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Targeted Regeneration

NOVEMBER 2024

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Portfolio Targeting High Value Liver & Ocular Indications

PROGRAM	INDICATION	RESEARCH	PRE-CLINICAL	PHASE 1	PHASE 2
SZN-043 E3 & ASGR1	Severe Alcohol-Associated Hepatitis				
SZN-413 FZD4	Retinopathies			Boehringer Ingelheim	
SZN-8141 FZD4, VEGF	wet AMD, DME				
SZN-8143 FZD4, VEGF, IL-6	wet AMD, DME, UME				
SZN-113 FZD127	Fuchs' Endothelial Corneal Dystrophy (FECD)				
	Geographic Atrophy (GA)				

Leader in Antibody Engineering to Modulate Wnt Signaling in a Range of Tissues and Diseases

- **Innovator** in modulating the Wnt pathway for tissue regeneration
  - Novel and clinically validated treatment strategy for large markets with high unmet need
  - Significant strategic interest in Wnt signaling approach: Merck, Roche, BI
- Therapeutic relevance of Wnt signal activation supported by **clinical POC** in DME
- Multiple candidates entering or in Phase 1 clinical trials
- **Phase 1b trial** nearing completion for SZN-043 in severe alcohol-associated hepatitis
  - Safety, PK and PD established across a range of doses in cirrhotics and sAH patients
  - **Phase 1b efficacy data** anticipated in 1H 2025
- Proprietary antibody platforms with **robust patent estate** including FZD4/LRP antibodies
- Advancing potential best-in-class FZD4/LRP targeted SWAP to the clinic with partner **Boehringer Ingelheim** for retinal diseases
- Pipeline of multiple **ophthalmology development candidates** targeting highly prevalent retinal and corneal diseases

SZN-043

Hepatocyte-Targeted R-spondin Mimetic (SWEETS) Severe Alcohol-Associated Hepatitis

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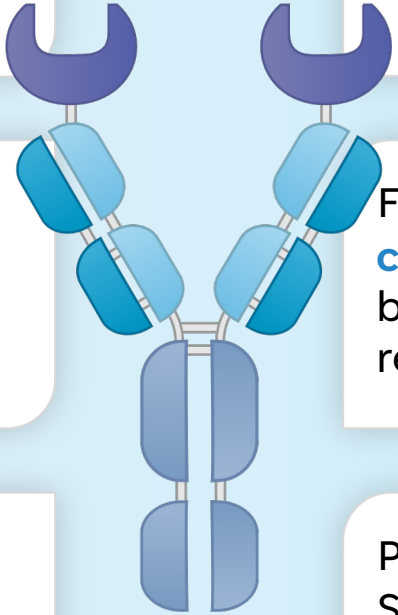
SZN-043 Program Summary

Antibody Targeted to Liver that Stimulates Hepatocyte Proliferation and Improves Liver Function

sAH is a severe liver disease with increasing incidence, **no approved therapies** and significant short-term mortality

Phase 1b study initiated in Q2 2024 in severe alcohol-associated hepatitis (sAH)

SZN-043 is a precision targeted bi-specific antibody that engages mature hepatocytes, enhances Wnt signaling and may improve liver function



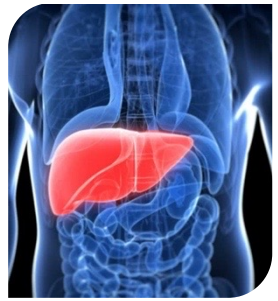
First cohort complete: observed a **potential clinical benefit** based on reductions in bilirubin and MELD score and no drug related SAEs

Phase 1a study demonstrated target engagement, activation of Wnt signaling, and effects on liver function in patients with a history of liver cirrhosis

Proliferative and functional effects of SZN-043 **directly address pathology** of alcohol-associated hepatitis –rapid hepatocyte loss leading to high mortality rate

SZN-043 Has The Potential to Transform Patient Outcomes in SAH

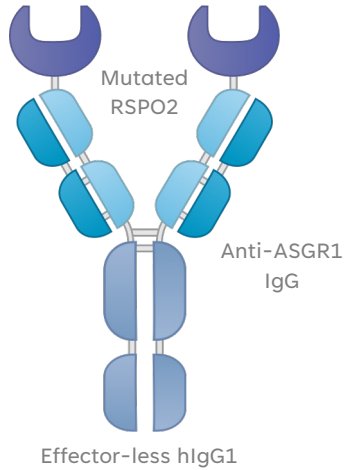
Disease Severity and Opportunity



- 130,000 patients in the U.S. hospitalized with SAH[1]
- 90-day mortality 30% in high MELD score patients[2]
- No approved drugs for SAH – steroid used in minority but no effect on mortality at 90 days[1]
- Potential for rapid development and regulatory path[1]
- Intermediate endpoints like MELD score strongly correlated with survival[3]

Targeted Wnt Signaling Approach

- MOA: SZN-043 designed to address underlying pathophysiology
- Hepatocyte proliferation & Wnt signaling correlated with improved survival
- Upregulation of Wnt signaling implicated in improved liver function
- Selectivity achieved through inclusion of ASGR1 binder



Sources: 1. Analysis by Clearview Health Partners for Surrozen; HCUP National Inpatient Sample (NIS); Physician Market Research
2. Hughes et al (2018). PLoSONE13(2):e0192393
3. Mehta H, Dunn W (2022). J Clin and Exp Hepatology

SZN-043 Phase 1 Clinical Trial Summary

Safety & PK (n=42)

- No drug related adverse events reported in patients with cirrhosis at any dose
- No drug related SAEs to date in sAH patients
- PK consistent with expectations and supportive of the planned doses, schedule and route of administration for sAH

Effects on liver function, PD Activity & Target Engagement

- Demonstrated dose dependent pharmacodynamic (PD) activity through activation of Wnt signaling as assessed by methacetin breath test*
- Target engagement was confirmed via transient increases in alkaline phosphatase (ALP)**
- Effects on liver physiology as measured by HepQuant***

Ongoing enrollment in sAH at escalating doses

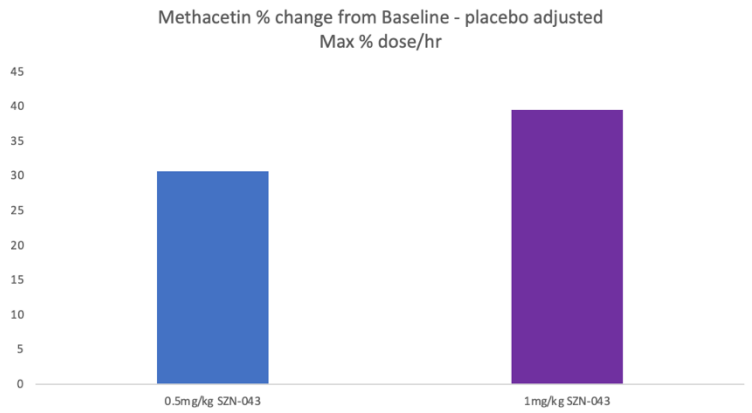
- Completed 0.5mg/kg cohort 1 with no drug related SAEs and no AST/ALT elevations
- Dosing days 0, 4 with 90 day follow-up
- There were no deaths at day 30 of the study
- Company observed a potential clinical benefit based on reductions in bilirubin and MELD score

*Methacetin breath test measures activation of the Wnt pathway via the metabolism of a Wnt target gene (CYP1A2) substrate
**Increases in ALP are indicative of SZN-043 binding to its targeting receptor ASGR1 and reduction in its capacity to clear ALP, consistent with observations in other ASGR1 binding agents
***HepQuant is a test that measures cholate clearance, a liver specific function that quantifies liver function

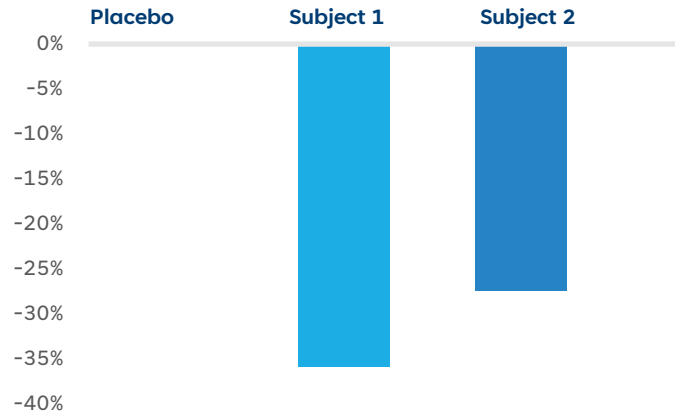
SZN-043 Demonstrated a Clear Pharmacodynamic Effect

Wnt Signal Activation, Improved Portal Hepatic Filtration Rate and Improved Disease Severity Index

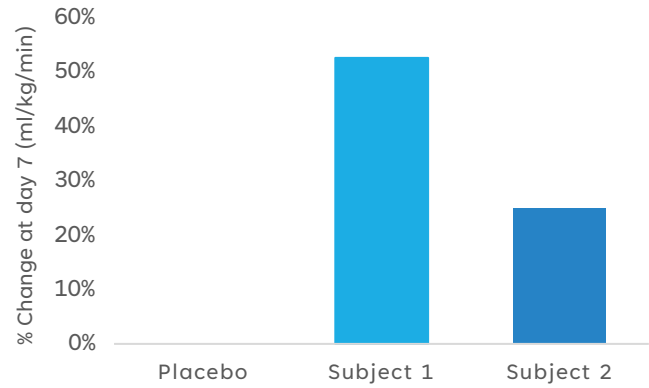
Methacetin Breath Test



Disease Severity Index



Portal Hepatic Filtration Rate



- MBT has been validated as a safe and effective tool for evaluating functional hepatic mass
- MBT measures metabolism of methacetin by Wnt pathway gene (CYP2A1)
- CYP2A1 is elevated by Wnt signal activation
- SZN-043 demonstrated dose dependent activation of Wnt Pathway In cirrhotics

- HepQuant has been validated as a method to measure liver physiologic changes including cell and circulatory function in the liver
- HepQuant measures cholate clearance, a liver specific function that quantifies liver function and perfusion
- SZN-043 demonstrated improved portal hepatic filtration rate and a reduction in the disease severity index

1) Mcrae et al. Translational Research (2023)

SZN-043 Clinical Development: Next Steps

Phase 1 Study Features and Timeline

- 9 sites in 5 countries
- Dose escalation in 1b from 0.5 to 1.0 and 1.5mg/kg x 2 (n=24 total)
- Assess PK, PD, safety and potential efficacy signals
- Robust external comparator database of sAH patients (n=265) established with academic collaborator
- Expect POC data in 1H'25

Phase 2/3 Design

- Seek regulatory feedback on clinical trial design elements and expected filing requirements
- Based on precedent guidance, expect primary endpoint to be 90-day survival
- Potential for study to enroll < 300 patients to demonstrate ~ 50% survival improvement
- Potential for Breakthrough Therapy Designation or Fast Track

Other Development Milestones

- CMC regulatory requirements complete (Lonza manufacturing) and GMP batches for Phase 2/3 underway
- Toxicology studies complete in 2 species with no adversities

Surrozen Ophthalmology Franchise

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Ophthalmology Franchise Executive Summary

Wnt signaling is implicated in multiple diseases and tissues in the eye

Surrozen has considerable discovery capabilities in ophthalmology

- Antibody discovery and pre-clinical models established for corneal and retinal diseases

Surrozen ophthalmology discovery franchise has already generated one development candidate

- SZN-413 licensed to Boehringer Ingelheim in October 2022
- Potential best-in-class FZD4/LRP bi-specific antibody for retinal diseases like neovascular AMD and DME
- Narrowly defined license enables Surrozen to pursue additional FZD4 targeted antibodies on its own

Surrozen has multiple novel ophthalmology development candidates

- SZN-8141 and 8143 for wet AMD, DME and UME | FZD4/VEGF combination (one molecule) and FZD4/VEGF/IL-6 combination (one molecule)
- SZN-113 for Geographic Atrophy (FZD127)
- SZN-113 for Fuchs' Endothelial Cell Dystrophy (FZD127)

Potential for INDs in 2025 and 2026

Strategic Interest in Targeting Wnt Pathway for Therapeutics

Roche acquisition of AntlerA in 2024: Wnt mimetic library

Ophthalmology

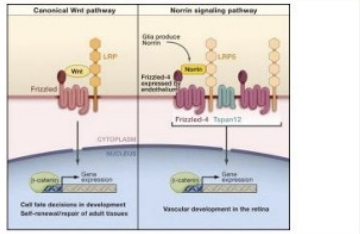
Roche

ANTLERA

Investigating Wnt agonism with ANT-Pharm

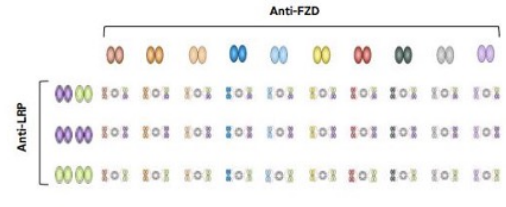
Acquisition of AntlerA, a leader in Wnt signaling, added a library of anti-FZD/LRP drug candidates

Wnt pathway



- 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: A combinatorial Wnt-mimetic library platform



- 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 nAMD and DME
- Potential to expand to indications beyond Ophthalmology where Wnt signaling plays an important role

MoA figure modified from Clevers, H; Cell (2009); nAMD=wet age-related macular degeneration; DME=diabetic macular edema

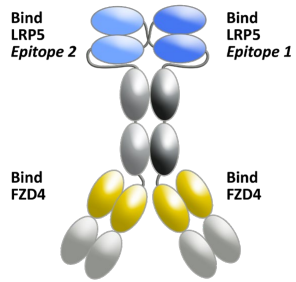
150

Merck acquisition of EyeBio in 2024 for $1.2BN: FZD4/LRP5 targeting for DME and nvAMD

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**

Restoret™/MK-3000[1]:



MERCK 25

SZN-413 Program

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SZN-413 Program Summary Antibody Targeted to FZD4

Novel mechanism for treatment of retinopathies that can directly reduce leakage and potentially reduce VEGF production

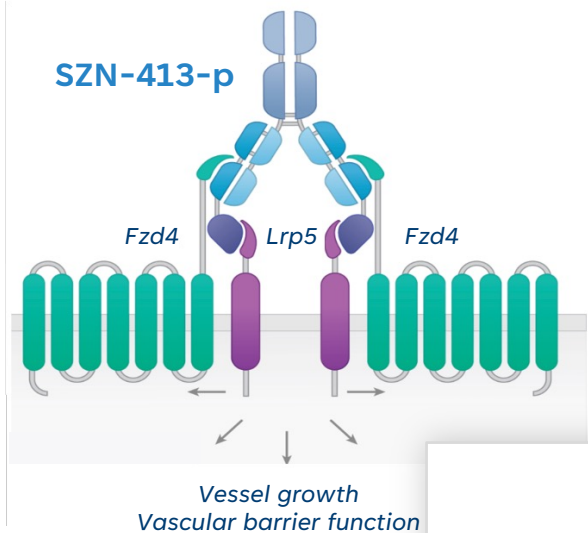
FZD4/Norrin signaling in the eye is known to mediate proper function of retinal vascular endothelial cells

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

SZN-413 was licensed to Boehringer-Ingelheim (BI) under an October 2022 collaboration and licensing agreement

- Surrozen received $12.5M upfront; potential milestones of up to $586.5M; mid-single to low double-digit royalties
- $10M milestone payment received in 2024 for Start of Development (SOD)
- $22.5M received to date
- Potential for additional near-term milestones through IND

SZN-413: Potential for Full Reversal of Patient's Retinopathy



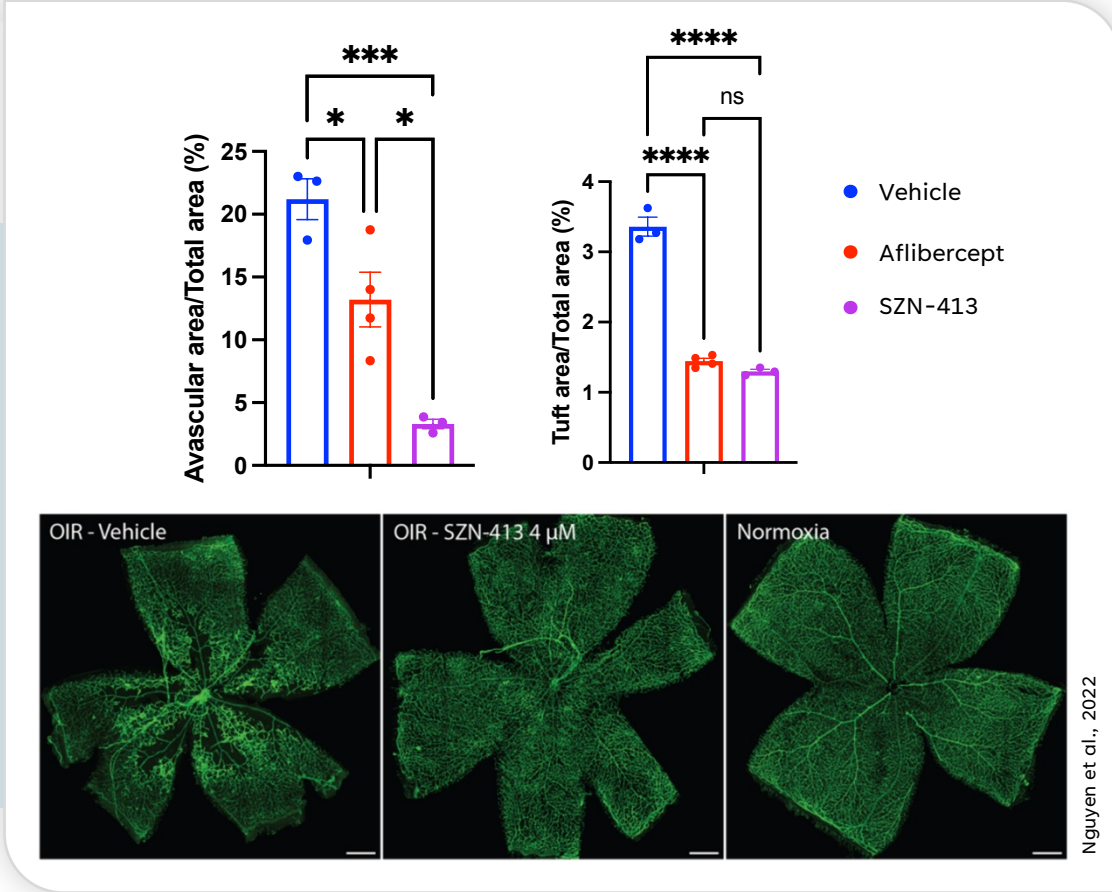
Retinal Vascular Program

SZN-413 addresses retinal non-perfusion and vascular leakage simultaneously

FZD4/Norrin signaling plays critical role in maintenance of retinal vasculature integrity

SZN-413 (FZD4 SWAP Wnt Mimetic):

- Stimulated Wnt signaling Increased tight junction protein expression in endothelial cells
- Restored norrin function in Ndp KO mice
- Reduced avascular area & pathologic NV tuft formation in OIR model
- Reduced vascular leakage in VEGF-induced retinal model



Surrozen FZD4/LRP/VEGF/IL-6 Programs

Targeting Multiple Pathways for Retinal Vascular Diseases

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Executive Summary

Recent interest in FZD4 agonists and IL6 antagonists:

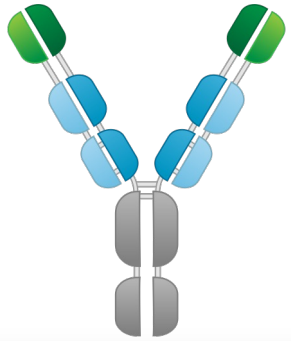
- Clinical POC for FZD4 in DME, Eyebio AMARONE, Feb 2024
- Eyebio acquisition by Merck, May 2024
- AntlerA acquisition by Roche, July 2024
- Roche/Genentech anti-IL6 antibody Vamikibart in Ph3 (UME)/Ph2 (DME)
- Kodiak Eylea/anti-IL6 combo KSI-501 in Ph3 (wAMD), KSI-101 in Ph1b (UME)

Surrozen's next generation FZD4 agonists program:

- Potential best in class efficacy through combination with anti-VEGF and/or anti-IL6

FZD4/Multi-Target Strategy

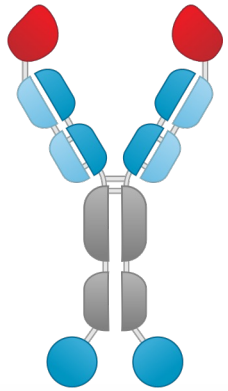
Multiple Potential Candidates



FZD4

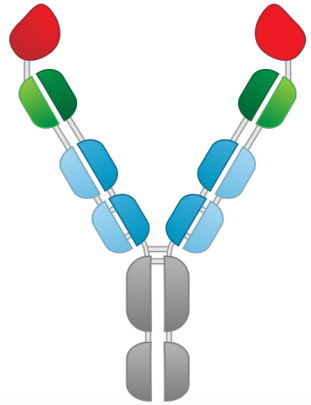
Generate new FZD4 agonists:

- New formats
- New binders to FZD4 and/or LRP



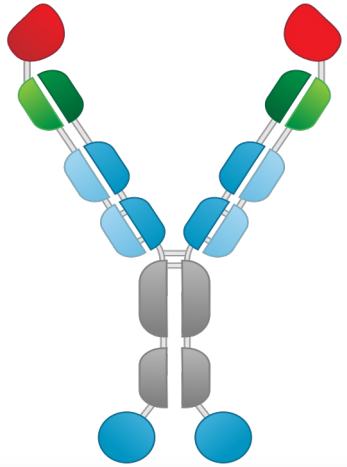
FZD4-αVEGF

Combination of FZD4 agonism and VEGF/VEGFR antagonism may have benefits over single agent alone for DME/ wet AMD treatment



FZD4-αIL6

IL6 is one of the major contributors to retinal vascular inflammation. Combination of the two may have benefits in DME/wet AMD, UME

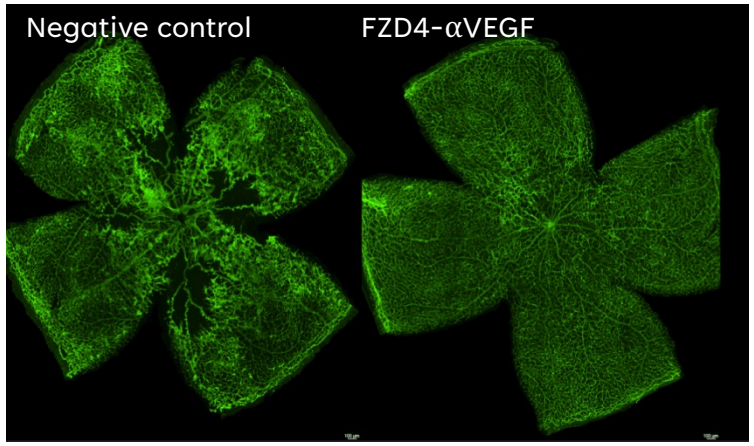


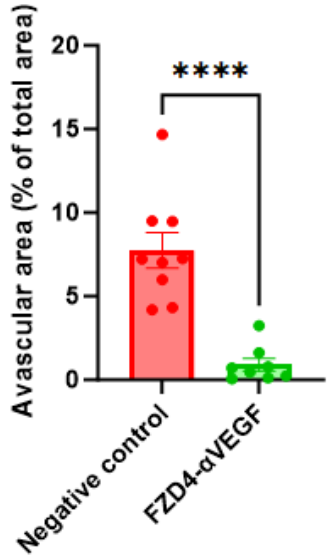
FZD4-αVEGF-αIL6

Combining all three in one molecule could become the best-in-class treatment for multiple type of retinopathies

FZD4-αVEGF is Efficacious in Mouse Models

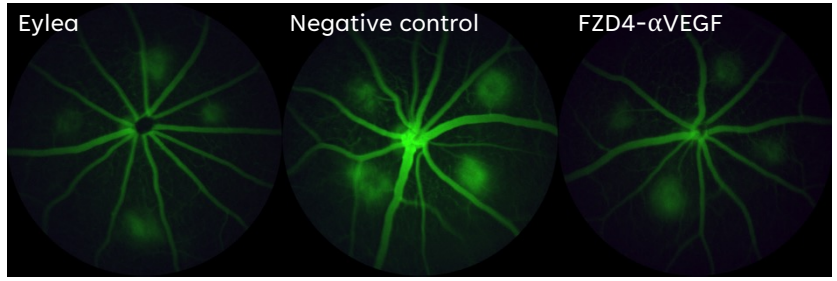
Oxygen-Induced Retinopathy (OIR)



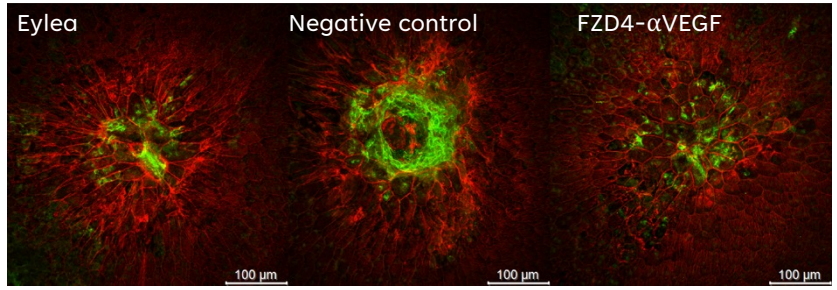


Laser-Induced Choroidal Neovascularization (CNV)

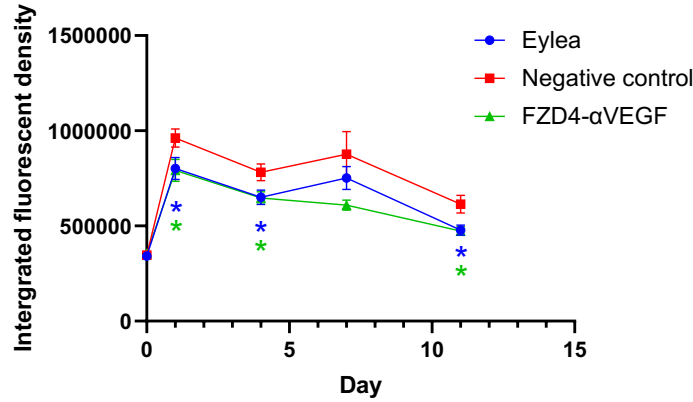
Fundus fluorescein angiography at day 11



CNV lesions at day 11



Vascular leakage by fundus fluorescein angiography



Cornea Endothelium Program

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Cornea Program: Wnt Signal Activation/Corneal Regeneration

Fuchs' endothelial cell dystrophy (FECD) is a disease characterized by corneal swelling and ultimately vision loss caused by progressive loss of corneal endothelial cells

Current therapies limited to endothelial transplant or resection at late-stage of the disease

Unmet need for therapies that mitigate disease progression and/or improve surgical outcomes

Wnt activation could regenerate corneal endothelial cells potentially reducing corneal swelling and improving vision

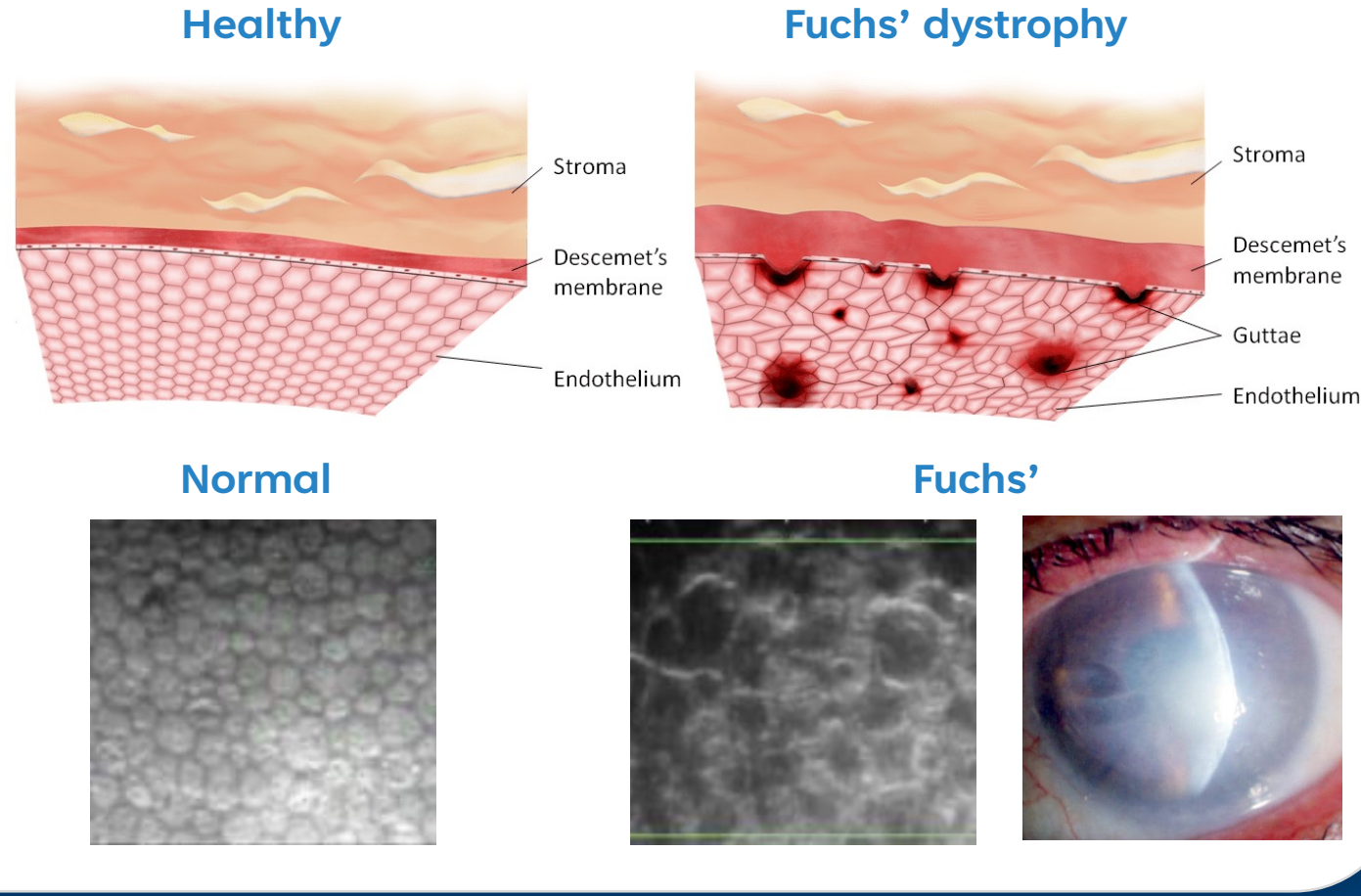
Surrozen has already shown:

- Wnt activation with Surrozen SWAP molecules enhances proliferation of primary human corneal endothelial cells in vitro
- Evidence of wound healing in mouse and rabbit acute corneal endothelial injury models showing improved corneal clarity
- Lead molecule – SZN-113 – exhibits excellent pharmaceutical properties
- **Market research identifies large unmet need,** commercial opportunity and clear path to approval in FECD

Fuchs' Endothelial Cell Dystrophy

Loss of Endothelial Cells Causes Cornea Swelling and Vision Loss

- Ion pumps in corneal endothelium maintain corneal thickness and clarity
- Loss of corneal endothelial cells causes corneal swelling, haziness and vision loss, accompanied by ECM deposition ("guttata")
- High Prevalence:
  - 2.9M diagnosed patients with moderate-to-severe FECD
  - Treatment option includes surgery
  - Market research: Strong opportunity for WNT approach to avoid or delay surgery



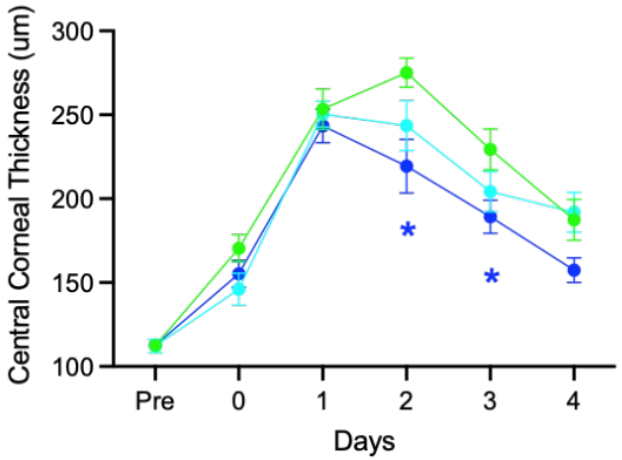
Cell loss and ECM deposits cause corneal swelling, haziness, and vision loss

Photo References: Newsomeye; Research Gate; eMedicine

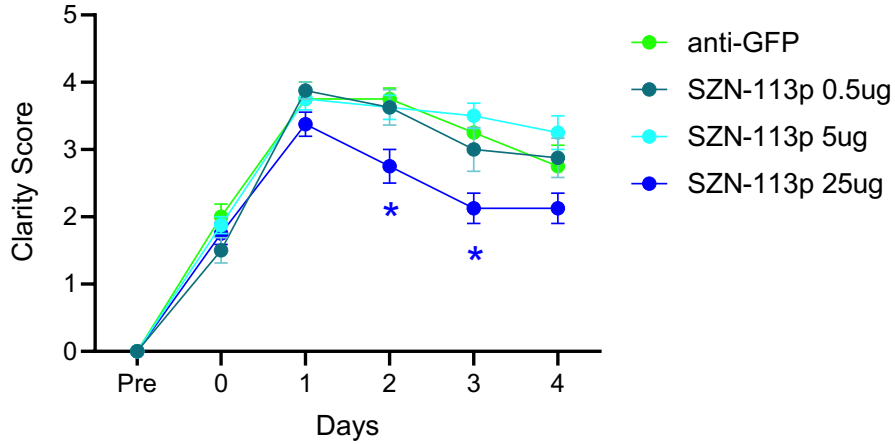
SZN-113 is Efficacious in Mouse and Rabbit Cryoinjury Models

SZN-113 Reduced Corneal Thickness and Improved Clarity Score

Central Corneal Thickness

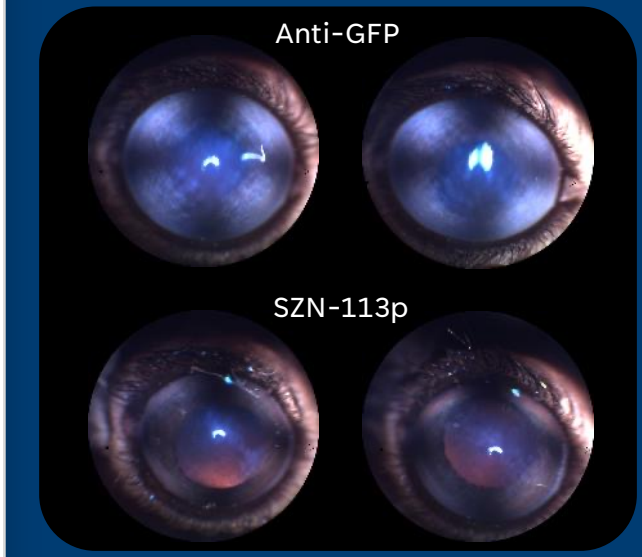


SPOTS Clarity Score



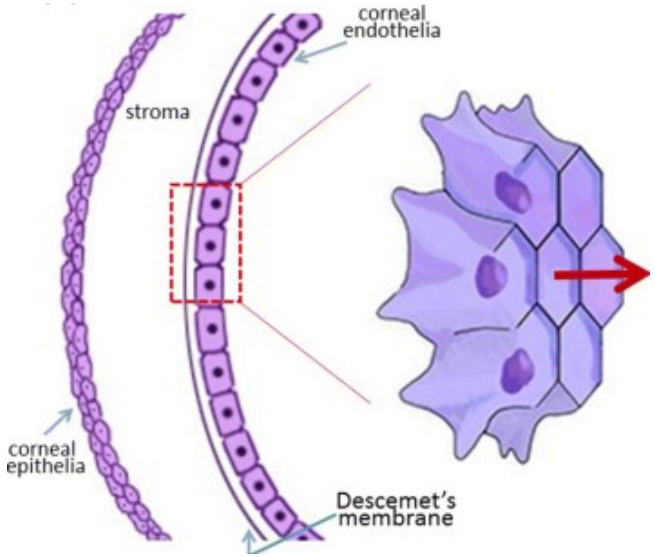
- Trans-corneal injury induces endothelial cell loss and corneal edema (thickening)
- SZN-113 rapidly and significantly reduced central corneal thickness
- SZN-113 rapidly induced improvement in corneal clarity (scoring by cornea specialist (SPOTs grading scale) blinded to treatment group)

Day 3



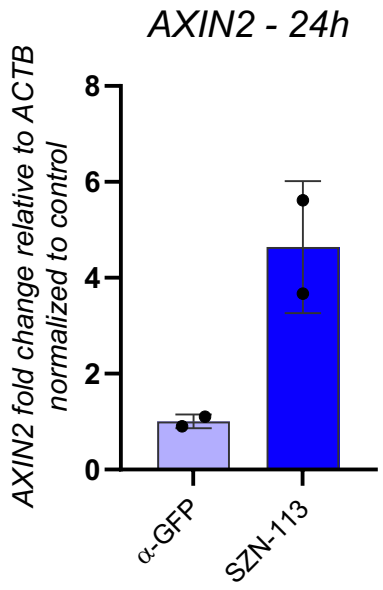
Improved corneal clarity 3 days after injury and treatment

SZN-113 Stimulates Proliferation in Human Cornea Cultures

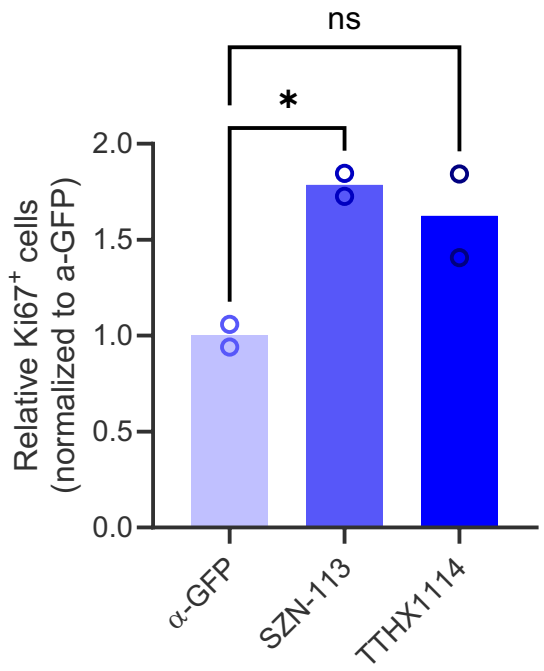


Human endothelial “skirts” can be removed and cultured

Wnt activation



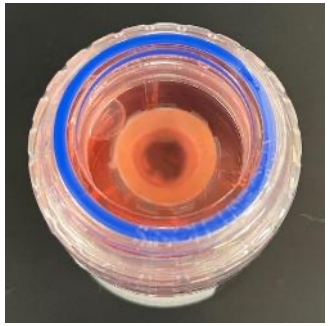
Proliferation

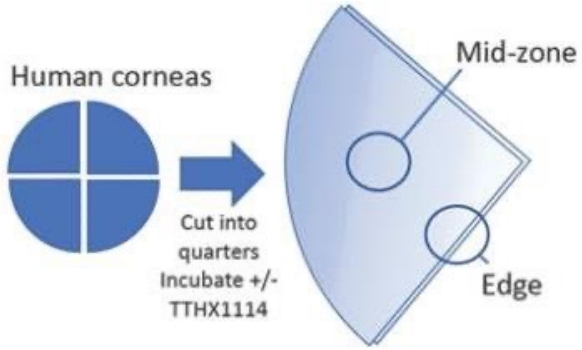


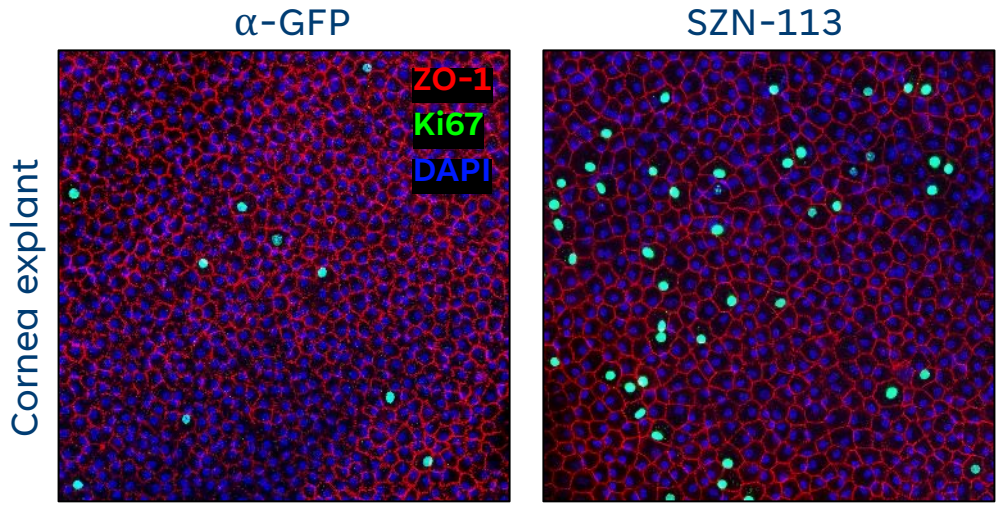
SZN platforms and competitors' comparison

- SZN-113 activates Wnt pathway
- SZN-113 as good or better than engineered FGF

SZN-113 Stimulates Proliferation in Human Cornea Cultures

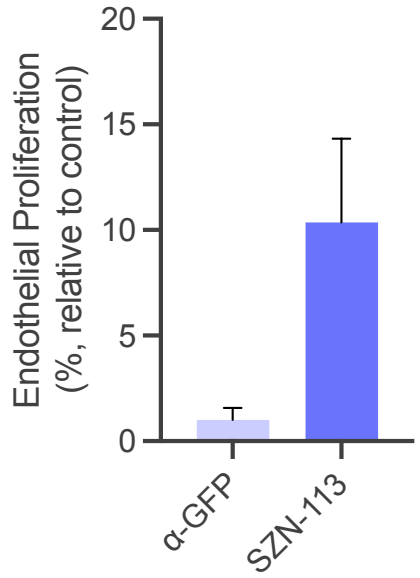




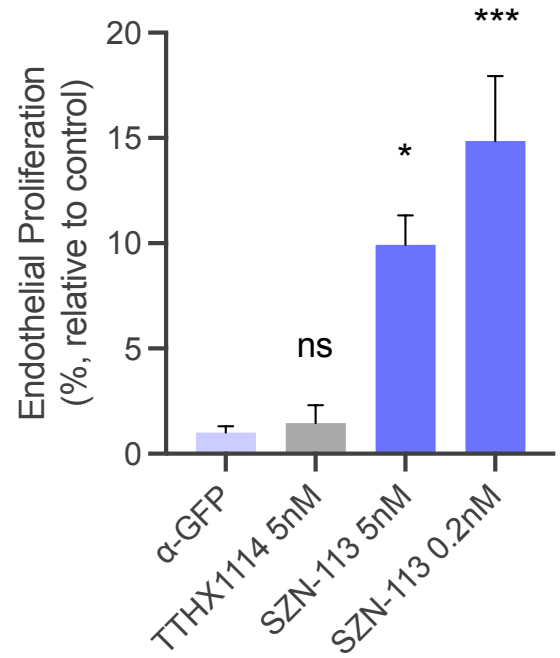


Human corneas (healthy and Fuchs') from eye bank can be incubated and treated *in vitro* for several days

Healthy donor cornea



Disease donor cornea



SZN-113 induces ~10-fold increase in proliferation in healthy human and FECD disease tissue

Dry AMD and Geographic Atrophy

SURROZEN

Dry AMD: Potential Wnt Signaling Impact on Multiple Effected Cells

Dry age-related macular degeneration (AMD) is characterized by progressive blurred central vision caused by thinning of the macula; a key cause is degeneration of the retinal pigment epithelium (RPE)

High unmet need: large prevalence, current treatments only moderately delay disease progression

Surrozen's local-delivery approach:

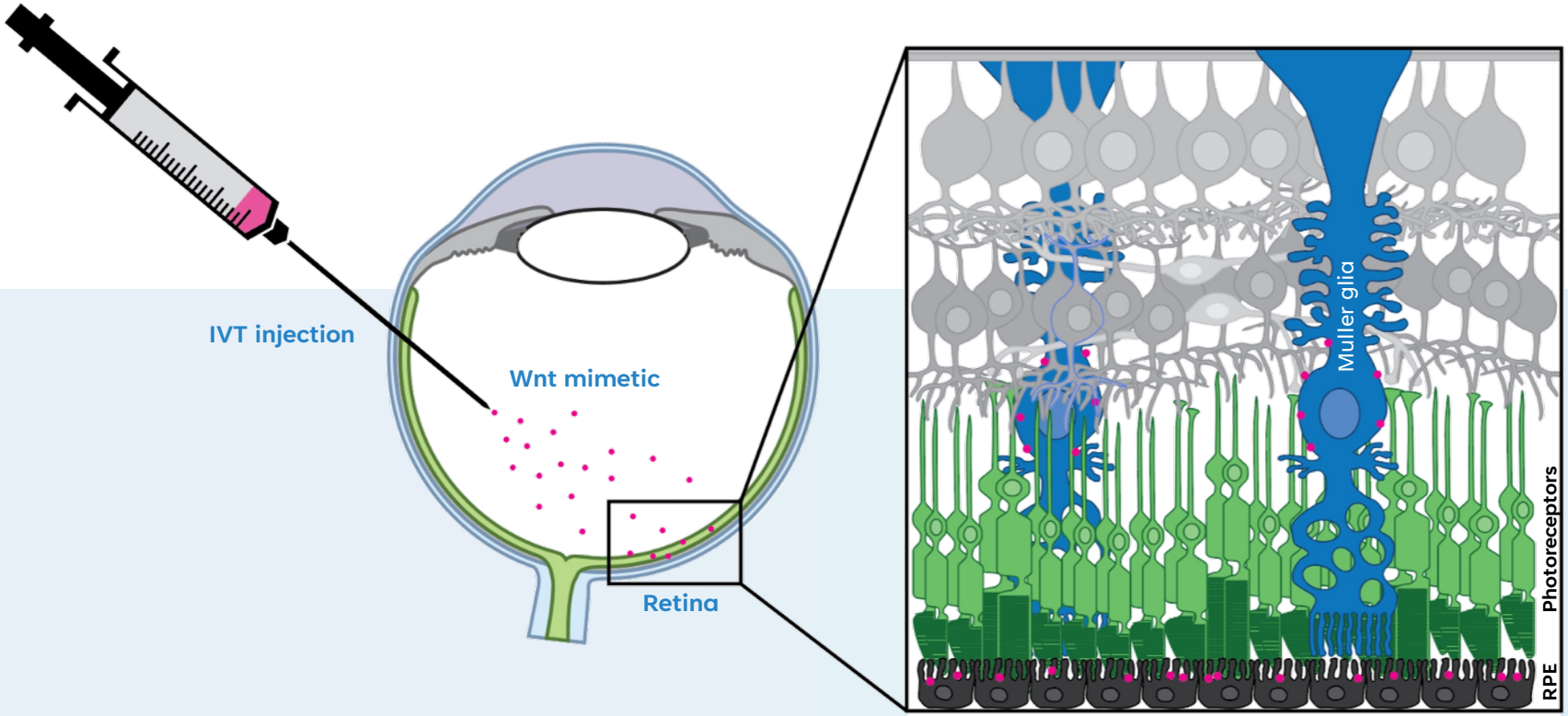
- Directly target RPE—these cells are critical for the survival of photoreceptors
- Stimulate Muller glia—the main supporting cells of the retina—to provide neuroprotection and potential regeneration upon disease onset

Surrozen SWAPs provide significant neuroprotection in a mouse model of MNU-induced photoreceptor degeneration and in retinal degeneration 10 (rd10) mutant mice

Activating Wnt signaling with Surrozen SWAPs **can proliferate and differentiate RPE** *in vitro*

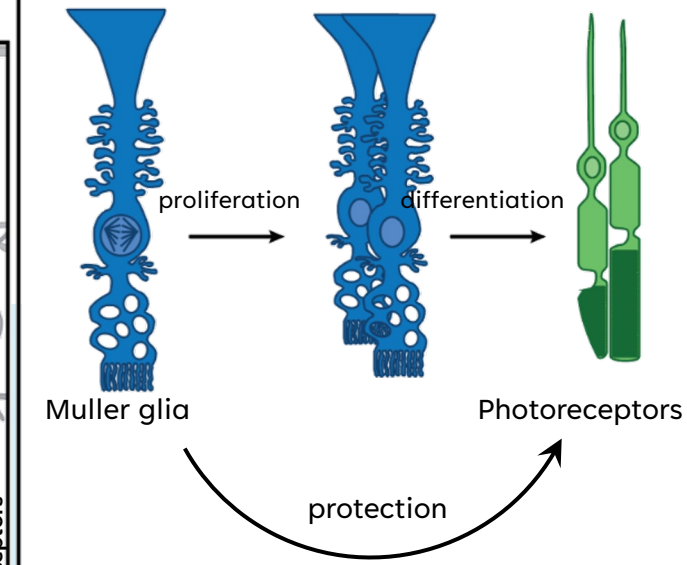
- Increased RPE layer cell proliferation seen in laser photocoagulation model *in vivo*

Leveraging Activation of Wnt Signaling in the Eye Can Provide Protection and/or Regeneration

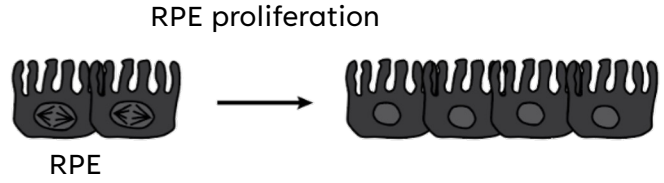


Upon Activation of Wnt/ß-catenin Signaling

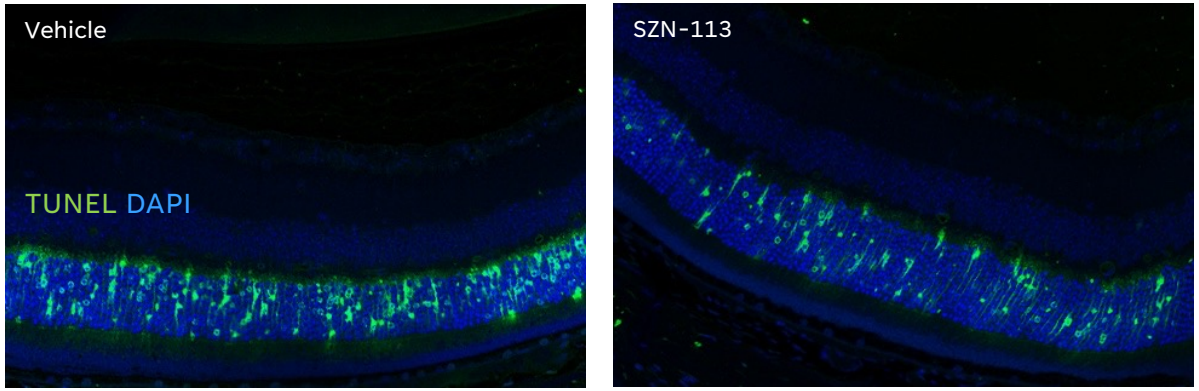
Photoreceptor protection and regeneration



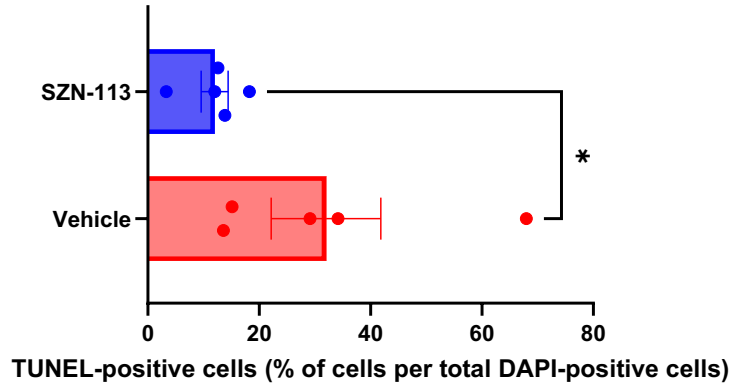
Retinal pigment epithelial regeneration



SZN-113 Can Provide Neuroprotection in a Mouse Model of MNU-induced Photoreceptor Degeneration

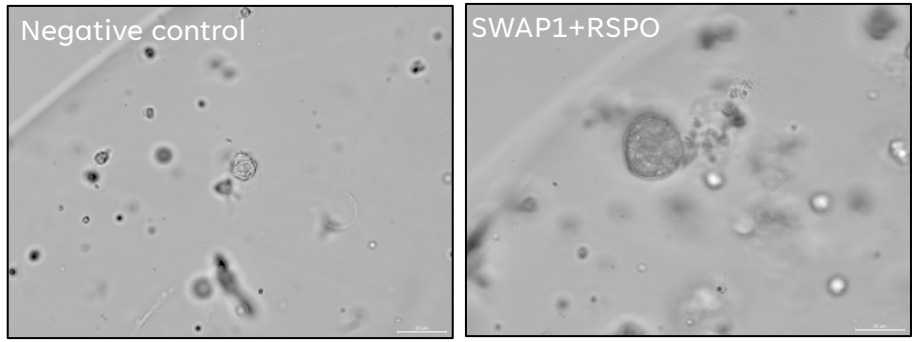


- MNU injury characterized by extensive photoreceptor cell death in the outer nuclear layer
- TUNEL staining detects DNA breaks associated with apoptosis (cell death)
- Multiple SWAP antibodies including SZN-113 mitigated the number of effected cells
- Location of apoptosis in the ONL is consistent with a photoreceptor specific effect



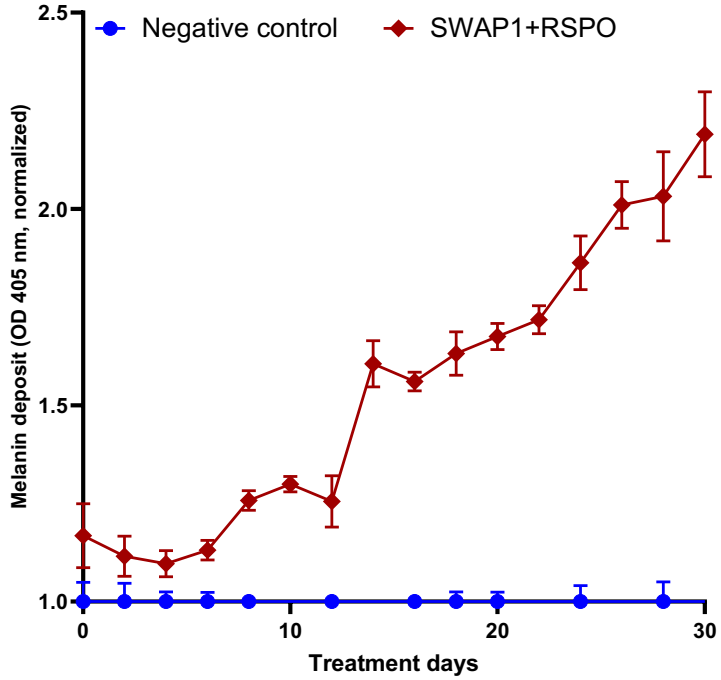
SWAPs Stimulate/Accelerate RPE Proliferation and Differentiation

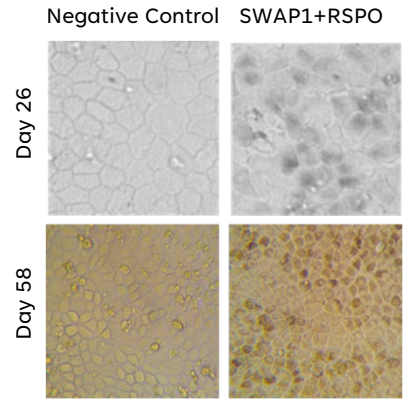
3D human primary RPE expansion (Day 14)



- FZD-specific SWAPs can stimulate proliferation of RPE cells in vitro
- RPE monolayer differentiation can be facilitated by FZD-specific SWAPs

hiPSC-RPE Melanin Deposit





Dry AMD Program Summary

Activation of Wnt signaling in Dry AMD has multiple potential effects:

- Regenerating and maintaining photoreceptors
- Regenerating and maintaining RPE cells

Internal experiments show Surrozen SWAP candidates:

- Provide neuroprotection in acute injury (MNU-induced) and progressive degeneration (rd10 mutant) models of photoreceptor degeneration
- Stimulate RPE proliferation and differentiation in *in vitro* models
- SWAP treatment increases proliferation in a mouse RPE damage model

Glossary

ADA	Anti-drug antibodies	Lille	Prognostic model for AH
AE	Adverse events (SAE – serious AE)	Lrp	Lipoprotein receptor-related protein
AH	Alcohol-associated hepatitis	MAD	Multiple ascending dose
ALP	Alkaline Phosphatase	MELD	Model for end-stage liver disease score
ALT	Alanine Aminotransferase	Mg	Milligrams
AMD	Age-related macular degeneration	MOA	Mechanism of action
ASGR1	Asialoglycoprotein receptor 1	Ndp	Norrie disease gene
AST	Aspartate aminotransferase	NV	Neovascularization
AT1/AT2	Alveolar type epithelial cell	OCA	Obeticholic acid
AUC	Area under the curve	PD	Pharmacodynamics
BW	Biweekly	PK	Pharmacokinetic
CCL4	Carbon tetrachloride	POC	Proof-of-concept
DME	Diabetic macular edema	QD	Daily
Dx	Diagnosis	MAD	Multiple ascending dose
ETOH	Ethyl alcohol	RPE	Retinal pigment epithelium
FECD	Fuchs' endothelial corneal dystrophy	SAD	Single ascending dose
Fzd	Frizzled	sAH	Severe alcohol-associated hepatitis
GFP	Green fluorescence protein	SOC	Standard of care
GLP	Glucagon-like peptide	SUSARs	Suspected unexpected severe adverse reactions
HNF 4 alpha	Hepatocyte nuclear factor 4 alpha	SWAP	Surrozen Wnt signal activating proteins
HV	Healthy volunteer	SWEETS	Surrozen Wnt enhancer engineered for tissue specificity
IgG	Immunoglobulin G	TA	Transaminase
IV	Intravenous	TAA	Thioacetamide
KO	Knock-out model	VEGF	Vascular endothelial growth factor