OPTHEA

OPT-302: A novel therapy for Wet AMD

Corporate Presentation, January 2017

Megan Baldwin PhD, CEO & Managing Director

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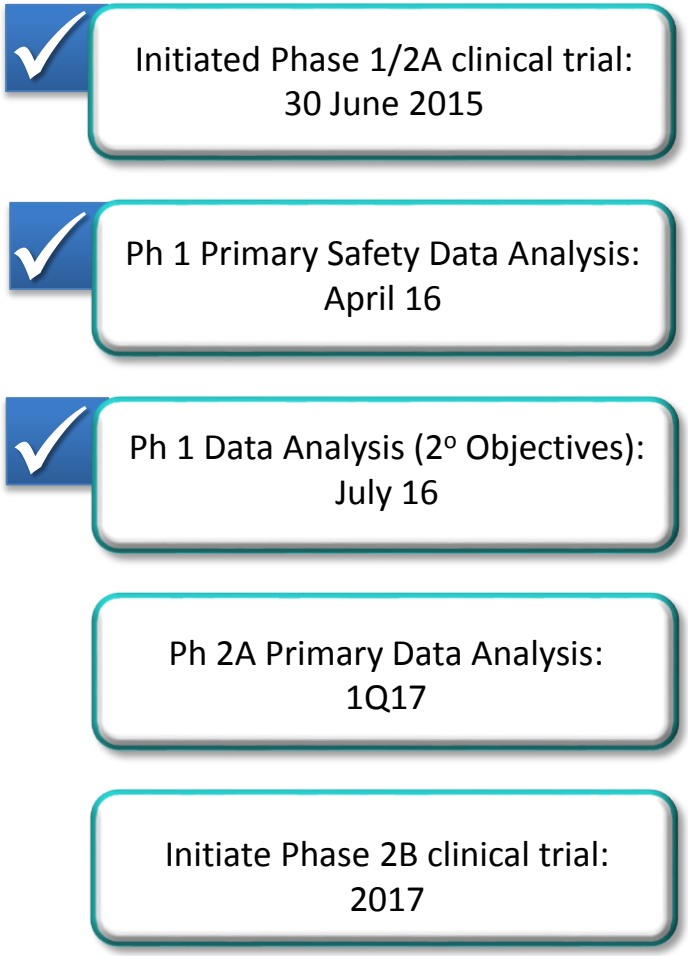
Corporate Summary

- OPT-302 blocks VEGF-C and VEGF-D
- In development for treatment of wet AMD
- Potential in a range of eye diseases
- Combination therapy with approved a-VEGF-A therapies to more completely shut-down VEGF pathway
- Targets mechanism of resistance and sub-optimal clinical response to existing therapies targeting VEGF-A
- Combination therapy for wet AMD is a multi-billion dollar market opportunity
- Phase 1/2A clinical trial ongoing under FDA approved IND at US clinical sites
- Management team with substantial experience in developing drugs targeting the VEGF pathway
- Near-term clinical milestones

OPT-302 Wet AMD Program: Milestones



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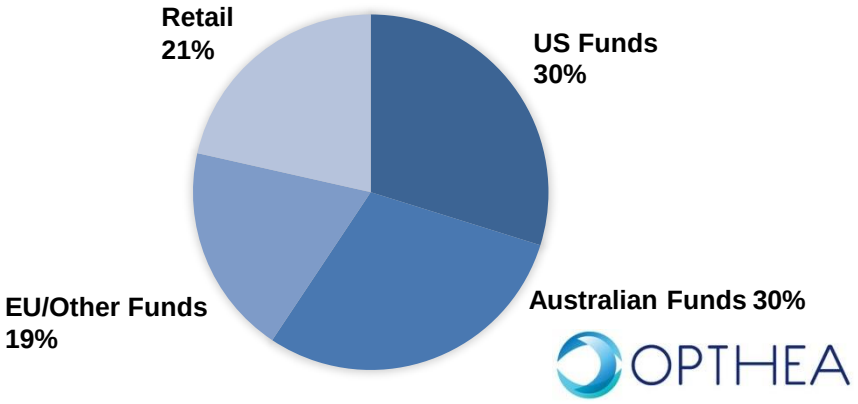
Financial Position (Unaudited)

Key Financial Details	ASX: OPT
Ticker Symbol	ASX:OPT
Share Price (Jan 5 2017)	~A$0.875
Total Ordinary Shares on Issue	150,237,078
Options on Issue	49,675,922
Market Capitalisation (Jan 5 2017)	~A$131m (~USD95m)
Trading Range (last 12 months)	A$0.30 – 0.915
Cash Balance (Dec 31 2016)	~A$13.1m
Forecast Net Operating Cash Burn (CY 2017)	~$7.8m
Top 10 Shareholders Own	69%
Institutional Holders	79%

Substantial Shareholders	% Holding
Biotechnology Value Fund (BVF)	18%
Baker Bros (NY, USA)	9%
Packer & Co.	8.5%

Share Price Performance (Jan 2015 - Jan 2017)

Shareholders by Region



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Board of Directors & Executive Management

Megan Baldwin, PhD, MAICD
CEO and Managing Director
Appointed CEO Feb ’14, joined company in 2008. Over 20 years experience in research and drug development of therapies targeting angiogenesis. Previously held roles at Genentech (now Roche) in US, in R&D and commercial divisions. PhD in Medicine on VEGF-D biology from University of Melbourne & Ludwig Institute for Cancer research

Geoffrey Kempler, B.Sc Grad. Dip. App.Soc. Psych
Non-Executive Chairman
Appointed Chairman Dec ’15. Extensive experience in the global biopharmaceutical industry. Founded and currently CEO Prana Biotechnology. Listed Prana on both ASX and NASDAQ. Strong investment markets experience and networks of domestic and international sophisticated investors. Qualified psychologist, BSc (Monash University) and Grad. Dip. App.Soc.Psych (Swinburne)

Michael Sistenich, MSc
Non-Executive Director
Appointed Dec ‘15. Over 18 years of experience as a healthcare specialist in international investment management and investment banking. He is currently Chief Executive Officer at Nohla Therapeutics Inc and his previous roles include Director of Corporate Finance at Bell Potter Securities and Director of International Equities and Head of Global Healthcare Investments at DWS Investments, Deutsche Bank, Frankfurt

Mike Tonroe, ACA, MAICD
Chief Financial Officer and Company Secretary
Appointed May ‘14. Over 20 yrs experience in finance and company secretarial roles. Previously CFO of the Australian Synchrotron Company Limited. Holds Graduate Degree in Business Studies from Buckingham University and is a Chartered Accountant.

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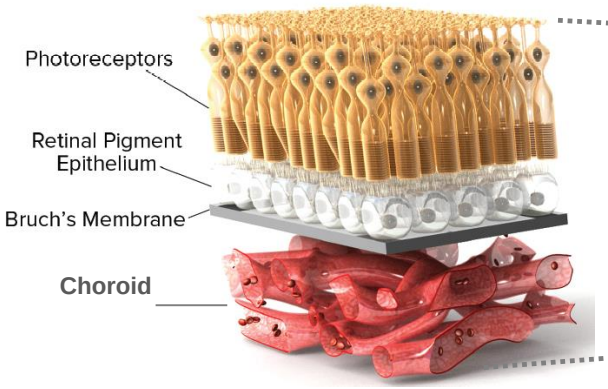
The Disease Process of Wet AMD

Normal Retina



'Wet' AMD

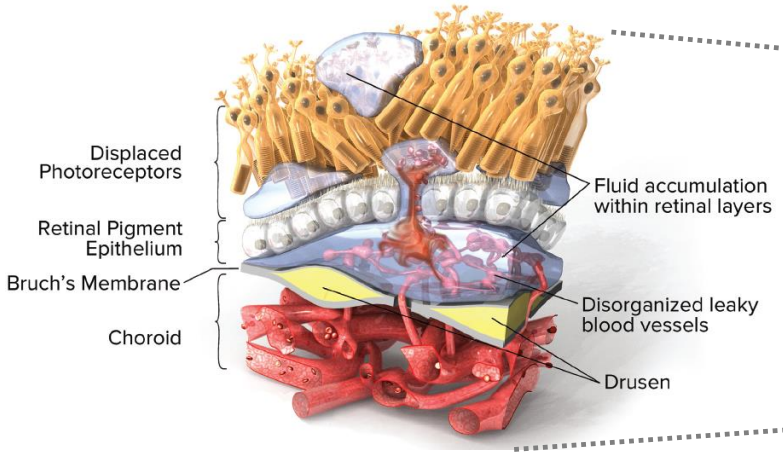


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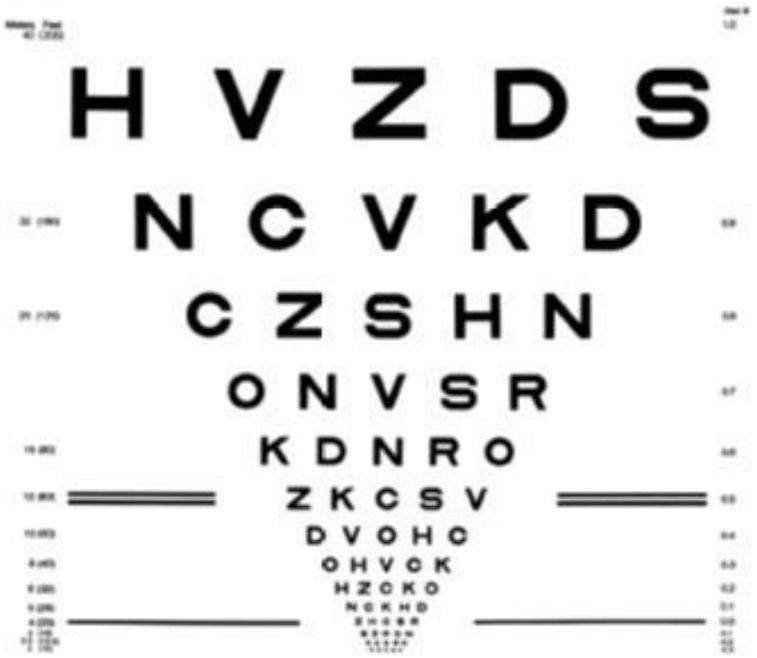
Figure: The Angiogenesis Foundation

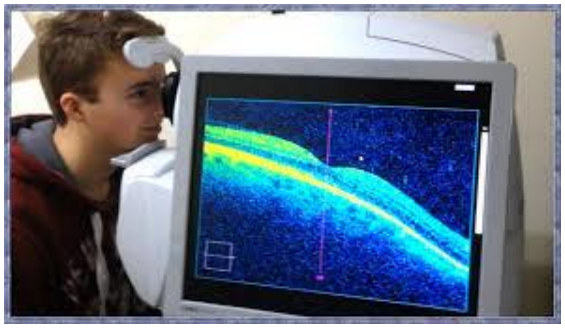
Monitoring Patients & Endpoints in Wet AMD Trials

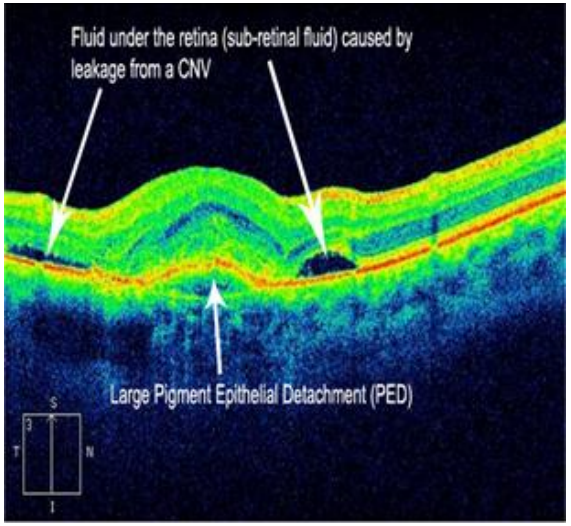
Visual Acuity



Change in Visual Acuity
(# letters) from Baseline

SD-OCT





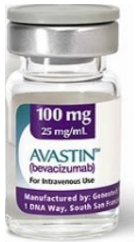
Change in Retinal Subfield Thickness
(CST) from Baseline –
Indicator of fluid

Large and Growing Market Opportunity

- Wet AMD is the leading cause of blindness in the western world in people >55 yrs
- Increasing prevalence due to ageing population
- Estimated 1.8m in US have wet AMD*
- Prevalence expected to double by 2020
- Existing therapies targeting VEGF-A are sub-optimally clinically effective in the majority of patients – major unmet medical need
- Approved therapies for wet AMD target VEGF-A, but not VEGF-C or VEGF-D
- VEGF-C and VEGF-D activate the same, as well as independent pathways, to VEGF-A
- VEGF-C and VEGF-D may mediate resistance to VEGF-A inhibitors, such as the blockbuster drugs Lucentis®, Eylea® and Avastin®

Our approach is novel and differentiated from the existing therapies, yet targets a validated pathway in wet AMD disease progression

2015: >$7BN
40% Market Share



60% Market Share

Market Opportunity*:
>$10BN
Worldwide

*Cowen Analyst Report: Ophthotech July 7 2015

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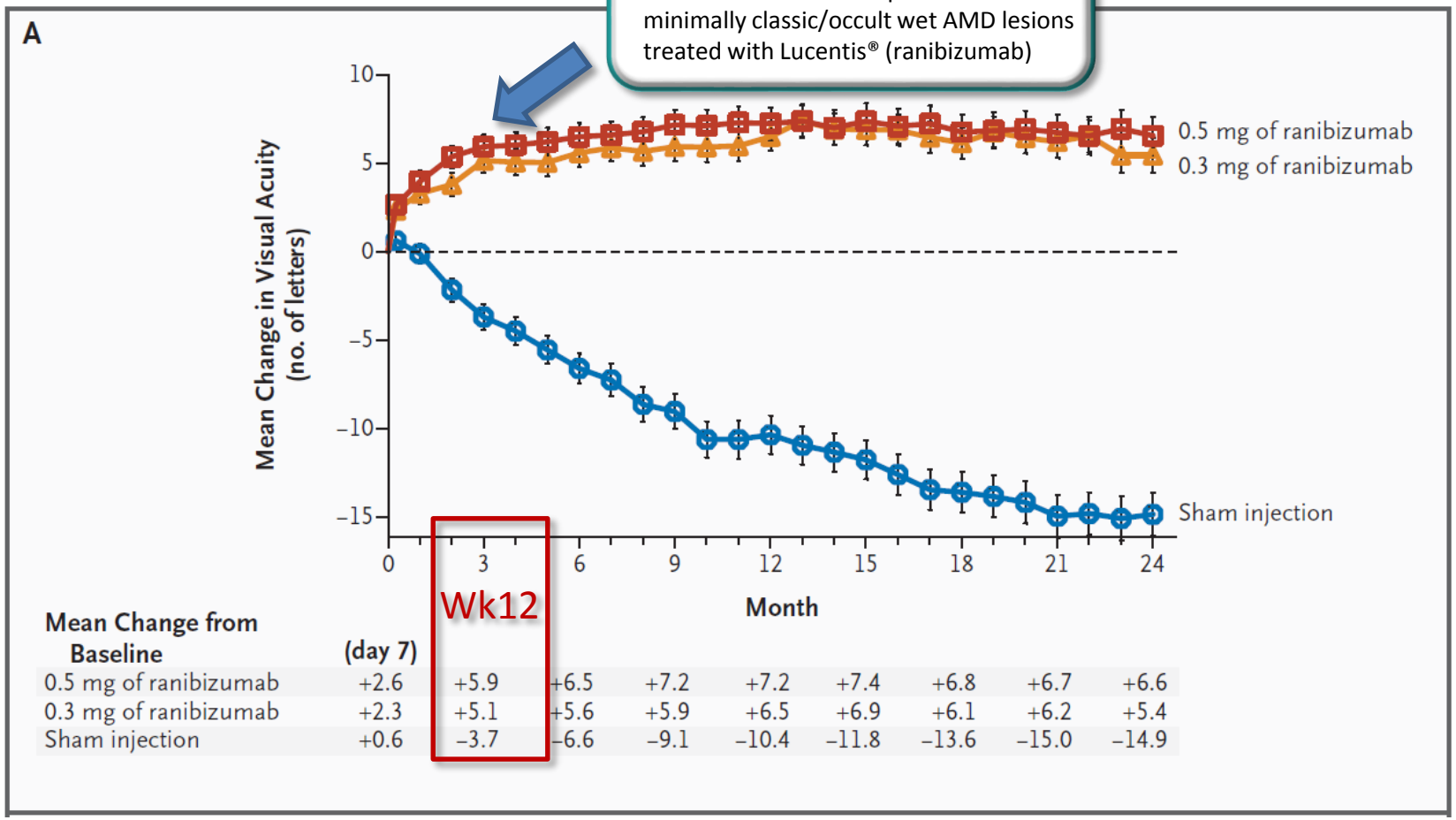
Lucentis® Phase 3 Registrational Studies

In MARINA trial (Phase 3 registrational): ~6 letters gain in vision compared to baseline at Week 12 in patients with minimally classic/occult wet AMD lesions treated with Lucentis® (ranibizumab)



Mean Change from Baseline	(day 7)								
0.5 mg of ranibizumab	+2.6	+5.9	+6.5	+7.2	+7.2	+7.4	+6.8	+6.7	+6.6
0.3 mg of ranibizumab	+2.3	+5.1	+5.6	+5.9	+6.5	+6.9	+6.1	+6.2	+5.4
Sham injection	+0.6	−3.7	−6.6	−9.1	−10.4	−11.8	−13.6	−15.0	−14.9

MARINA Ph3 Trial

Rosenfeld et al., NEJM, 355;14, pp 1419-1431, 2006

Naïve patients with minimally classic or occult lesions
Monthly Lucentis® (ranibizumab) or Sham

- At 3 mos, 5.9 letter gain
- At 2yrs, 33% ≥ 15 letter gain

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An Unmet Medical Need for Wet AMD

Despite receiving a VEGF-A inhibitor (Lucentis®, Eylea® or Avastin®):

>50% do not achieve significant vision gain

2/3 will continue to have fluid at the back of the eye

25% will have further vision loss at 12 mos

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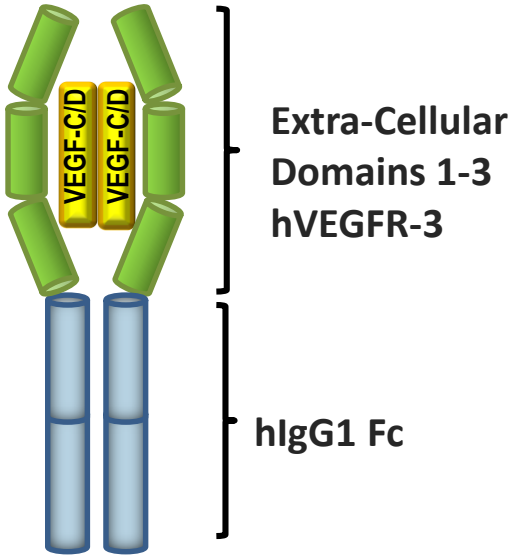
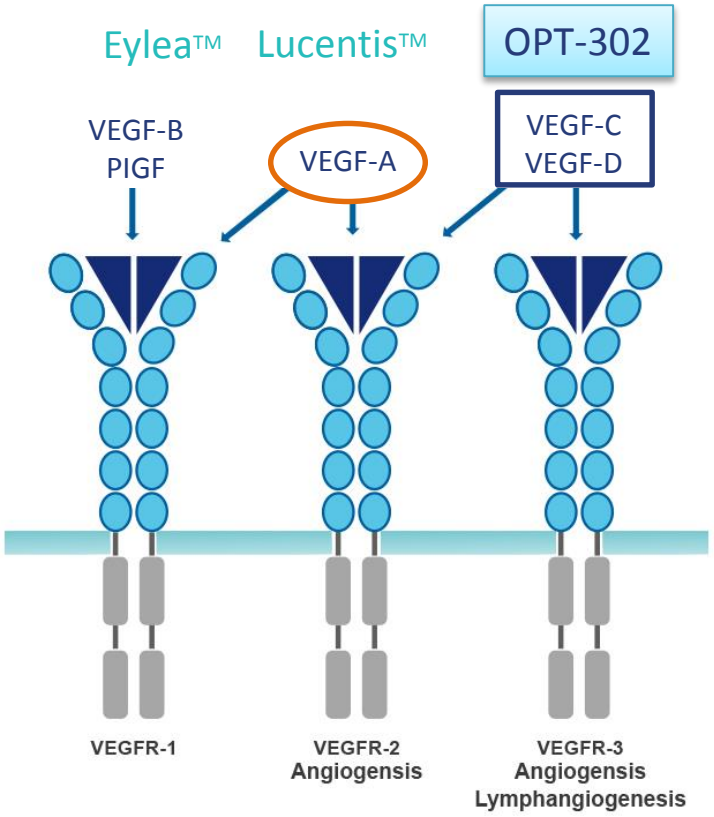
OPT-302 for Wet AMD

- **Lead molecule:**
  - OPT-302 (soluble VEGFR-3, VEGF-C/-D 'Trap')
- **Mechanism:**
  - Blocks VEGF-C and VEGF-D:
    - Inhibits blood vessel growth
    - Inhibits vessel leak
- **Strategy:**
  - To develop OPT-302 for use in combination with existing VEGF-A inhibitors for the treatment of wet AMD
  - Achieve complete blockade of the VEGF pathway
  - Blocks a mechanism of 'escape' from existing therapies



Resistance to anti-VEGF-A Monotherapy

- Long-term single-agent therapy with VEGF-A inhibitors is associated with sub-optimal response
  - Sub-optimal improvements in visual acuity (<15-letter gain)
  - Persistent retinal fluid
- Resistance to VEGF-A monotherapy may be related to other VEGF family members
- **OPT-302 combination therapy achieves more complete suppression of the VEGF/VEGFR pathway**
- **Targets incomplete response to VEGF-A inhibition**



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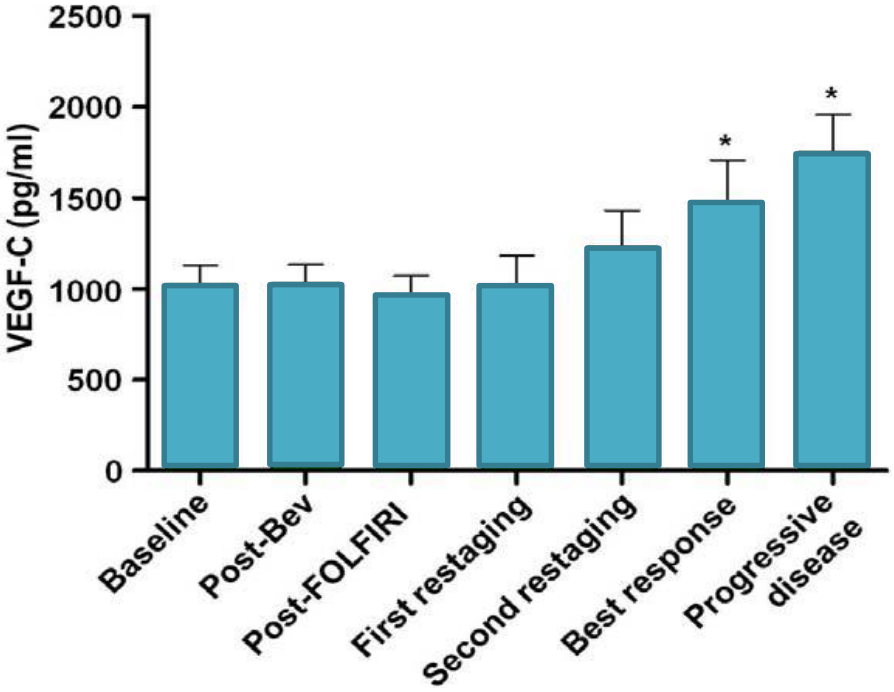
VEGF-A inhibition upregulates VEGF-C/-D in Cancer



"The association of alternate VEGF ligands with resistance to anti-VEGF therapy in metastatic colorectal cancer" - Lieu et al., 2013.

BLE 2. Specific Transcripts That Were Upregulated or Downregulated in a Statistically Significant Manner in acizumab-Evasive Gliomas[a]

G Subset	Direction of Change	Transcript	Change	*P*
Gs	Upregulation	Receptor tyrosine kinase Ephrin A4	2.3-fold	.008
		Matrix metalloproteinase-7	2.3-fold	.009
		Basic fibroblast growth factor	2.1-fold	.009
		Matrix-remodeling associated	1.9-fold	.01
		Insulin-like growth factor binding protein 2	2.1-fold	.02
		Receptor tyrosine kinase C-met	2.5-fold	.003
	Downregulation	Vascular endothelial growth factor-B	2.1-fold	.04
BEGs	Upregulation	Vascular endothelial growth factor-A	2.2-fold	.004
		Vascular endothelial growth factor-C	2.1-fold	.003
		Aquaporin 4	2.3-fold	.001
		Aquaporin 10	2.2-fold	.002
	Downregulation	Basic fibroblast growth factor	1.9-fold	.03
		Receptor tyrosine kinase Ephrin A4	1.8-fold	.04
		Matrix metalloproteinase-7	1.9-fold	.04

"Mechanisms of evasion to antiangiogenic therapy in glioblastoma"
Rose et al., 2010.

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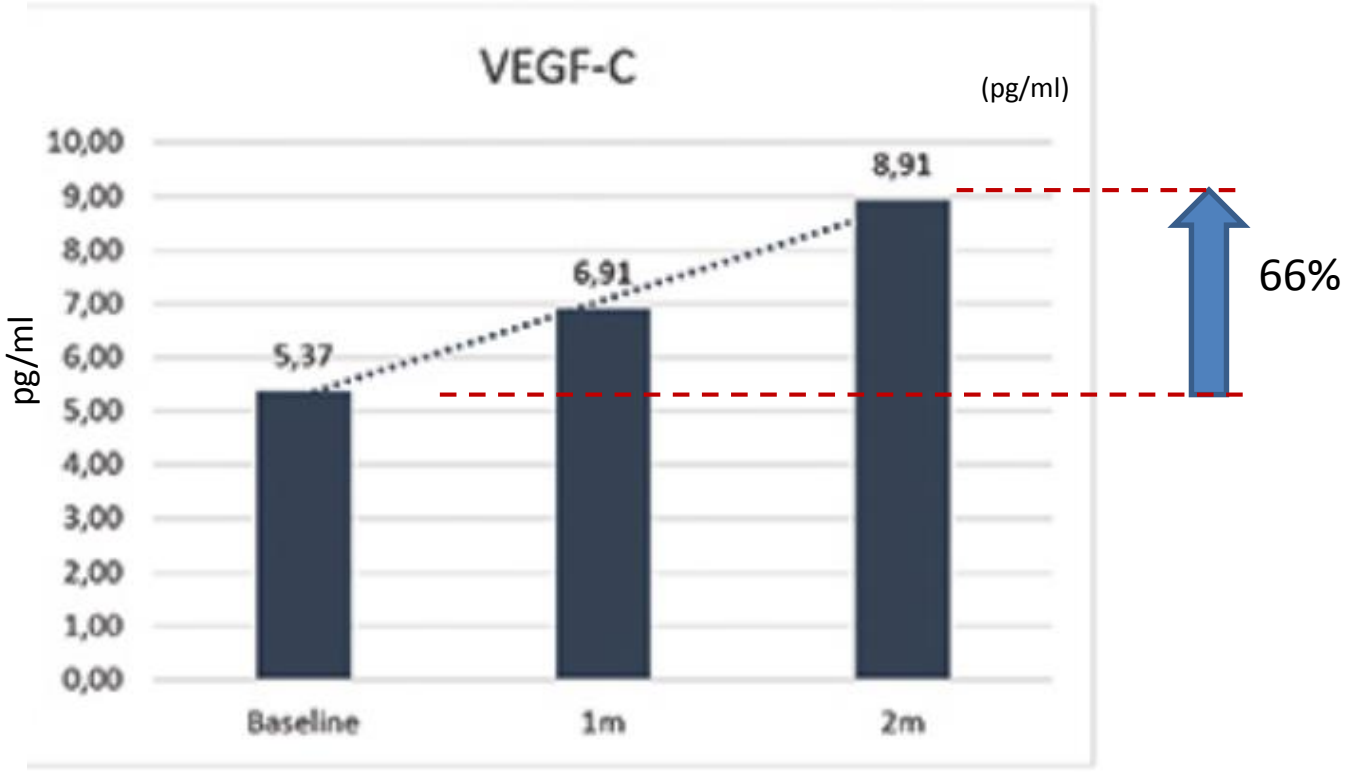
Elevated VEGF-C in Wet AMD Patients

- VEGF-C levels in the retina increase with disease severity
- Aqueous levels of VEGF-C are significantly increased at 1 and 2 months following IVT injection of Avastin® (a-VEGF-A mAb) to wet AMD pts*



* *ARVO (Association for Research in Vision & Ophthalmology) Annual Meeting 2016, Cabral et al., Program 3341, Poster D0144*

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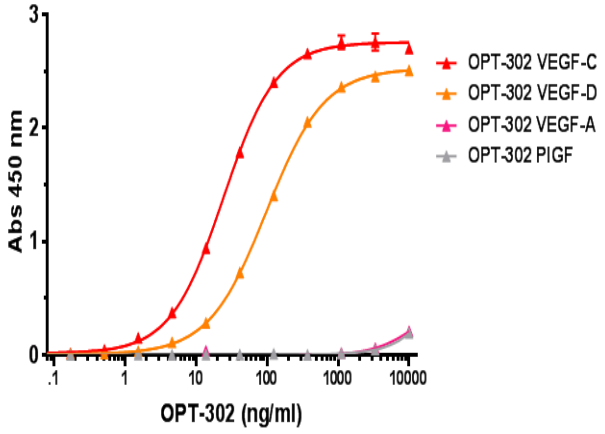
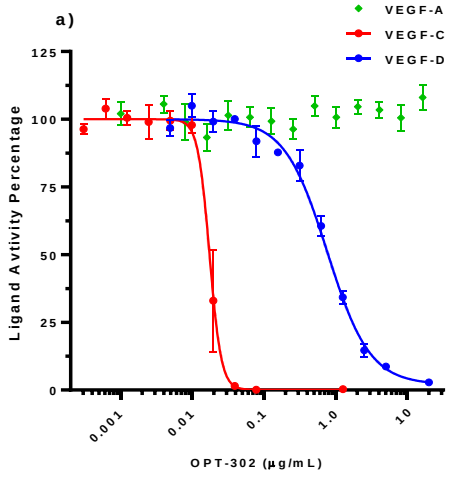
OPT-302

- OPT-302: a soluble form of VEGFR-3
- Comprises the extracellular domains 1-3 of VEGFR-3 and the Fc Fragment of human IgG1
- Potent inhibitor of VEGF-C (~5pM) and VEGF-D (~0.5 nM)
- A 'trap' that binds and neutralises the activity of VEGF-C and VEGF-D, blocking binding to the receptors VEGFR-2 and VEGFR-3

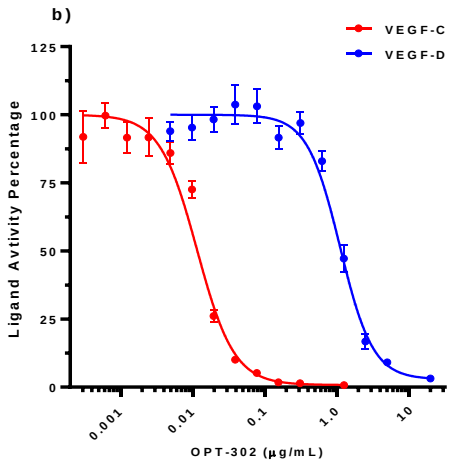
ELISA



VEGFR-2 bioassay



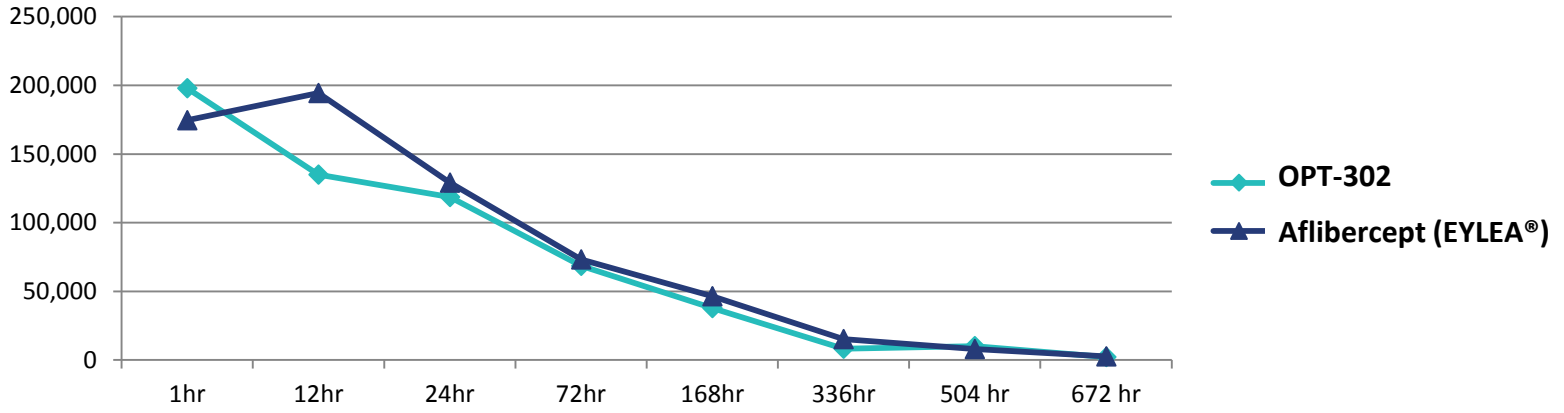
VEGFR-3 bioassay



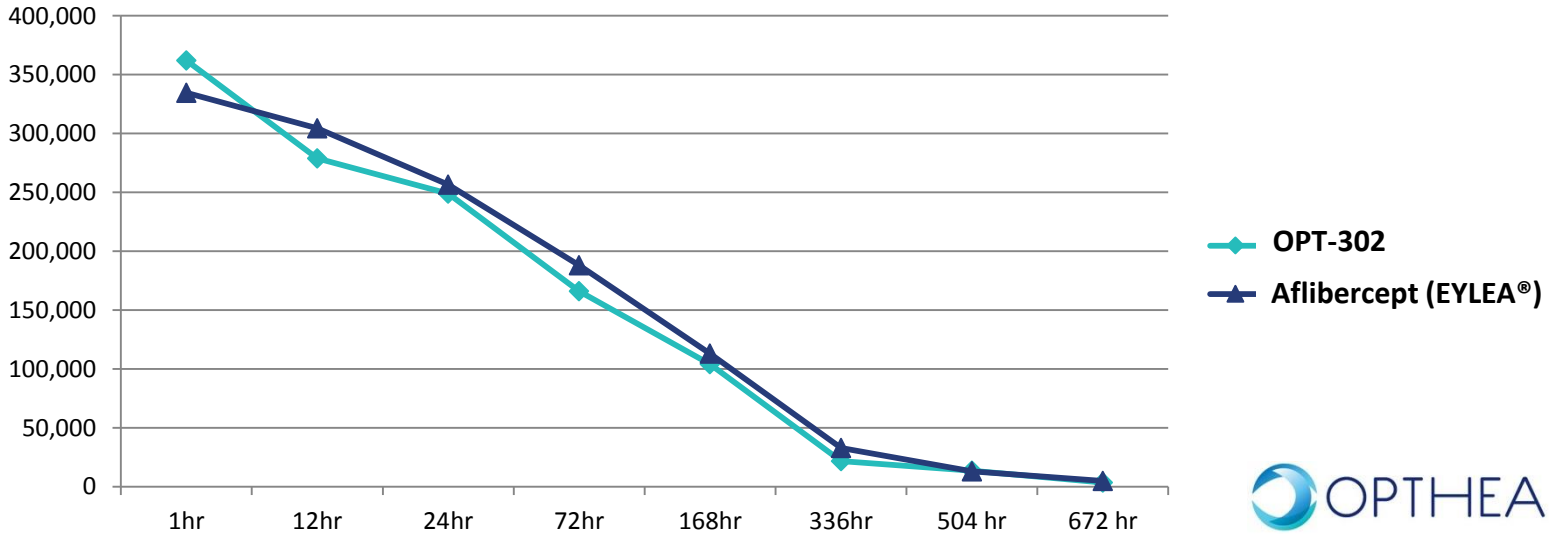
The Ocular Biodistribution & PK of OPT-302 is Comparable to EYLEA® in Rabbits

OPT-302 has comparable ocular biodistribution and PK profile in rabbits to EYLEA®.

Retina



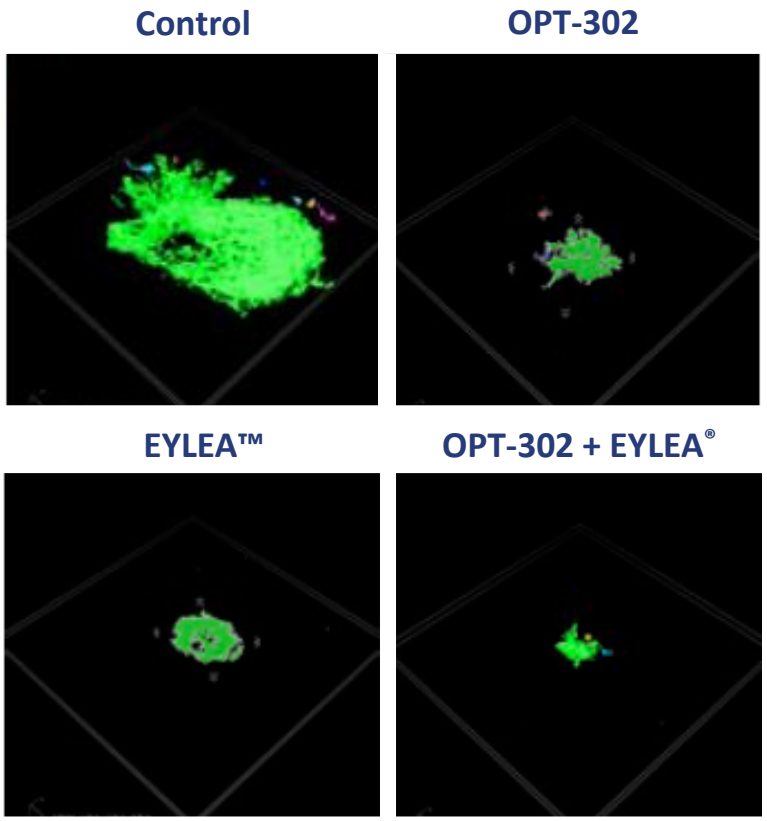
Vitreous Humor

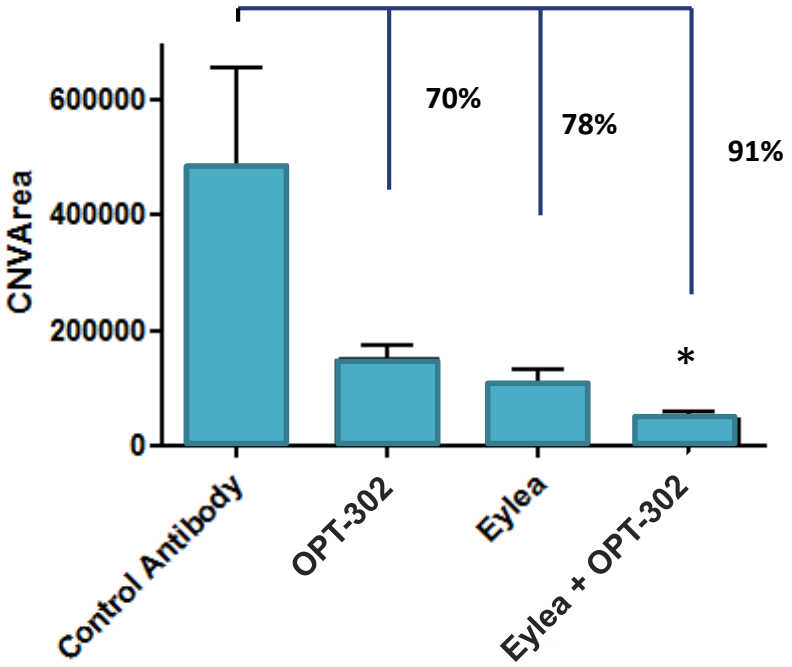


Concentration ngEq/mL

OPT-302 Activity in Mouse Wet AMD Model

Combined inhibition of VEGF-A (Eylea®), VEGF-C and VEGF-D (OPT-302) is more effective than inhibition of VEGF-A alone





** Pairwise comparison: OPT-302 vs Eylea + OPT-302 (p<0.02)*
Eylea vs Eylea + OPT-302 (p<0.05)

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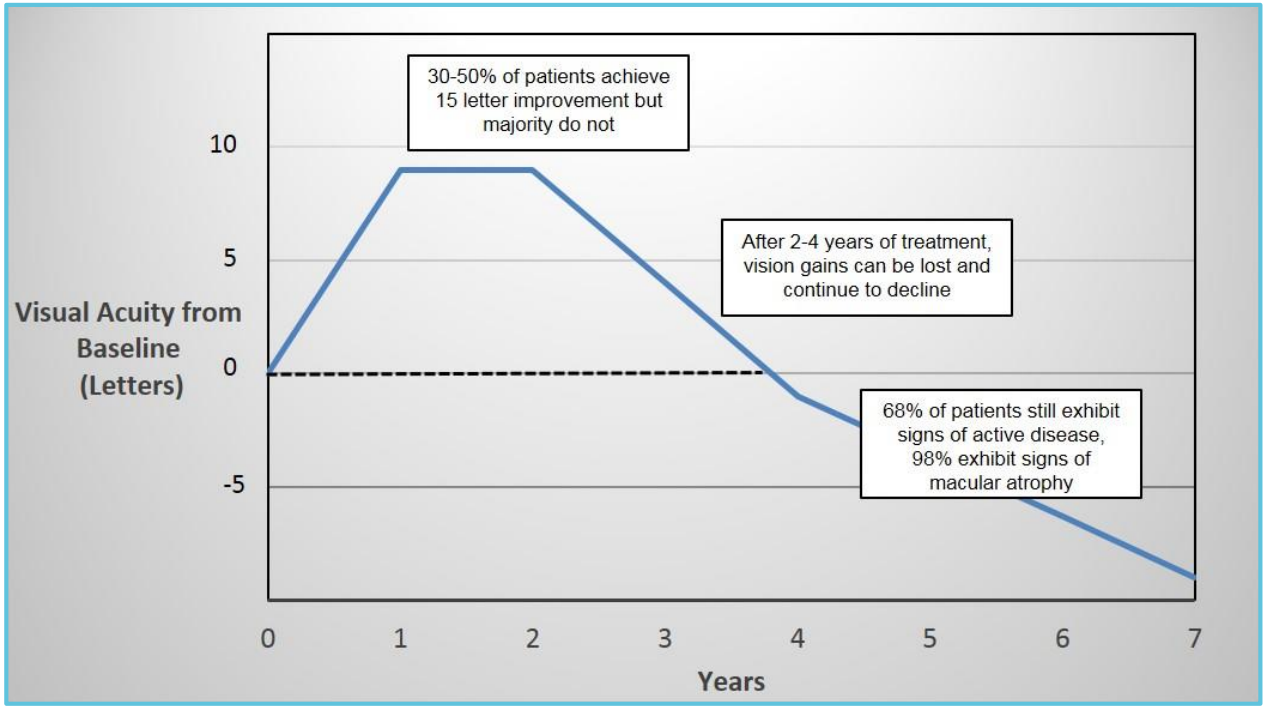
Tammela et al. Nature., 2008

The Opportunity for OPT-302



- To increase the **number** of patients who experience a significant gain in vision
- To increase the **magnitude** of the vision gain
- To **prolong response** to therapy and prevent visual decline
- Potential to **reduce** dosing frequency

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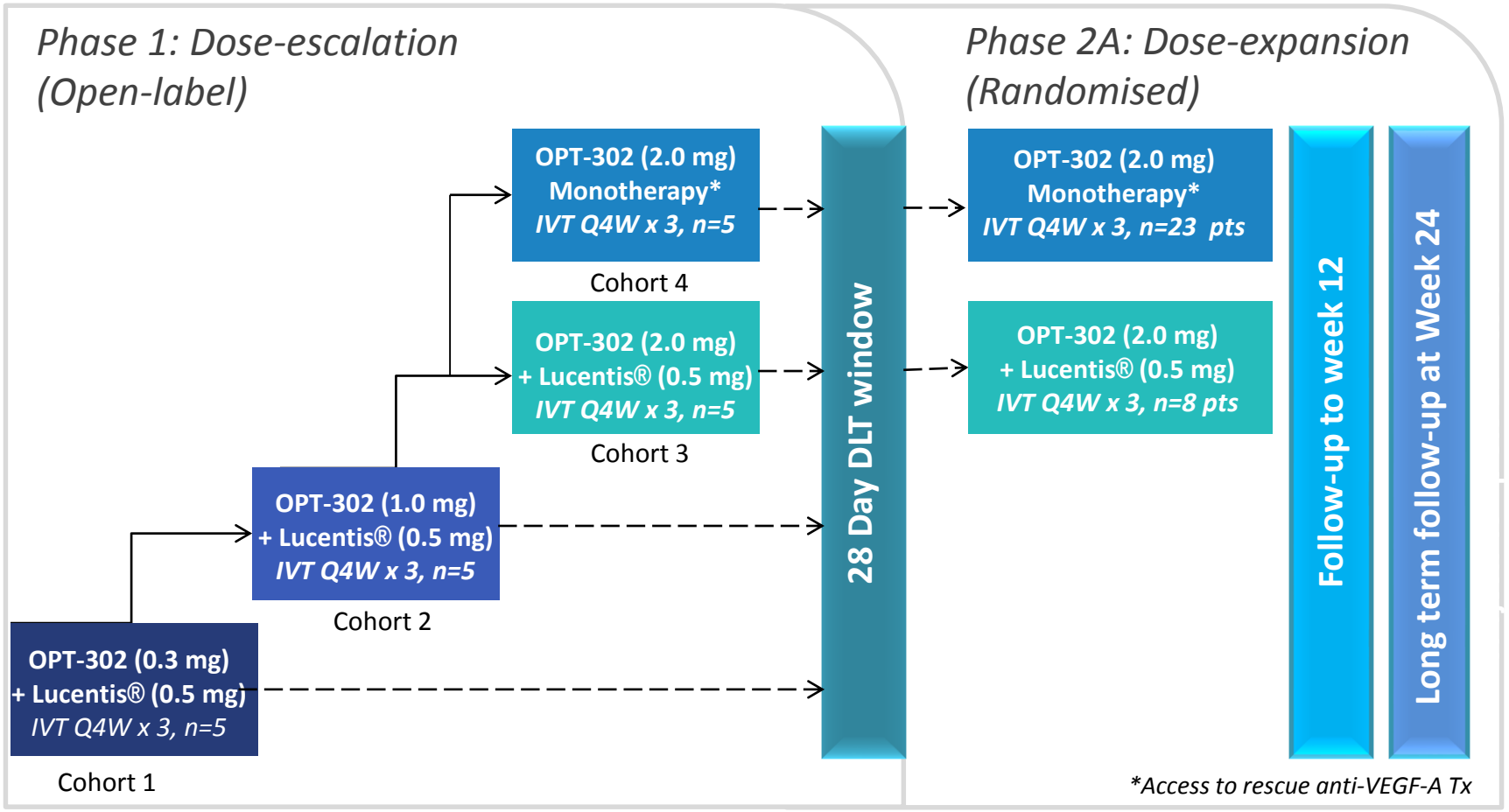
OPT-302 Phase 1/2A

ClinTrials.gov ID#: NCT02543229

Dose-escalation & dose-expansion of repeated IVT injections



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OPT-302 Phase 1/2A Study Objectives

- **Primary Objectives:**
  - To evaluate the safety and establish the dose of OPT-302 administered by intravitreal (IVT) injection in combination with IVT Lucentis® in subjects with wet AMD
- **Secondary Objectives:**
  - Mean change in BCVA (visual acuity) (ETDRS) from baseline
  - Mean change in central retinal thickness from baseline (SD-OCT)
  - Mean change in CNV lesion area from baseline (FA)
  - Mean time to, and number of, retreatment injections of anti-VEGF-A therapy during long term follow-up (week 12 to 24)
  - Need for 'rescue therapy' with ranibizumab in subjects receiving OPT-302 monotherapy
  - Pharmacokinetics (PK) of OPT-302
  - Incidence of anti-OPT-302 antibody formation
- **Exploratory Objective(s):**
  - To evaluate changes in systemic levels of angiogenesis-related biomarkers

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OPT-302 Phase 1: Patient Demographics

- Run under FDA IND at 14 clinical sites in the US
- 20 pts (mean age 74.8)
- 14/20 females, 6/20 males
- 17/20 occult, 2/20 min classic, 1/20 predominantly classic
- Each patient received 3 intravitreal injections of Lucentis® of OPT-302 either alone or in combination with Lucentis ® every 4 weeks, with a week 12 follow-up one month after the third dose.
- **70% difficult to treat patients sub-responsive to anti-VEGF-A therapy**
- 30% treatment-naïve

Cohort	Treatment	# Naïve Pts	# Prior Treated Pts
1	OPT-302 (0.3 mg) + Lucentis® (0.5 mg)	2	3
2	OPT-302 (1.0 mg) + Lucentis® (0.5 mg)	0	5
3	OPT-302 (2.0 mg) + Lucentis® (0.5 mg)	2	3*
4	OPT-302 (2.0 mg)	2	3

**One pt with metastatic ovarian cancer died prior to the week 12 (day 78) visit due to intercurrent illness unrelated to study drugs.*

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OPT-302 Safe & Well Tolerated in Phase 1 Study

- **OPT-302 successfully met primary safety objective in Phase 1 dose escalation study**
- No dose limiting toxicities (and MTD not reached) through week 12 in:
  - OPT-302 monotherapy (2.0 mg), and
  - Cohorts of OPT-302 (0.3, 1, 2 mg) in combination with Lucentis® (0.5 mg)
- No signs of infection (endophthalmitis)
- No clinically significant changes in:
  - Intraocular pressure
  - ECGs
  - Blood pressure
  - Blood chemistry or other vital signs
- No evidence of drug-related immunogenicity

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Phase 1 Secondary Endpoints

- Overall, 16/19 evaluable pts maintained or gained vision from baseline to week 12
- No patient lost more than 3 letters. All of the patients that lost VA from baseline received combination OPT-302 + Lucentis® therapy.

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Naïve Patients

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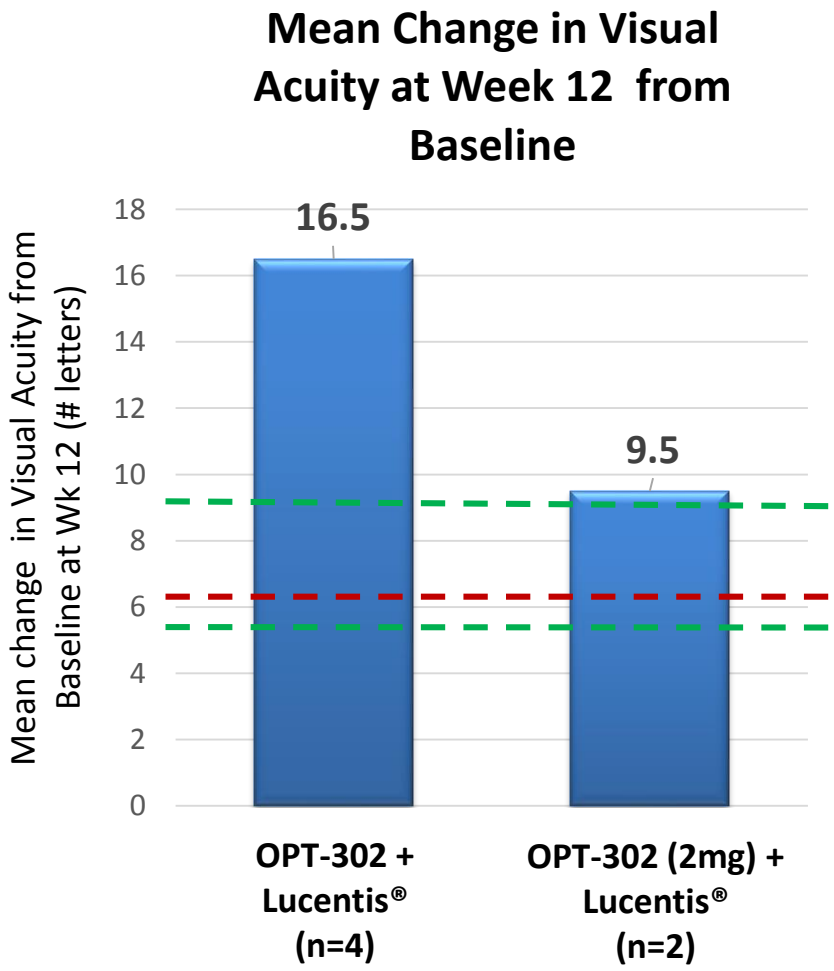
Treatment-Naïve Patients: Visual Acuity



8.7 Letters Fovista® + Lucentis® W12 (Ophthotech Ph2b)

5.9 Letters Lucentis® W12 (MARINA Ph3)

5.1 Letters Lucentis® W12 (Ophthotech Ph2b)

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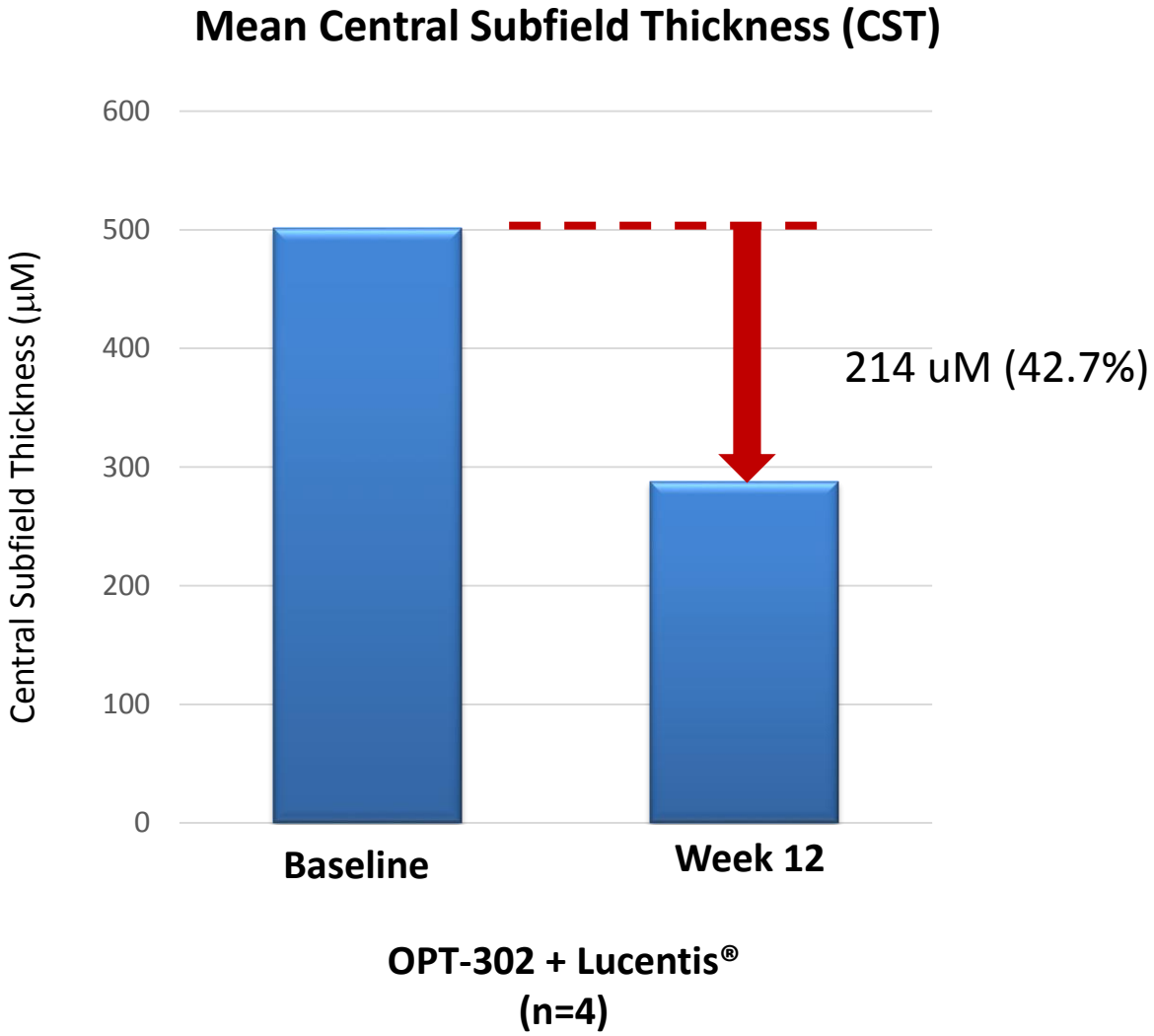
Treatment-Naïve Patients: Retinal Thickness



Prior-Treated Patients

(Sub-responsive to anti-VEGF-A)

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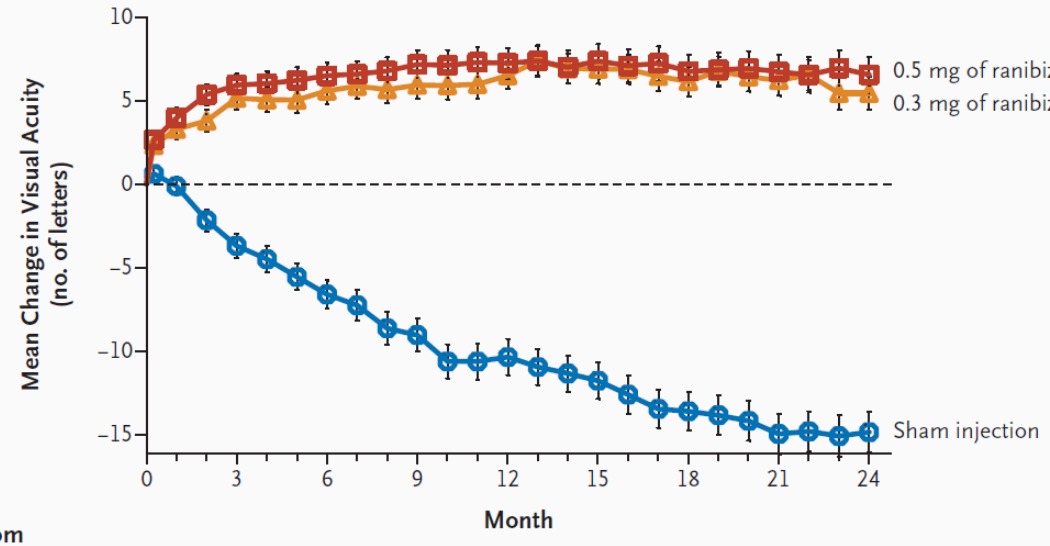
Prior-Treated Patients: Visual Acuity

- Majority of vision gain in Lucentis® treated patients occurs within 3 months
- Plateau “ceiling effect”of response with no other treatment options
- Difficult to treat patient population, very large market opportunity
- Mean number of Prior anti-VEGF-A therapies: 10.5 (Mean 3 – 55)



Mean Change from Baseline	(day 7)								
0.5 mg of ranibizumab	+2.6	+5.9	+6.5	+7.2	+7.2	+7.4	+6.8	+6.7	+6.6
0.3 mg of ranibizumab	+2.3	+5.1	+5.6	+5.9	+6.5	+6.9	+6.1	+6.2	+5.4
Sham injection	+0.6	−3.7	−6.6	−9.1	−10.4	−11.8	−13.6	−15.0	−14.9

MARINA Phase 3 in wet AMD. Rosenfeld et al., NEJM, 355;14, pp 1419-1431, 2006

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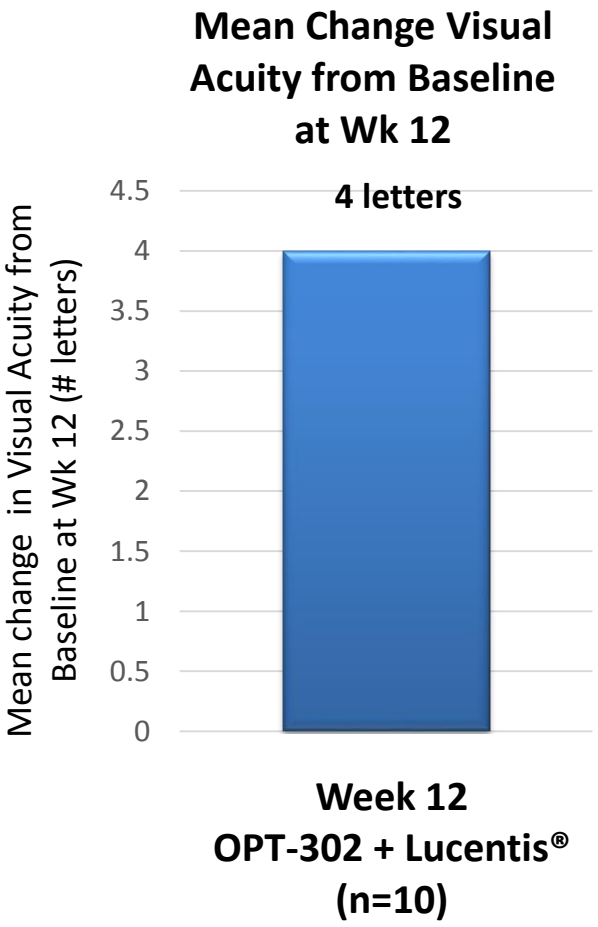
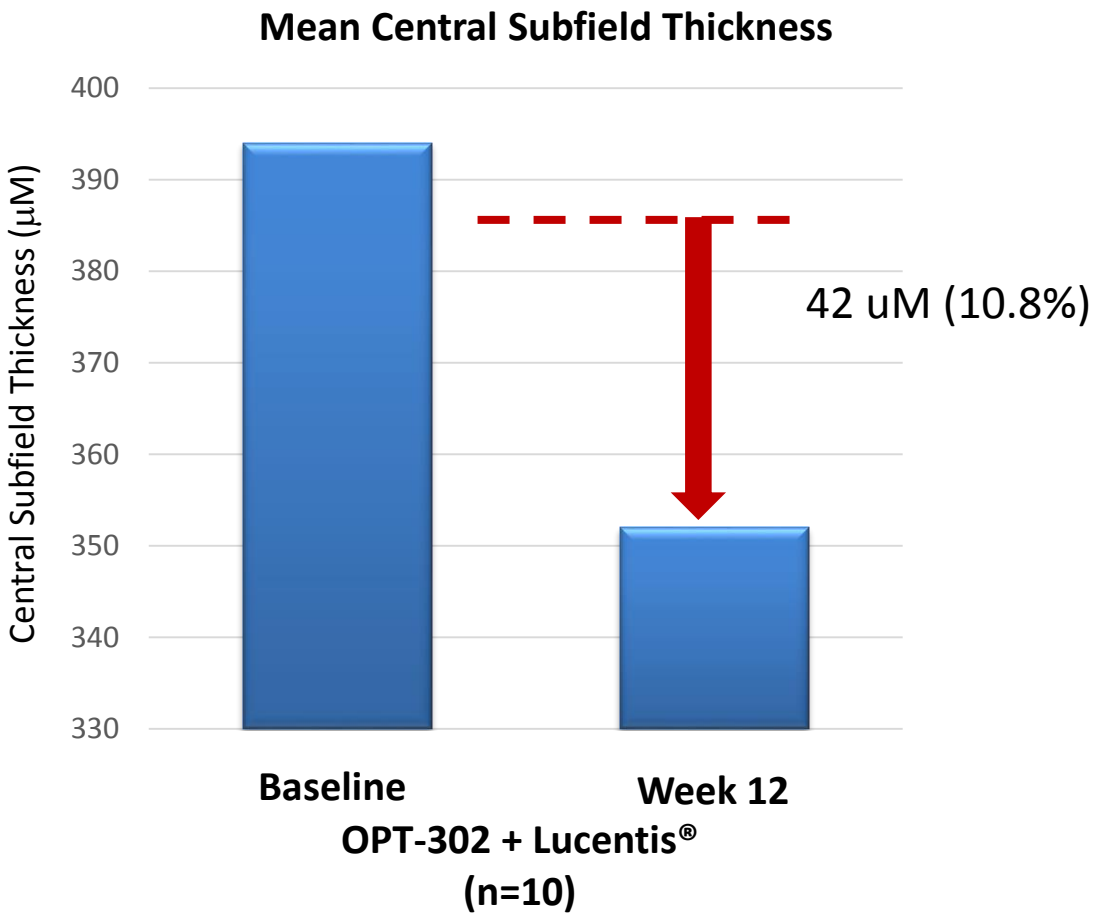
Prior-Treated Patients: Visual Acuity & Retinal Thickness





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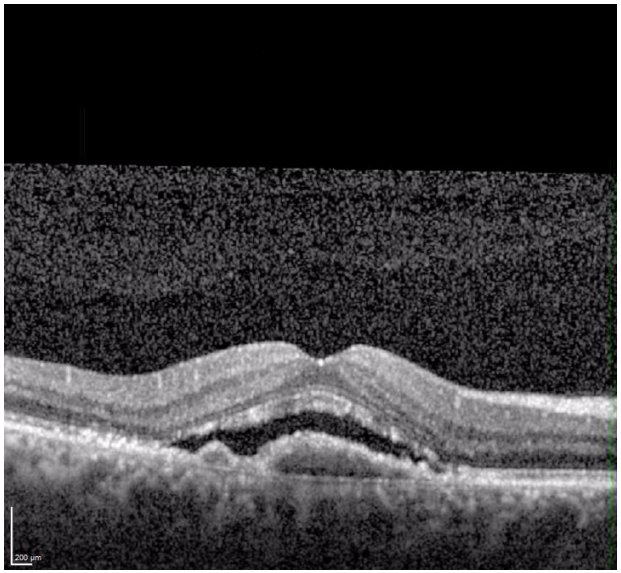
Prior-Treated Patient: OPT-302 (0.3 mg) + Lucentis® (0.5 mg)

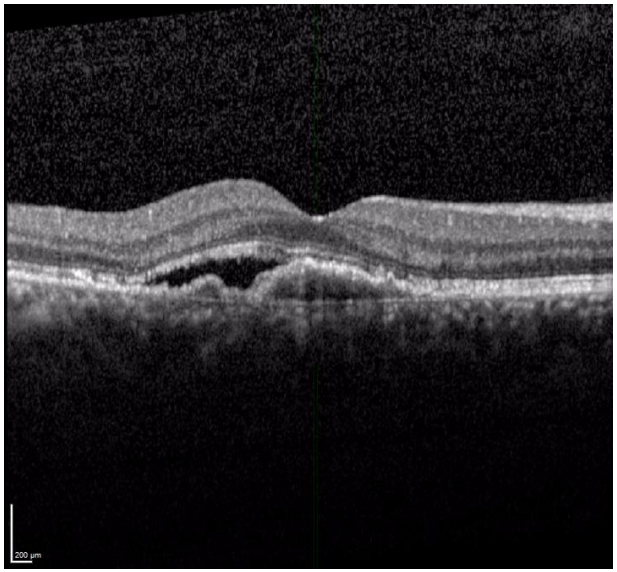
- Male aged 64
- Occult lesion
- Prior treatment: Eylea®/REGN-910-3 x6

Baseline



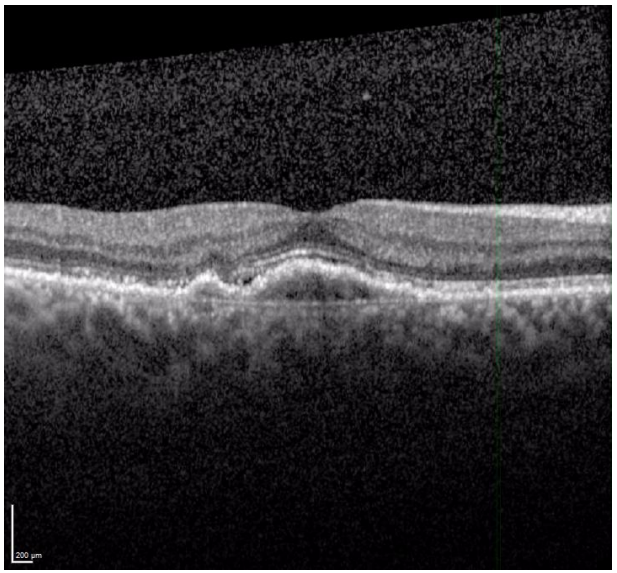
VA: 77 letters
CST: 365 µM

Week 4



VA: 83 letters
CST: 281 µM

Week 12



VA: 79 letters
CST: 298 µM

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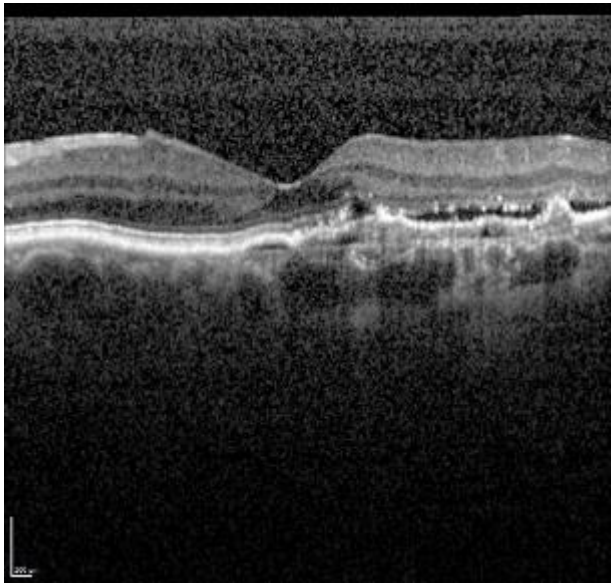
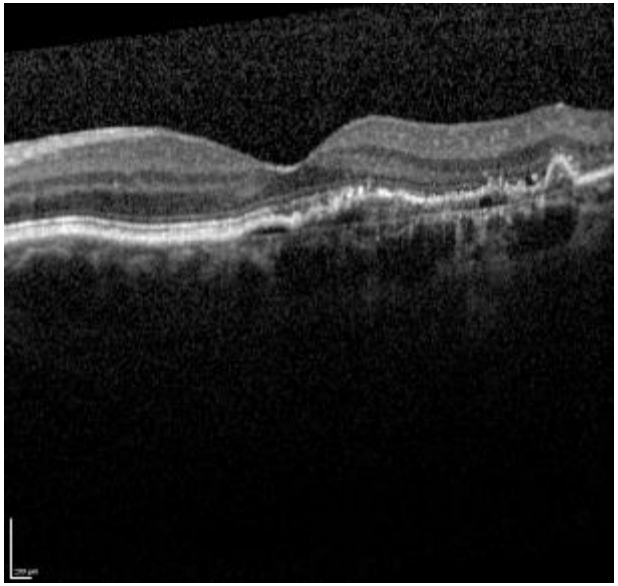
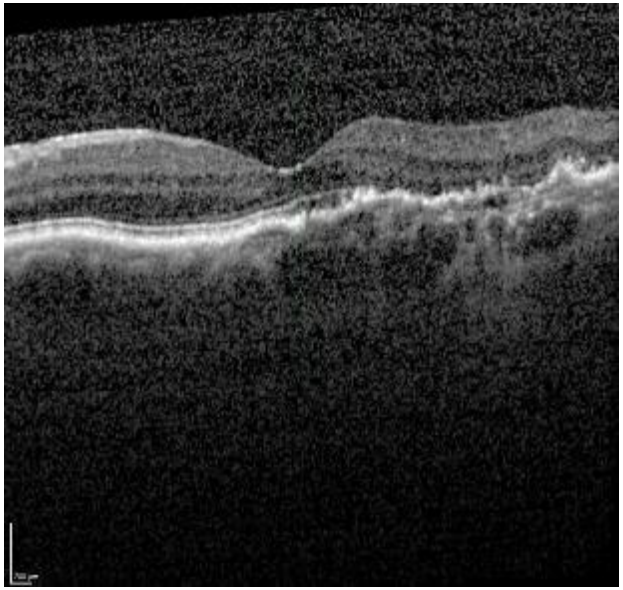
Prior-Treated Patient: OPT-302 (1.0 mg) + Lucentis® (0.5 mg)

- Female aged 71
- Occult lesion
- Prior treatment: Avastin® x10

Baseline	Week 4	Week 12
		
VA: 74 letters CST: 270 µM	VA: 74 letters CST: 258 µM	VA: 84 letters CST: 255 µM

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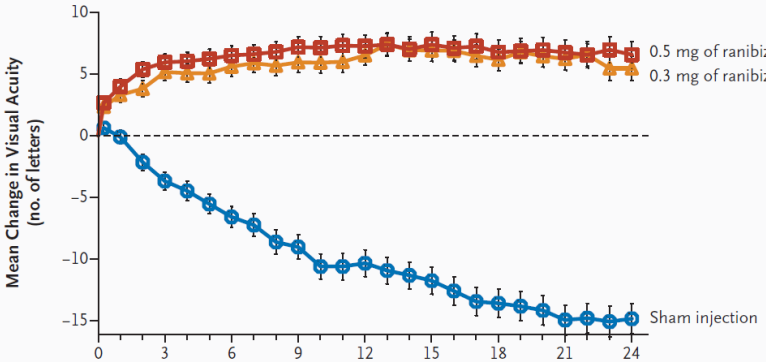
OPT-302 Monotherapy

OPT-302 Monotherapy

- OPT-302 monotherapy included to identify clinical activity without a background standard of care
- Natural history of wet AMD: without treatment, often chronic, rapid decline in visual acuity and increase in retinal fluid
- α-VEGF-A rescue therapy offered to patients at physician discretion or if patient met criteria of progression according to defined criteria
- 3/5 patients did not require 'rescue' therapy
- At week 12, in patients that did not require rescue therapy, mean VA gain of 3.3 letters from baseline (range 2 to 6 letters) and mean increase in CST of 18 uM
- 2 patients were rescued (at d25 and d29). At week 12, despite rescue with ranibizumab, both had lost vision compared to baseline



Mean Change from Baseline	(day 7)								
0.5 mg of ranibizumab	+2.6	+5.9	+6.5	+7.2	+7.2	+7.4	+6.8	+6.7	+6.6
0.3 mg of ranibizumab	+2.3	+5.1	+5.6	+5.9	+6.5	+6.9	+6.1	+6.2	+5.4
Sham injection	+0.6	−3.7	−6.6	−9.1	−10.4	−11.8	−13.6	−15.0	−14.9

MARINA Phase 3 in wet AMD. Rosenfeld et al., NEJM, 355;14, pp 1419-1431, 2006

Phase 1 Secondary Endpoints: VA and OCT Data Summary

- Overall, 16/19 evaluable pts maintained or gained vision from baseline to week 12

Treatment	Mean VA gain at Wk12 from Baseline (# letters)	Mean Reduction in Central Subfield Thickness at Wk12 from Baseline
Combo OPT-302 + Lucentis® in both Naïve & Prior Tx pts (n=14)	+8	-91 µM
Combo OPT-302 + Lucentis® in Naïve pts (n=4)	+16.5	-214 µM
Combo OPT-302 (2 mg) + Lucentis® in Naïve pts (n=2)	+9.5	-262 µM
Combo OPT-302 + Lucentis® in Prior Tx pts (n=10)	+4	-42 µM
OPT-302 Monotherapy in Non-Rescue pts (n=3)	+3	+18 µM

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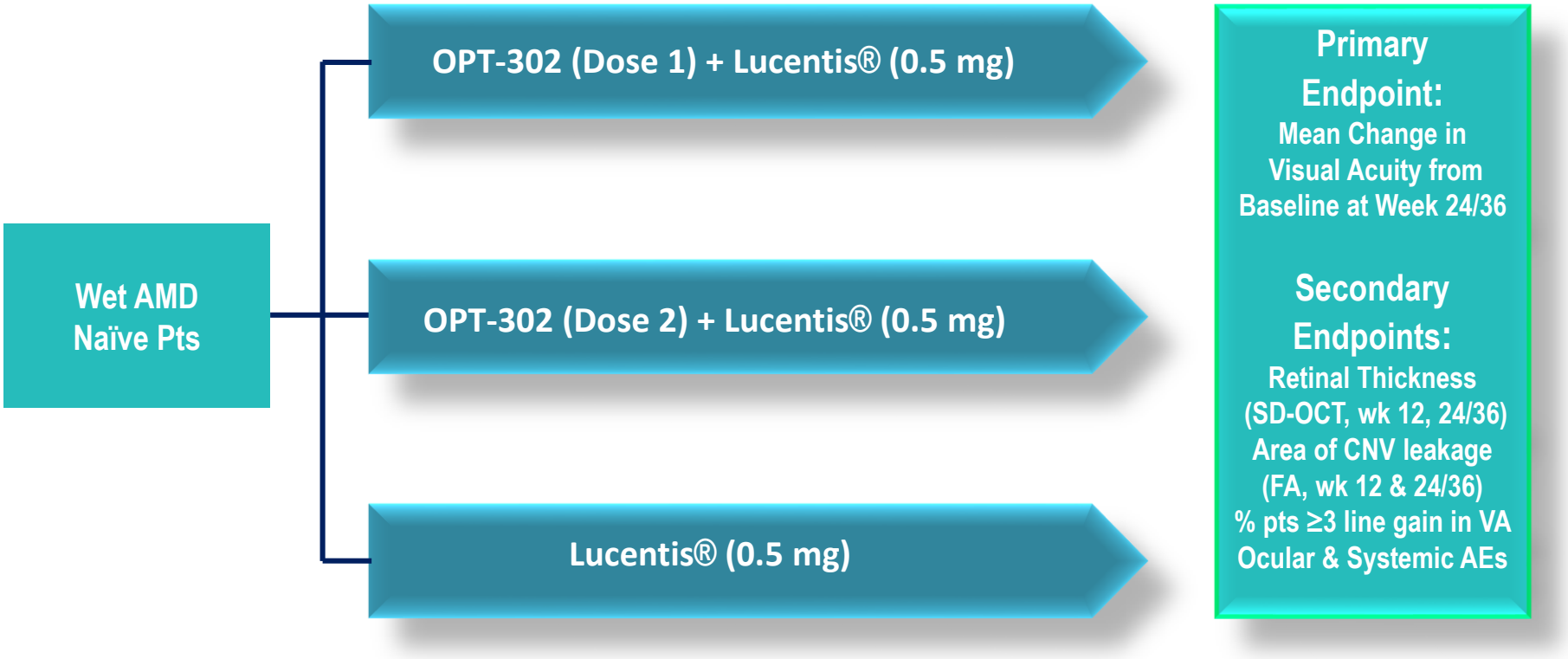
OPT-302 Proposed Phase 2B in Wet AMD

Combination OPT-302 + Lucentis® vs Lucentis®



All treatment arms : IVT dosing at every 4 weeks (x 6 or x9)

- *Phase 2b Prospective, randomized, controlled trial*
- *3 arms (randomized 1:1:1)*
- *Primary Analysis at 6 months*

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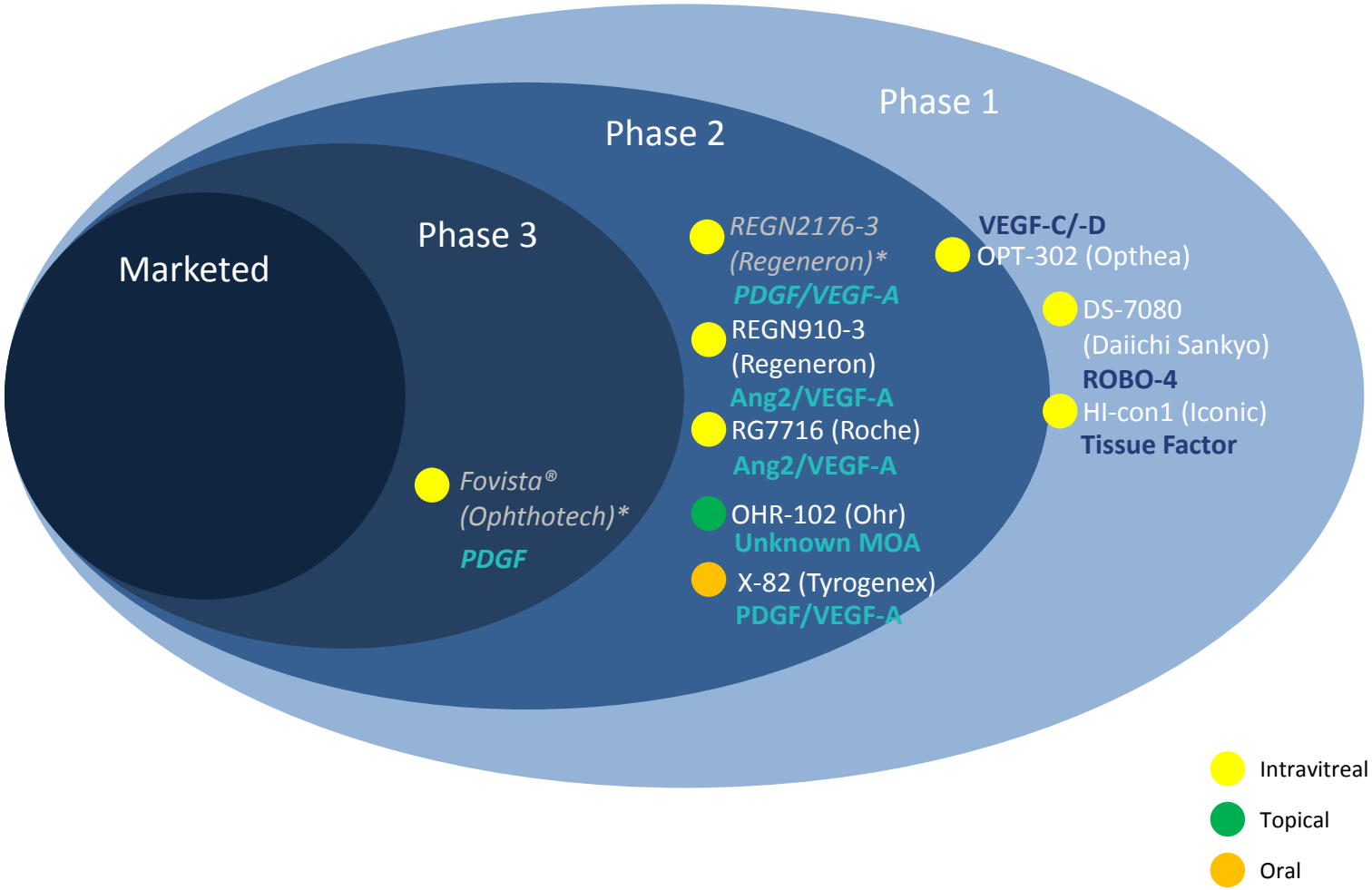
Wet AMD Landscape of Novel Targets (non-VEGF-A)



** Failed to meet primary endpoint in Phase 3 or Phase 2 wet AMD clinical trial/s*

OPT-302 Intellectual Property

Summary covering sVEGFR-3 for eye disease

COMPOSITION OF MATTER	TERM
Covering sVEGFR-3 (inc. OPT-302) • Granted Patents: Europe, Japan, Canada, Australia • Granted Patent: USA	 2022 2026
Covering OPT-302 • Recently filed new specific composition of matter PCT international patent application	~2034
'USE' PATENT	
• US Patent granted covering generic use of sVEGFR-3 capable of binding VEGF-C to inhibit blood vessels in mammal having disease characterised by expression of VEGFR-3 in blood vessels	2023
PATENT TERM EXTENSION/EXCLUSIVITY	
+5 years under patent term extension OPT-302 entitled to data exclusivity (DE) and market exclusivity (ME) in many jurisdictions, eg. • US (12 years DE for biologics) • Europe (10 years made up of 8 years DE + 2 years ME) • Japan (up to 8 years de facto DE) • South Korea (5 years DE) • Canada (up to 8 years incl. up to 6 years DE + 2 years ME) • Australia (5 years DE)	

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OPT-302 Program Highlights

- OPT-302 has broad development potential in a range of eye diseases
- Targets validated pathway involved in wet AMD progression and mechanism of escape from existing therapies that is differentiated to a-VEGF-A therapies
- Large unmet medical need for wet AMD, current treatments only target VEGF-A
- OPT-302 met primary objective of Phase 1 study (safe & well tolerated)
- Evidence of clinical activity:
  - Naïve pts: promising results suggest OPT-302 + Lucentis® may lead to improved outcomes over Lucentis® alone
  - Prior Tx pts: improved outcomes in difficult to treat, sub-responsive pts
  - Monotherapy: evidence of clinical activity without background std of care
- A consistency of responses in pts:
  - With different treatment histories
  - Across various secondary outcome measures (VA, OCT)
- Data warrants further investigation of OPT-302 in Ph2B randomised, controlled trial
- Phase 2A fully recruited, primary data analysis 1Q'17
- Phase 2B wet AMD trial to initiate in 2017

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Snapshot

Asset	OPT-302: - Soluble form VEGFR-3 - A 'trap' similar to Eylea™ with distinct MOA
Mechanism	Inhibits VEGF-C & VEGF-D - Anti-angiogenic - Inhibits vascular leakage
Rationale	Targets over-lapping & distinct pathways to VEGF-A inhibitors - Validated VEGFR-2 pathway via VEGF-C/-D inhibition - Also blocks VEGFR-3 pathway that is VEGF-A independent
Indication	Wet AMD - Leading cause blindness in Western world in adults > 50 yrs - ~1.8M people in US have wet AMD*
Market Opp.	~USD 10bn worldwide
Unmet Medical Need	~50% of people receiving Lucentis™/Eylea™ do not experience a significant gain in vision Majority (50-70%) continue to have retinal fluid
Existing Therapies	Target VEGF-A, but not VEGF-C/-D - Include blockbusters Lucentis®, Eylea®, off-label Avastin®
Landscape	OPT-302 potentially complementary to existing and emerging agents, incl. PDGFR, Ang2 inhibitors, based on MOA. Limited number of novel targeted therapies in development.
Intellectual Property	Granted Composition of Matter patients (2022-2026) Composition of Matter patent pending (~2034) Granted 'Use' Patent (2023, US). Additional patent term extensions.

Strategy	OPT-302 + a-VEGF-A achieves more complete blockade of VEGF pathway - Targets a mechanism of a-VEGF-A resistance - Trial investigating monotherapy and combination safety/activity
Preclinical Data	As monotherapy, reduces wet AMD lesion size and leakage to comparable extent as Eylea® Combination therapy significantly more effective than either agent alone
Clinical Trial	Phase 1/2A trial ongoing in US under IND - Primary Objective: Safety (met for Ph 1 over 3 month assessment) - **Early evidence of clinical activity in Ph1** - Routine, non-invasive endpts to monitor clinical activity, clear reg path
Near Term Clinical Milestones	Phase 1/2A Primary Data Analysis 1Q'17 Initiation of Phase 2B wAMD trial in 2017
Program	Potential to develop OPT-302 for other eye diseases, incl. DME
Company	Opthea raised funds (A$17.4m) for wet AMD program from US/EU/AUS healthcare investors (Nov'14) Significant value appreciation since fundraising

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