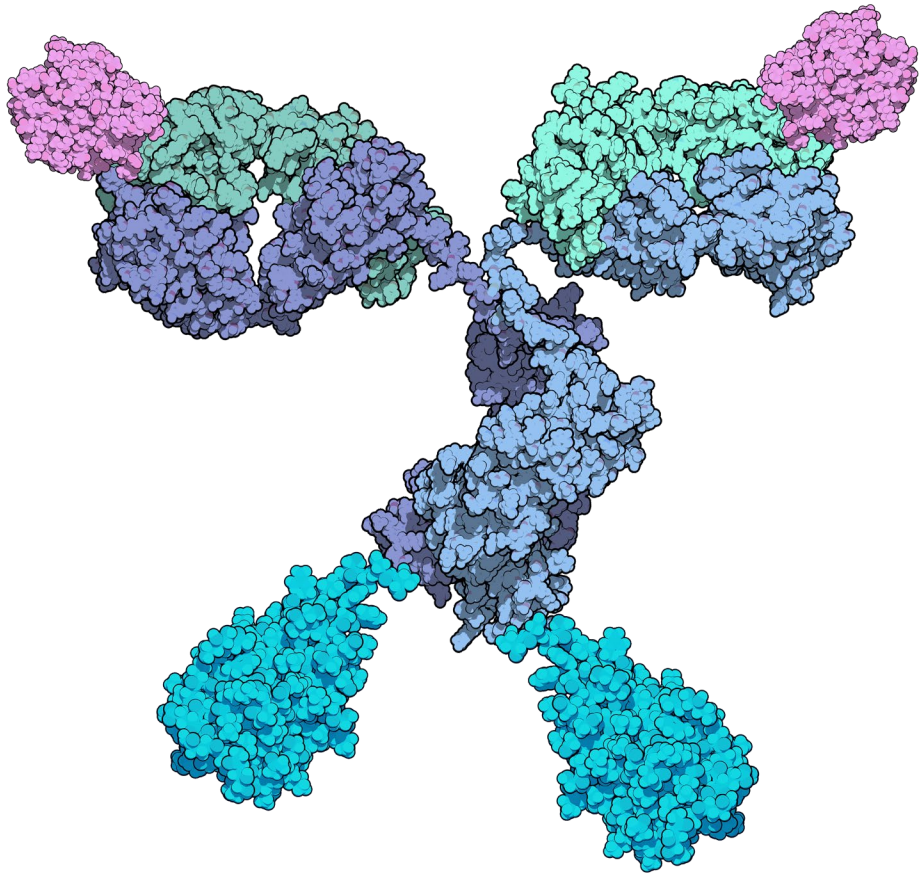
KDR/NFAT-RE HEK293 Cell Potency Assay



Binding Affinity to VEGF (Kd)

SZN-8141	Aflibercept
<1pM	<1pM

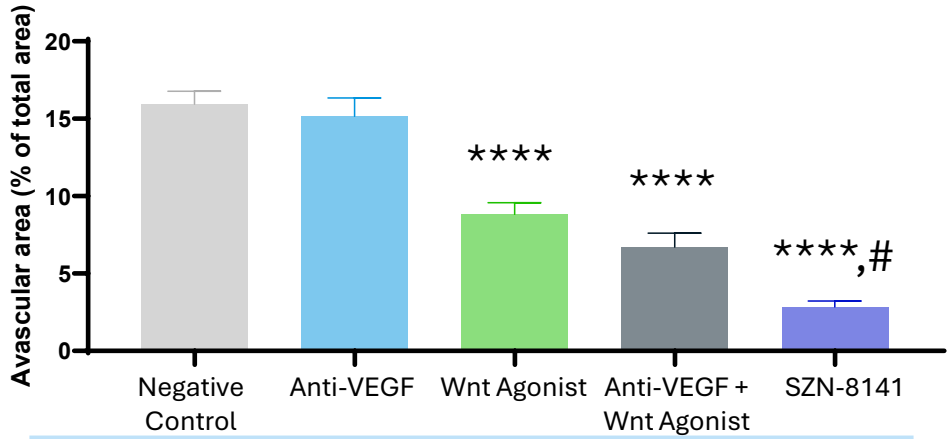
SZN-8141 Synergy Observed with Wnt and VEGF Dual Mechanism



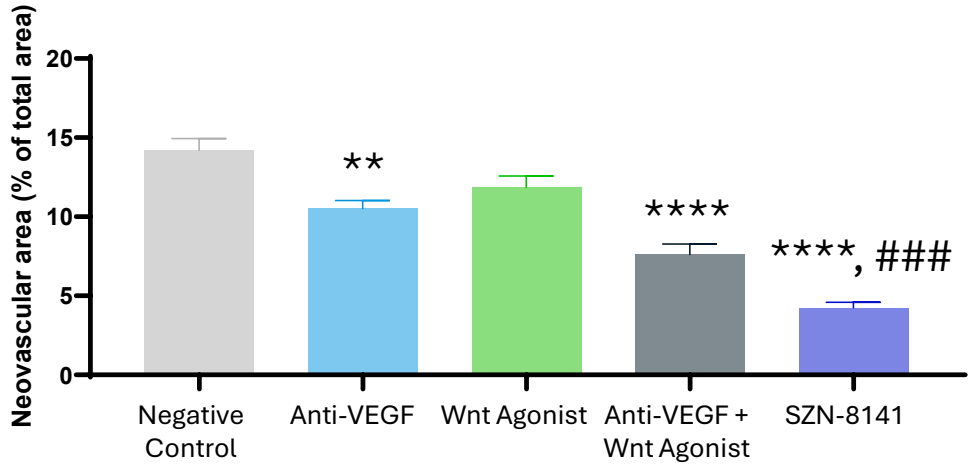
**p<0.01, ****p<0.0001 v. negative control; #p<0.05, ###p<0.001 v. anti-VEGF + FZD4 agonist; n=7 mice for negative control and n=15 mice for active treatment groups. Anti-VEGF is SZN-8141 with mutations to eliminate Wnt activity; Wnt agonist is SZN-8141 with mutations to eliminate anti-VEGF activity.

OIR Model (Delayed)

Reduction of avascular area



Reduction of neovascularization



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

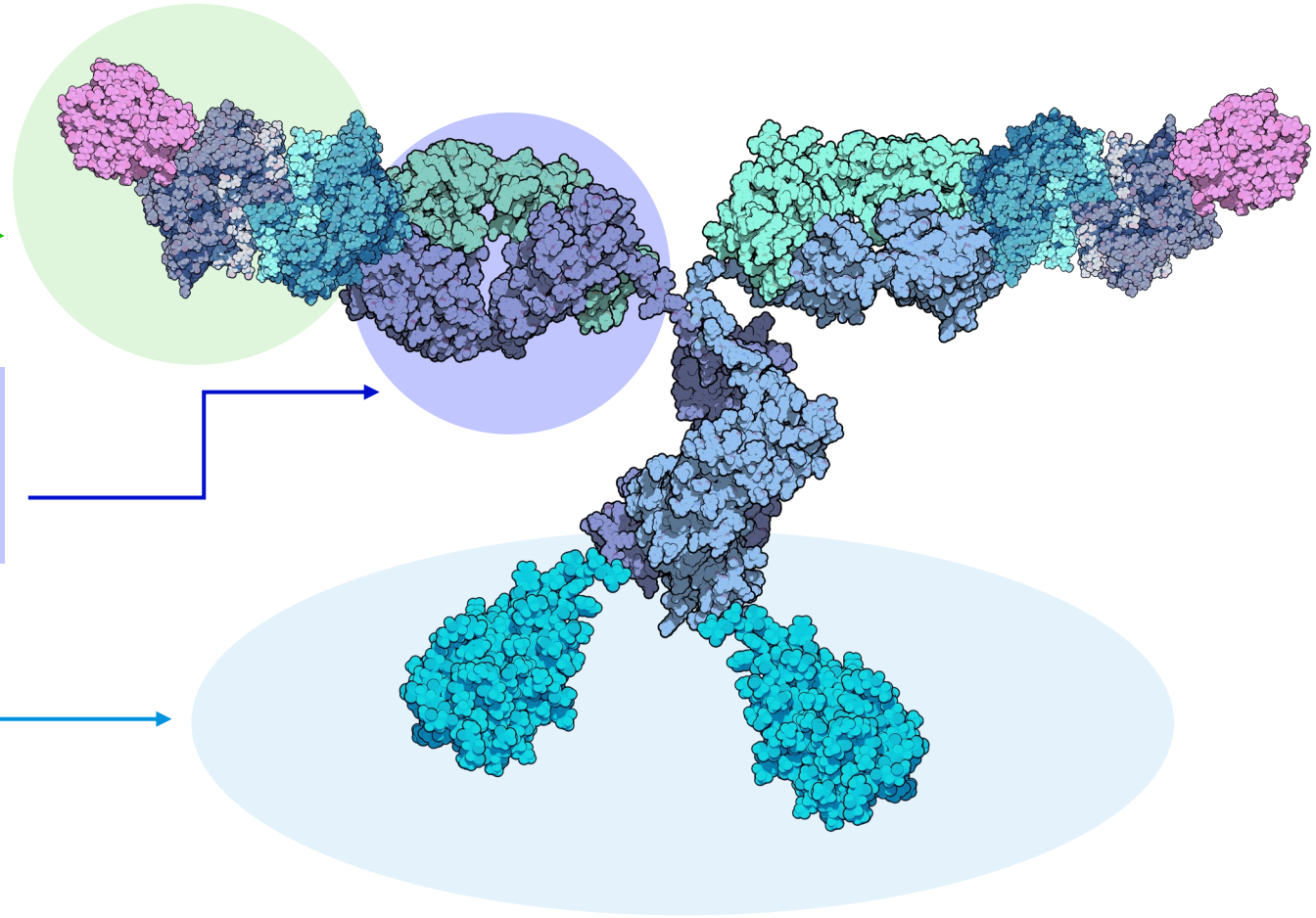
SZN-8143 – WNT + VEGF + IL-6

Wnt activation via FZD4/LRP5 Fv:VHH

IL-6 inhibition via anti-IL-6 IgG

- Reduces vascular permeability and VEGF production

VEGF inhibition via VEGF decoy receptors



IL-6 IgG1/LRP5VHH/FZD4 Fv/VEGF soluble decoy receptor

Additional Fc engineering to reduce liabilities

Fc and FcRn mutations to eliminate effector function (LALAPG) and reduce systemic exposure (AAA)

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

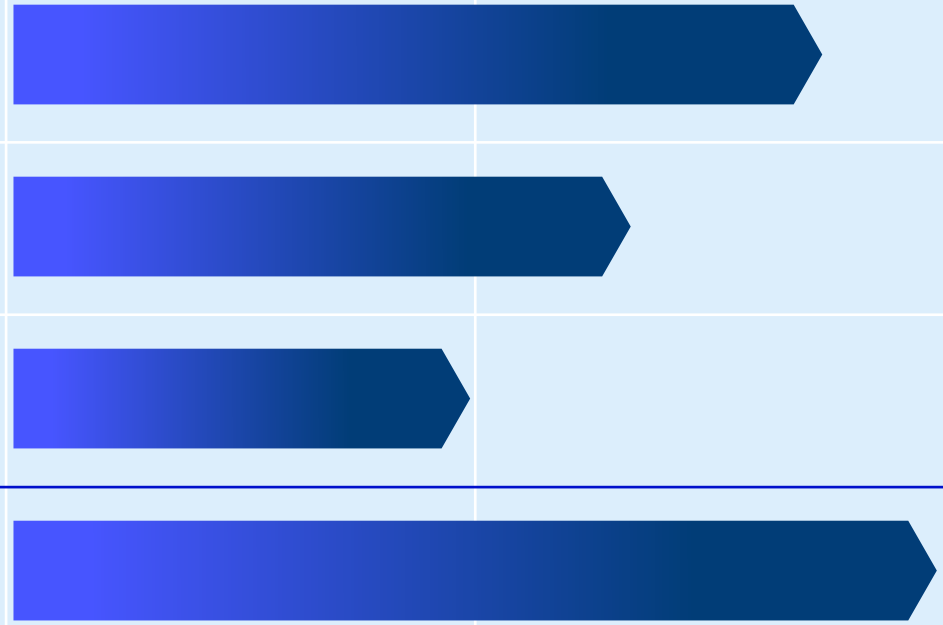
- Advanced form of macular degeneration that leads to progressive loss of central vision due to the degeneration of retinal cells
- Candidate demonstrated preclinical evidence of:
  - Neuroprotection in acute injury and progressive degeneration models of photoreceptor damage
  - Stimulation of RPE proliferation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

Ophthalmology Franchise: Multiple Novel High Value Candidates

PROGRAM	INDICATION	RESEARCH	PRE-CLINICAL	PHASE 1
SZN-8141 **FZD4, VEGF**	wet AMD, DME			
SZN-8143 **FZD4, VEGF, IL-6**	wet AMD, DME, UME			
Research	Ocular disease			
SZN-413* **FZD4**	Retinopathies			



Glossary: FZD - frizzled; VEGF - Vascular Endothelial Growth Factor; IL-6 – interleukin-6; AMD - Age-related Macular Degeneration; DME – Diabetic Macular Edema; UME – Uveitic Macular Edema

*Licensed to Boehringer Ingelheim.

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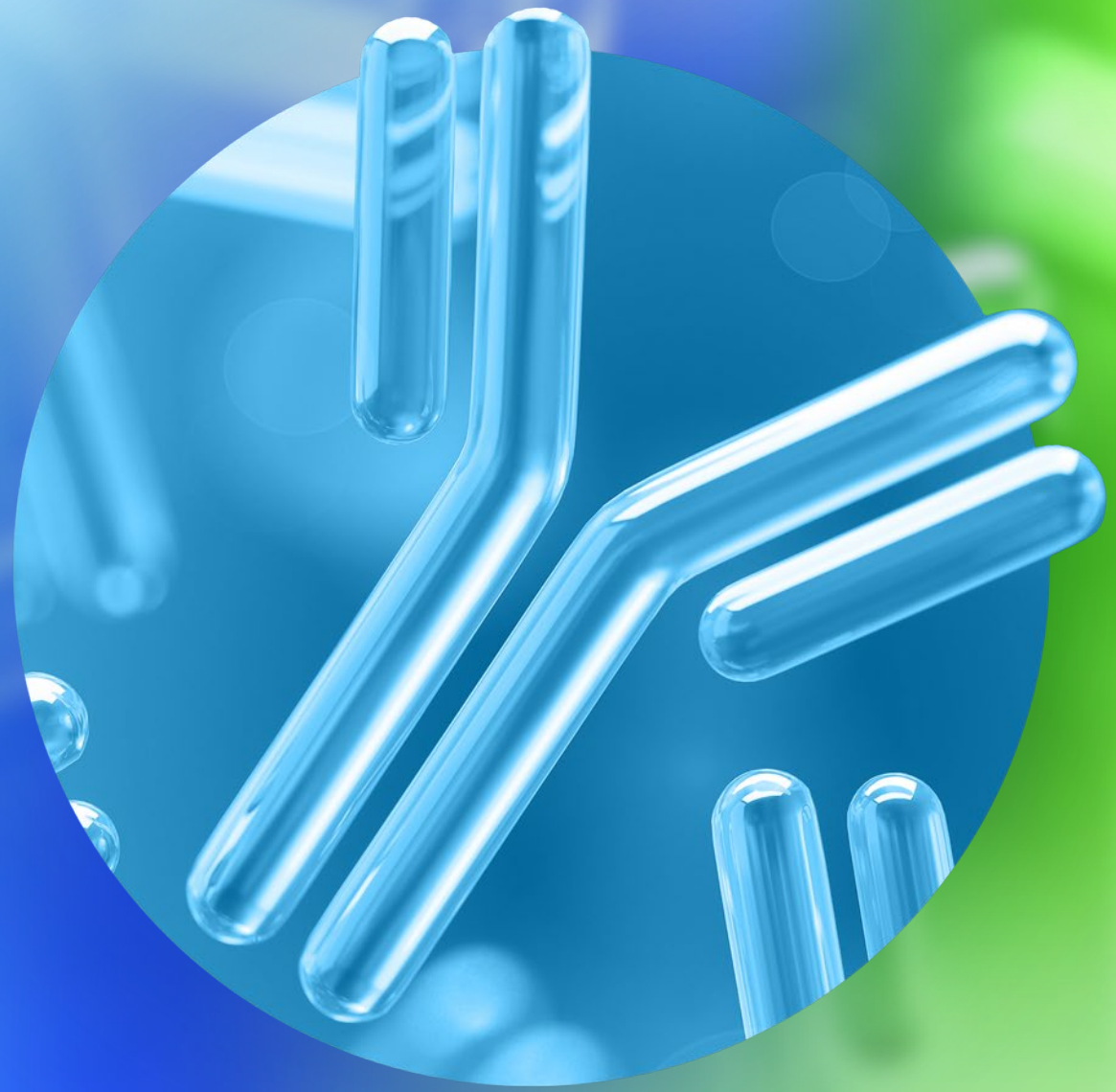

Glossary

AMD	Age-related macular degeneration	**Lrp**	Lipoprotein receptor-related protein
DME	Diabetic macular edema	**POC**	Proof-of-concept
ECM	Extracellular matrix	**OIR**	Oxygen induced retinopathy
FEVR	Familial exudative vitreoretinopathy	**RPE**	Retinal pigment epithelium
FZD	Frizzled	**UME**	Uveitic macular edema
IND	Investigational new drug	**VEGF/R**	Vascular endothelial growth factor/receptors
IL-6	Interleukin 6	**Wnt**	Wingless-related integration site