CORPORATE PRESENTATION

January 2020

UNITY
BIOTECHNOLOGY

SPECIAL NOTE REGARDING FORWARD-LOOKING STATEMENTS

This presentation and the accompanying oral commentary contain forward-looking statements, including: statements related to our understanding of cellular senescence and the role cellular senescence plays in diseases of aging; our expectations regarding the potential benefits, activity, effectiveness and safety of senolytic drug candidates; the status of our our preclinical, clinical and regulatory development plans and pipeline; our expectations with regard to the timing and results of our clinical studies; and our expectations with regard to our ability to acquire, discover and develop additional drug candidates and advance such drug candidates into, and successfully complete, clinical studies. These statements involve substantial known and unknown risks, uncertainties and other factors that may cause our actual results, levels of activity, performance or achievements to be materially different from the information expressed or implied by these forward-looking statements. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make. The forward-looking statements in this presentation represent our views as of the date of this presentation. We anticipate that subsequent events and developments will cause our views to change. However, while we may elect to update these forward-looking statements at some point in the future, we have no current intention of doing so except to the extent required by applicable law. You should, therefore, not rely on these forward-looking statements as representing our views as of any date subsequent to the date of this presentation.

For a further description of the risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to the business of the Company in general, see UNITY's most recently filed Quarterly Report on Form 10-Q for the quarter ended September 30, 2019, filed with the Securities and Exchange Commission on November 6, 2019, as well as other documents that may be filed by UNITY from time to time with the Securities and Exchange Commission.

This presentation concerns drug candidates that are under clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration. They are currently limited by Federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

At UNITY we are attacking the fundamental biology of aging to develop medicines that:

AGE-RELATED DISEASES

HEALTHSPAN

A NEW THERAPEUTIC APPROACH

LEADER IN CELLULAR SENESCENCE	• Emerging research shows that senescent cells are implicated in **multiple diseases of aging** • Selectively eliminating senescent cells targets a **root cause** of age-related disease • Building on our **deep understanding of senescence biology** and intellectual property, our approach generates potent and selective senolytic drug candidates • Pursuing diseases with **established endpoints and regulatory pathways**
CLINICAL EVIDENCE	• Phase 1 study showed **clear and substantial improvements** in **OA pain and function** • Initiated Phase 2 study of UBX0101 in 4Q19; 12 week data expected 2H20; 24 week data expected 1H21 • Initiation of Phase 1b study for higher dose and repeat dose UBX0101 in 1H20 with data expected in 2H20 • Ophthalmology IND expected in early 2020; Safety data in 2H20; Efficacy data in 1H21
EXPERIENCED TEAM	• Seasoned executive team with broad biotech experience • Strong track record of delivering for patients and investors
FINANCIAL POSITION	• Cash equivalents and investments balance of $120.3 million as of September 30, 2019 • Cash runway into second half of 2021

EMERGENCE OF NEW THERAPEUTIC APPROACH

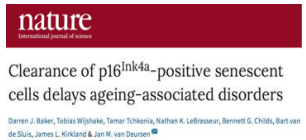
Leveraging cellular senescence biology

1960s — DISCOVERY OF CELLULAR SENESCENCE

1990s — IDENTIFICATION OF SENESCENCE BIOMARKER

2000s — LINK TO AGING PHENOTYPES & IDENTIFICATION OF SASP

2011 — SENOLYSIS AS AN APPROACH TO TREAT DISEASES OF AGING



FROM SCIENTIFIC INSIGHT TO THERAPEUTIC BENEFIT

nature
International weekly journal of science

Letter | Published: 02 November 2011

Clearance of p16^{Ink4a}-positive senescent cells delays ageing-associated disorders

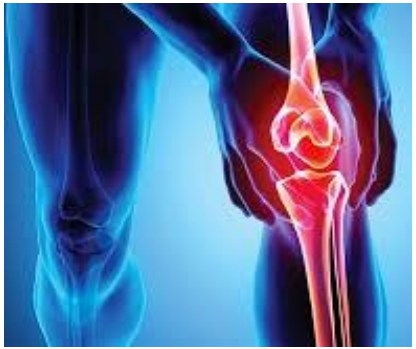
Darren J. Baker, Tobias Wijshake, Tamar Tchkonia, Nathan K. LeBrasseur, Bennett G. Childs, Bart van de Sluis, James L. Kirkland & Jan M. van Deursen

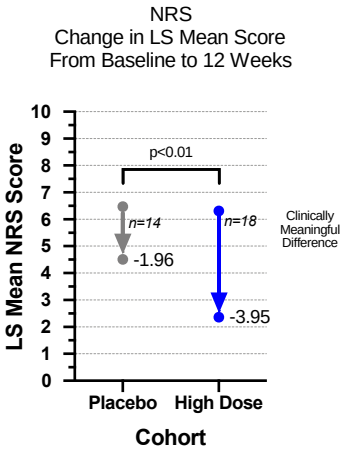
MAYO CLINIC

BUCK INSTITUTE FOR RESEARCH ON AGING

UNITY BIOTECHNOLOGY

Phase 1 clinical study in osteoarthritis showed improvements in pain and function

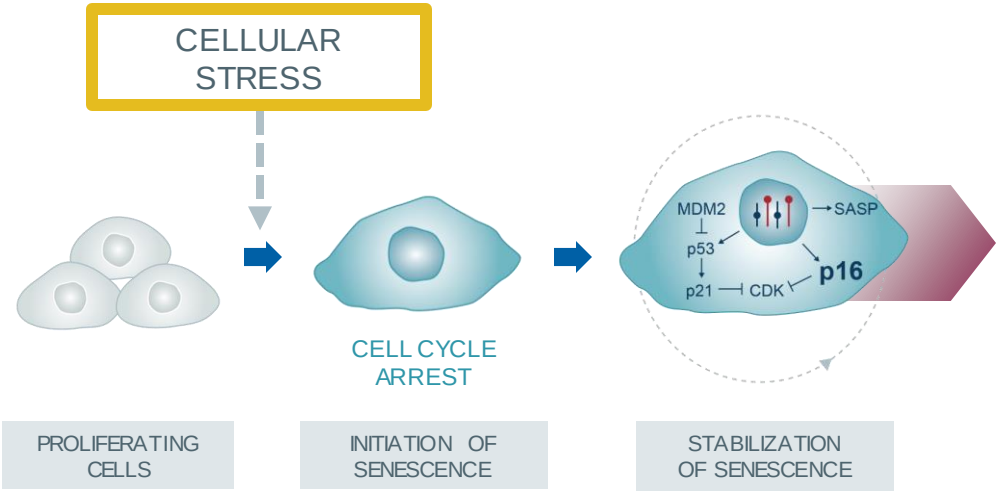




2011 → **2019**

UNITY BIOTECHNOLOGY

SENESCENT CELLS ARE IMPLICATED IN MULTIPLE DISEASES OF AGING



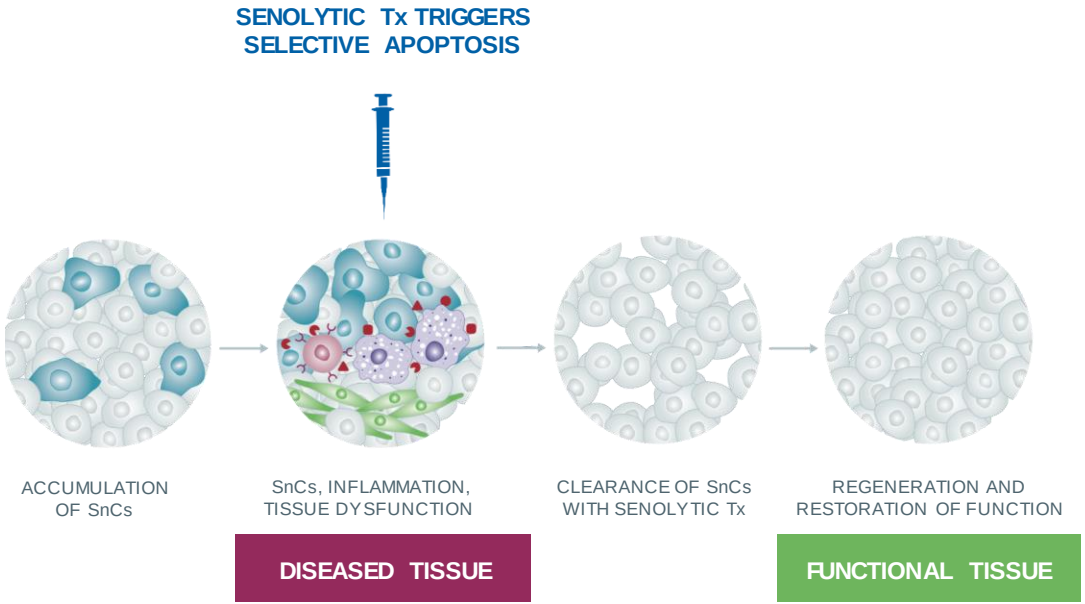
SASP

SASP FACTORS	DISEASES
INFLAMMATORY FACTORS (MMP1, MMP3, TNF-α, IL-1β, prostaglandins)	**INFLAMMATORY DISEASES** Osteoarthritis
PRO-FIBROTIC FACTORS (TGFβ1, TGFβ2, CTGF, TIMP-1, MCP-1)	**FIBROTIC DISEASES** Idiopathic Pulmonary Fibrosis, Chronic Kidney Disease, Polycystic Kidney Disease, Primary Sclerosing Cholangitis, Primary Biliary Cholangitis, NASH
GROWTH FACTORS (VEGF-α, IL-6, IL-8, Pai-1)	**VASCULAR DISEASES** AMD, Diabetic Retinopathy

Senescent cells represent a potential *root cause* of diseases of aging

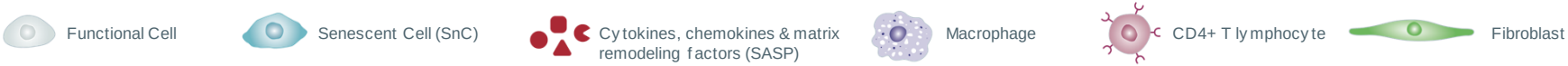
SASP = senescence-associated secretory phenotype

THE UNITY THERAPEUTIC APPROACH



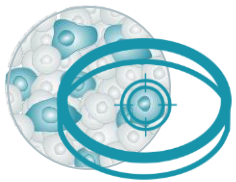
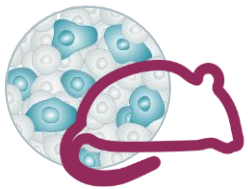
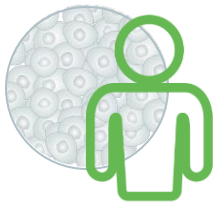
POTENTIAL CLINICAL ADVANTAGES

- Improved magnitude of effect by eliminating source of multiple factors
- Longer duration of therapeutic benefit
- Reduced frequency of dosing
- Local administration
- Disease modification



SASP = senescence-associated secretory phenotype

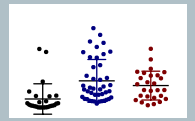
OUR PLATFORM FOR GENERATING POTENT AND SELECTIVE SENOLYTIC DRUG CANDIDATES

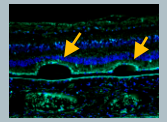





IDENTIFY

Identify SnCs in human disease:

- Cell type(s)
- Tissue location
- SnC burden
- SASP signature

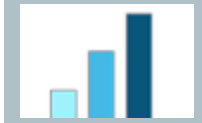

SnC presence

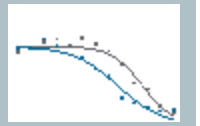

SnC location

TARGET

Eliminate SnCs selectively:

- Survival pathways
- Apoptosis mechanisms: BCL-2 family, p53/MDM2

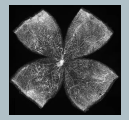

Apoptosis

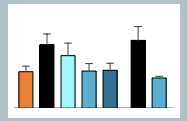

SnC elimination

IMPACT

Drive efficacy with senolytic:

- Efficacy endpoints
- SASP reduction
- Aged *in vivo* models
- Genetic models
- Disease models

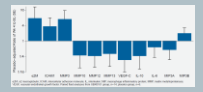

Tissue changes

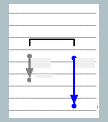

Disease impact

TRANSLATE

Senolytic impacts patient outcomes

- Safety and tolerability
- Function and symptoms
- Tissue microenvironment
- Disease modification

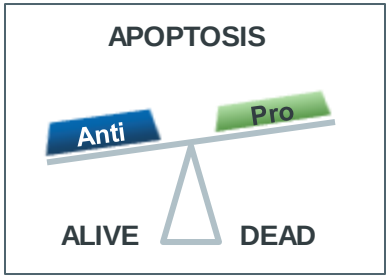

Biomarkers impact


Clinical improvement

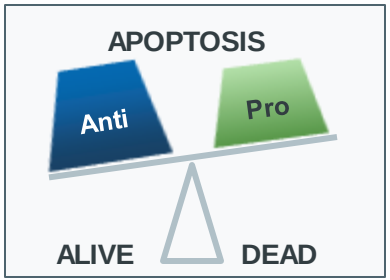
SnC = senescent cells

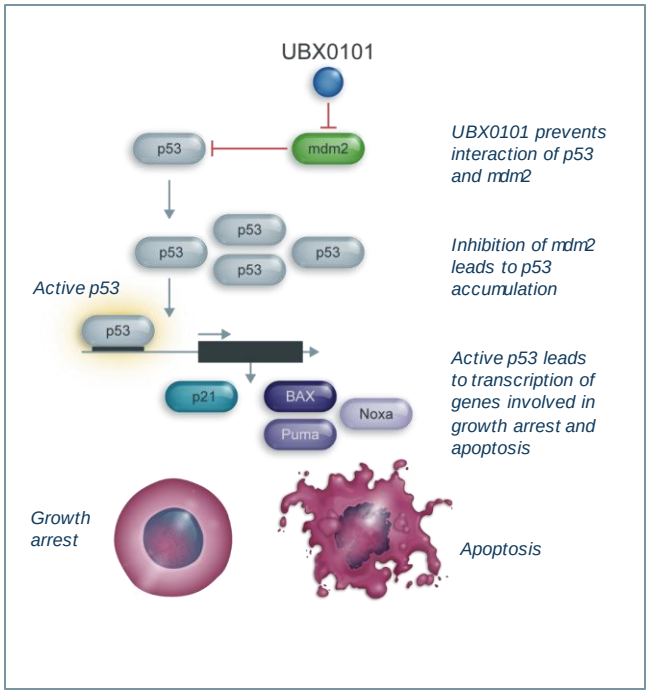
SENOLYTICS SELECTIVELY ELIMINATE SENESCENT CELLS BY TARGETING WELL-DEFINED SURVIVAL PATHWAYS

NON-SENESCENT CELL

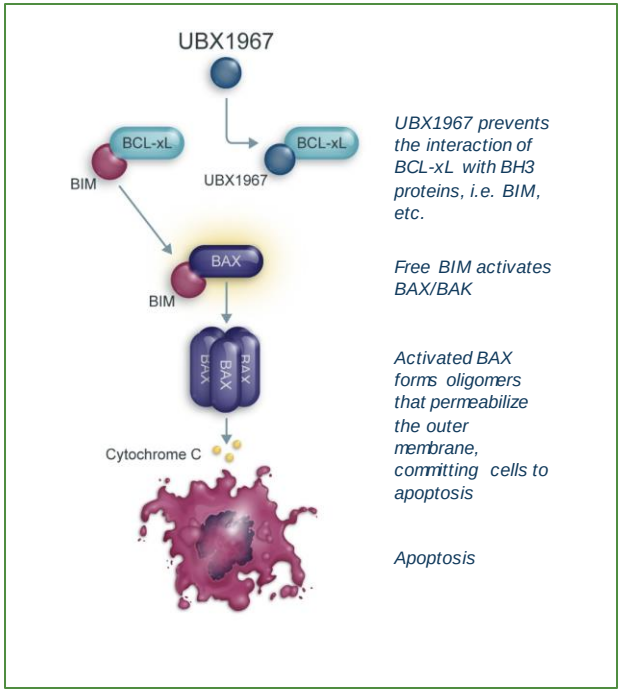


SENESCENT CELL





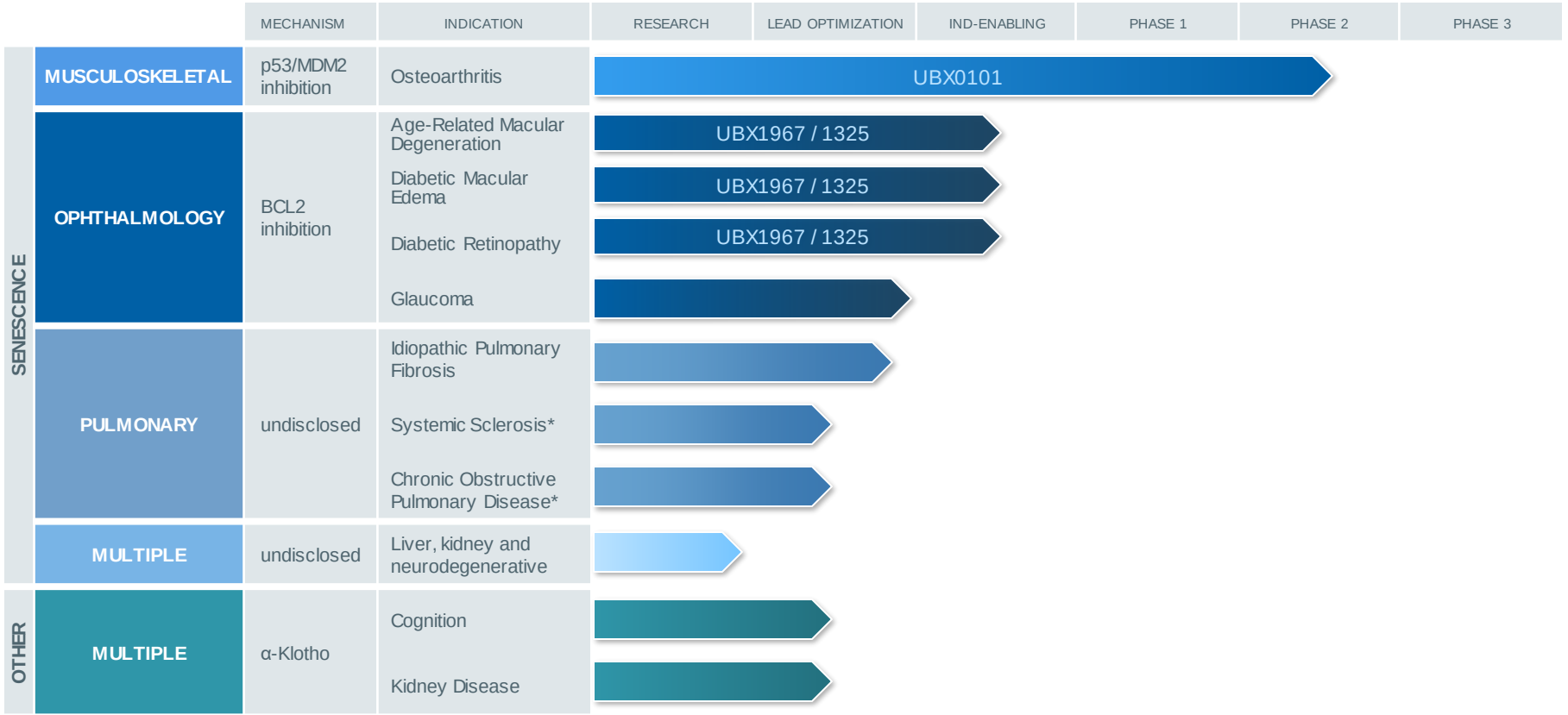
p53/mdm2



Bcl-2

UNITY PIPELINE

Pursuing broad range of diseases with established endpoints and regulatory pathways



*Following IPF Outcome

OSTEOARTHRITIS

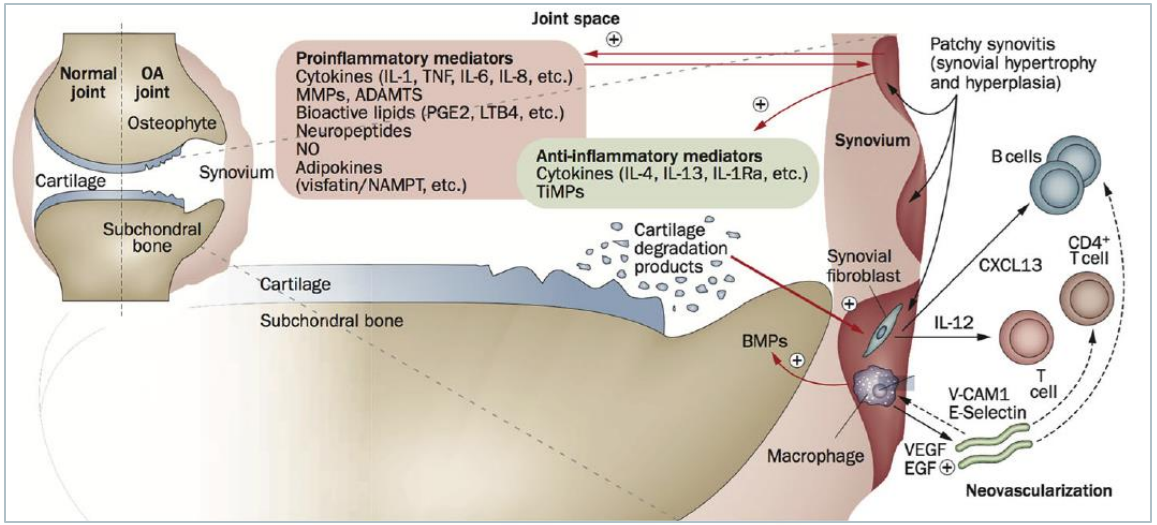
(MUSCULOSKELETAL INDICATION)

UNITY BIOTECHNOLOGY

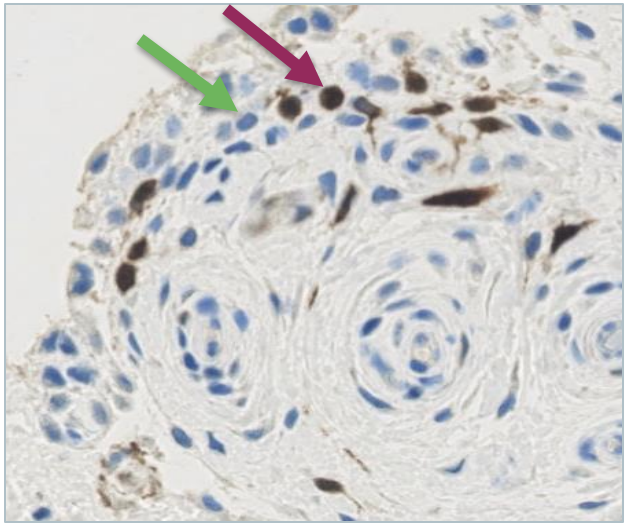
OSTEOARTHRITIS

~10-15% of population >60 years old; SoC is pain mitigation or joint replacement

OA IS BELIEVED TO BE A MULTIFACTORIAL DISEASE



In phase 0 study, **senescence burden correlated with disease severity**



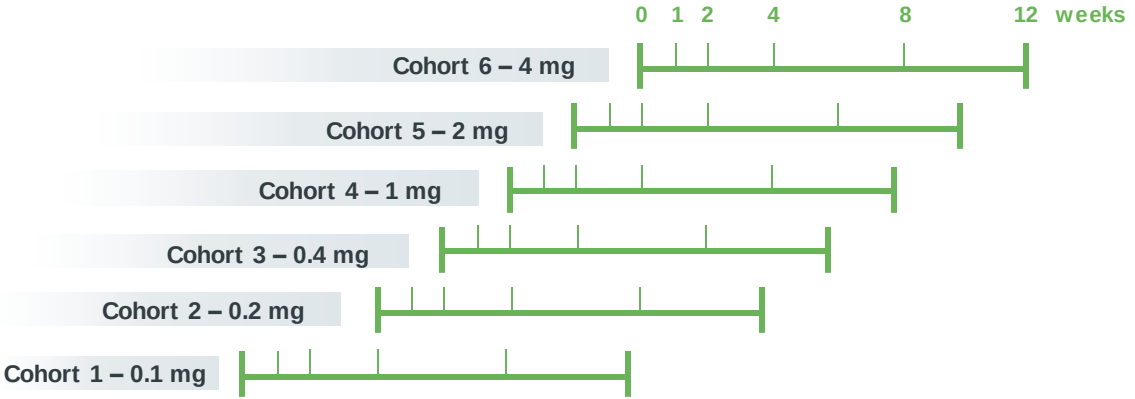
Mathiessen et al. *Arthritis Research and Therapy* 2017

A senolytic may remove source of multiple factors implicated in OA

UBX0101 PHASE 1 PROGRAM

Single Ascending Dose Study (Part A)
N=48, 3:1, Active to Placebo

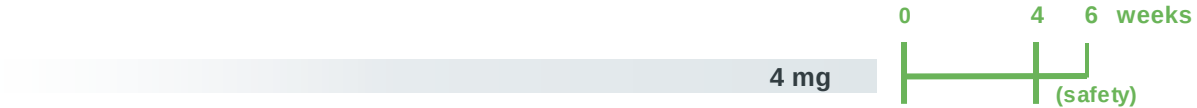
Entry Criteria:
- K-L 1-4
- NRS weekly average ≥4 and ≤ 9
- Evidence of Synovitis on MRI



Primary SAD Study
Primary Objective: Safety and tolerability to 12 weeks
Secondary Objectives: Plasma PK and 11-Point NRS daily pain assessment, WOMAC (Pain, Stiffness, Function and Total), Plasma and Synovial fluid SASP/OA biomarkers to 12 weeks

Biomarker Sub-study (Part B)
N=30, 2:1 Active to Placebo

Entry Criteria:
K-L 1-4
WOMAC-A ≥6
No NRS or MRI included



Biomarker Sub Study
Primary Objective: Safety and tolerability to 4 weeks
Secondary Objectives: Synovial fluid SASP/OA biomarkers, sparse plasma PK, WOMAC (Pain, Stiffness, Function and Total) to 4 weeks

UBX0101 WAS WELL TOLERATED

- No serious adverse events
- No AEs led to discontinuation from study
- No dose-dependence in AEs or in clinical laboratory findings
- The majority of AEs were mild (66% in Part A and 75% in Part B)

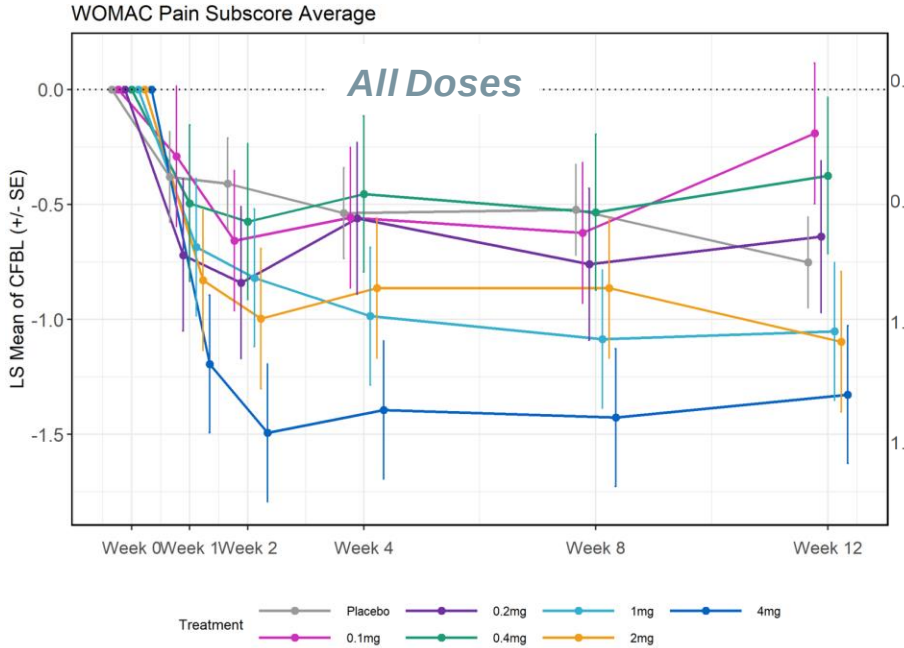
Treatment-emergent AE occurring in ≥ 2 patients

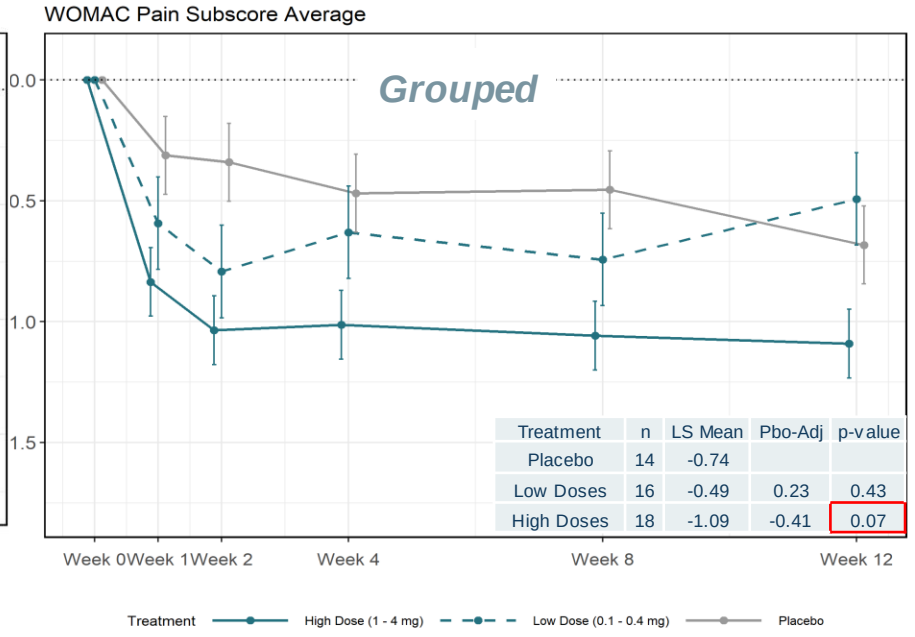
Preferred Term	Part A, 0.1- 4mg (N= 34) n (%)	Placebo (N= 14) n (%)	Part B (4 mg) (N= 20) n (%)	Placebo (N= 10) n (%)
Nasopharyngitis	2 (5.9)	1 (7.1)	0	0
Procedural pain	2 (5.9)	1 (7.1)	2 (10.0)	0
Arthralgia	3 (8.8)	1 (7.1)	0	1 (10.0)
Headache	4 (11.8)	1 (7.1)	0	0

SINGLE DOSE OF UBX0101 DECREASED PAIN

WOMAC-A

5 item, 0-4 point scale where a **0.5 point change** is considered clinically meaningful.





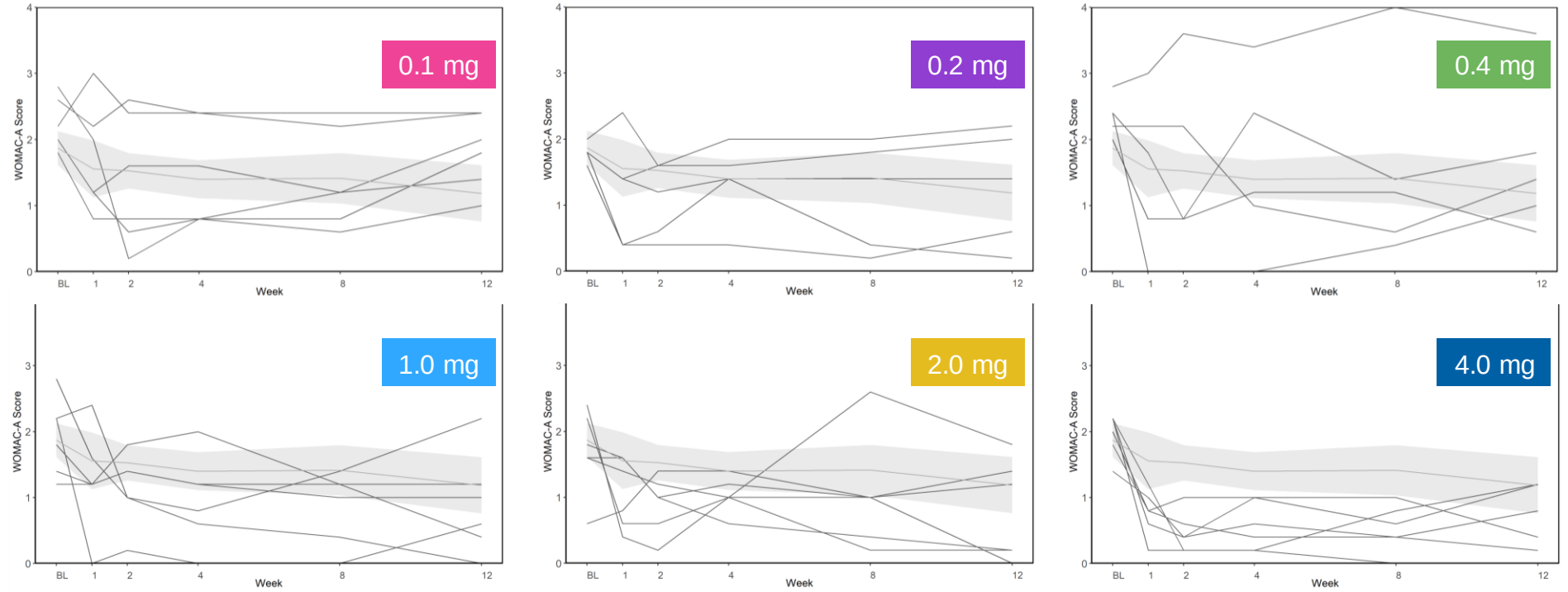
Treatment	n	LS Mean	Pbo-Adj	p-value
Placebo	14	-0.74		
Low Doses	16	-0.49	0.23	0.43
High Doses	18	-1.09	-0.41	0.07

Durable, dose-dependent and substantial effect

SINGLE DOSE OF UBX0101 DECREASED PAIN

WOMAC-A

5 item, 0-4 point scale where a **0.5 point change** is considered clinically meaningful.

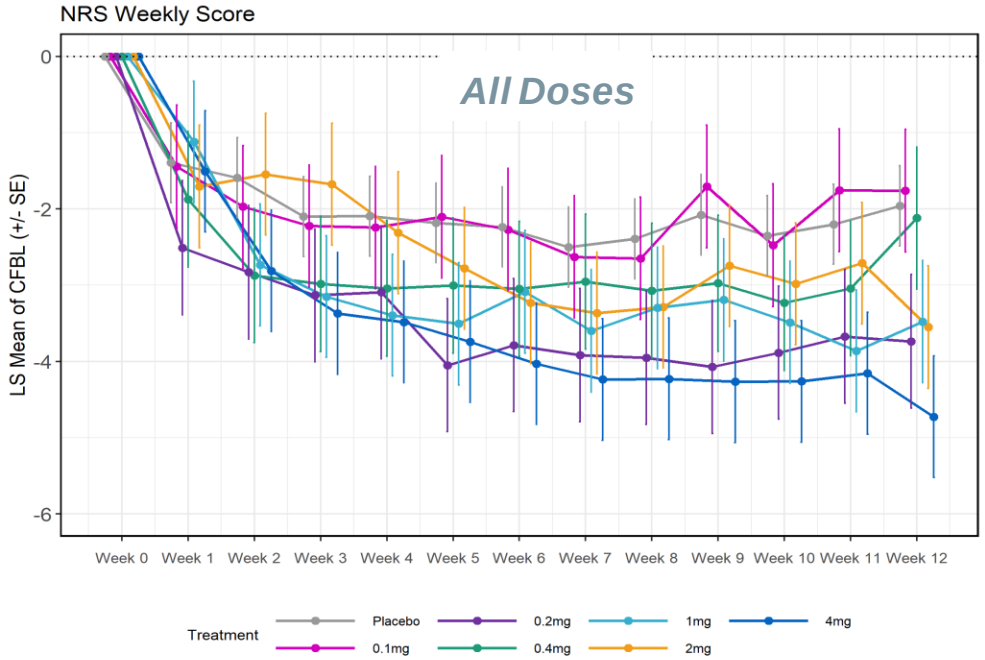


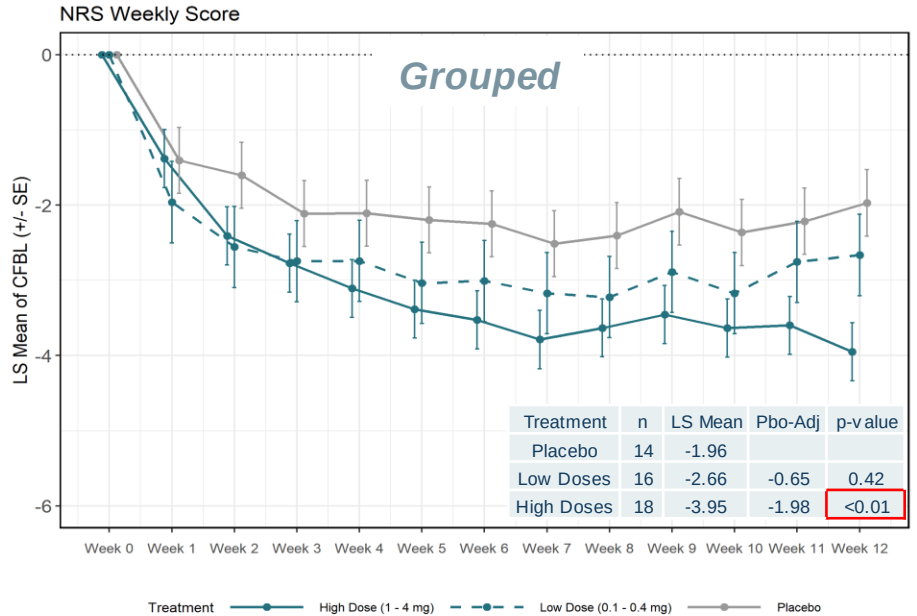
INDIVIDUAL VARIABILITY DECREASES AS DOSE INCREASES

SINGLE DOSE OF UBX0101 DECREASED PAIN

Numerical Rating Scale (NRS)

0-10 point scale where a **2 point change** is considered clinically meaningful.





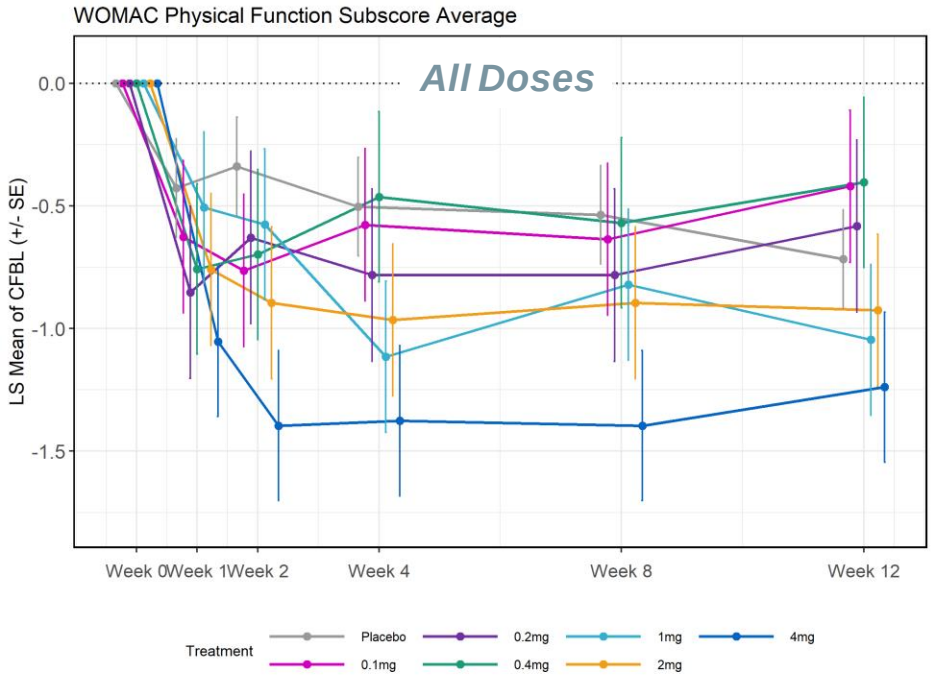
Treatment	n	LS Mean	Pbo-Adj	p-value
Placebo	14	-1.96		
Low Doses	16	-2.66	-0.65	0.42
High Doses	18	-3.95	-1.98	<0.01

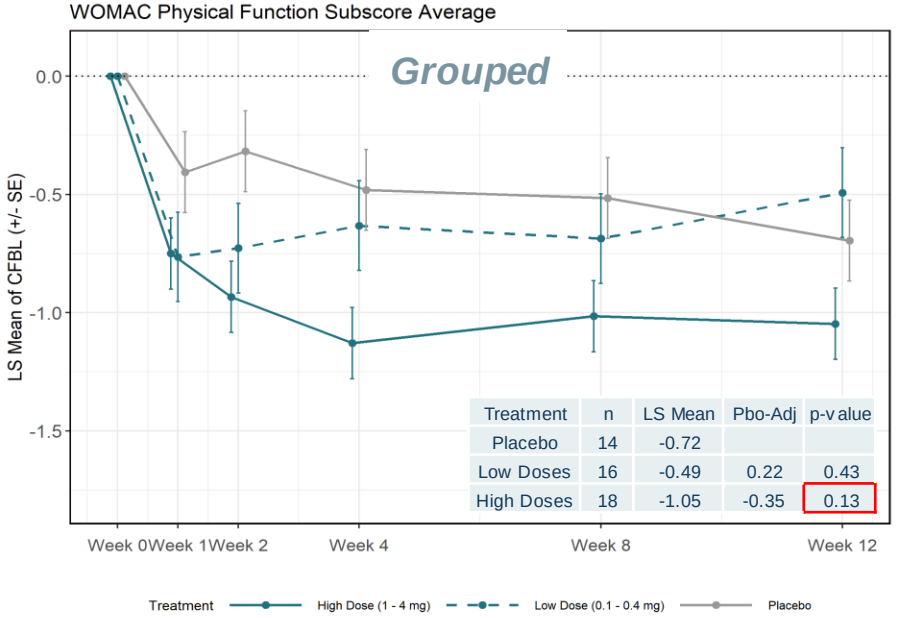
Durable, dose-dependent and substantial effect

SINGLE DOSE OF UBX0101 IMPROVED FUNCTION

WOMAC-C

17 item, 0-4 point scale where a **0.3 point change** is considered clinically meaningful.



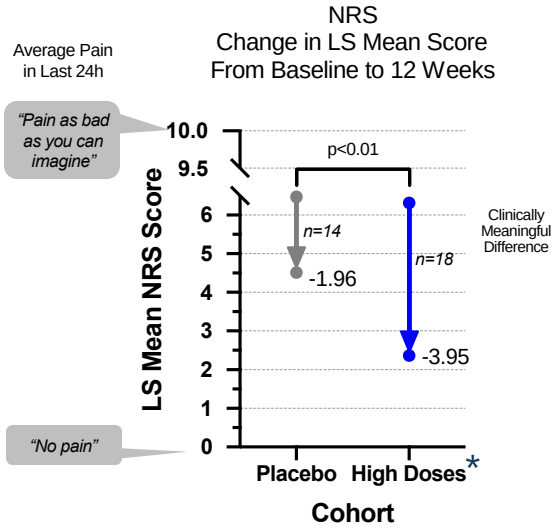


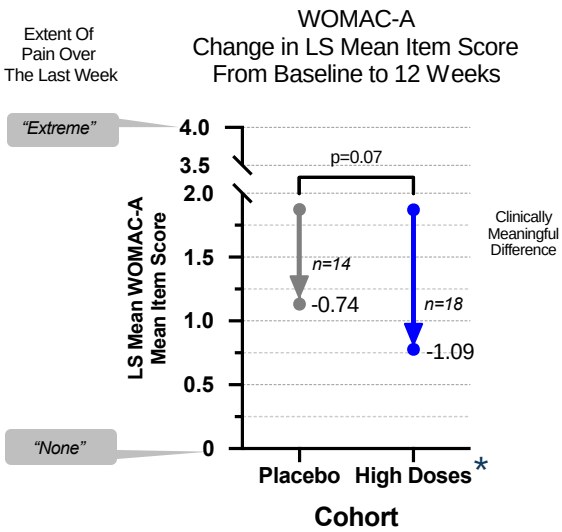
Treatment	n	LS Mean	Pbo-Adj	p-value
Placebo	14	-0.72		
Low Doses	16	-0.49	0.22	0.43
High Doses	18	-1.05	-0.35	0.13

Durable, dose-dependent and substantial effect

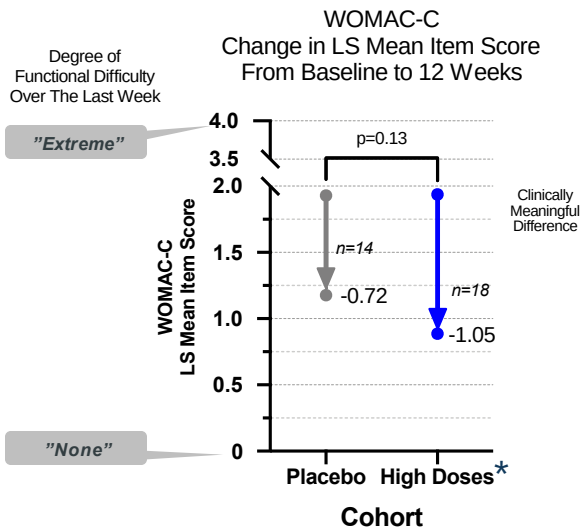
SINGLE DOSE OF UBX0101 IMPACTED PAIN AND FUNCTION AT 12 WEEKS

Pain Instruments





Physical Function Instrument



* High Doses = 1, 2, and 4mg

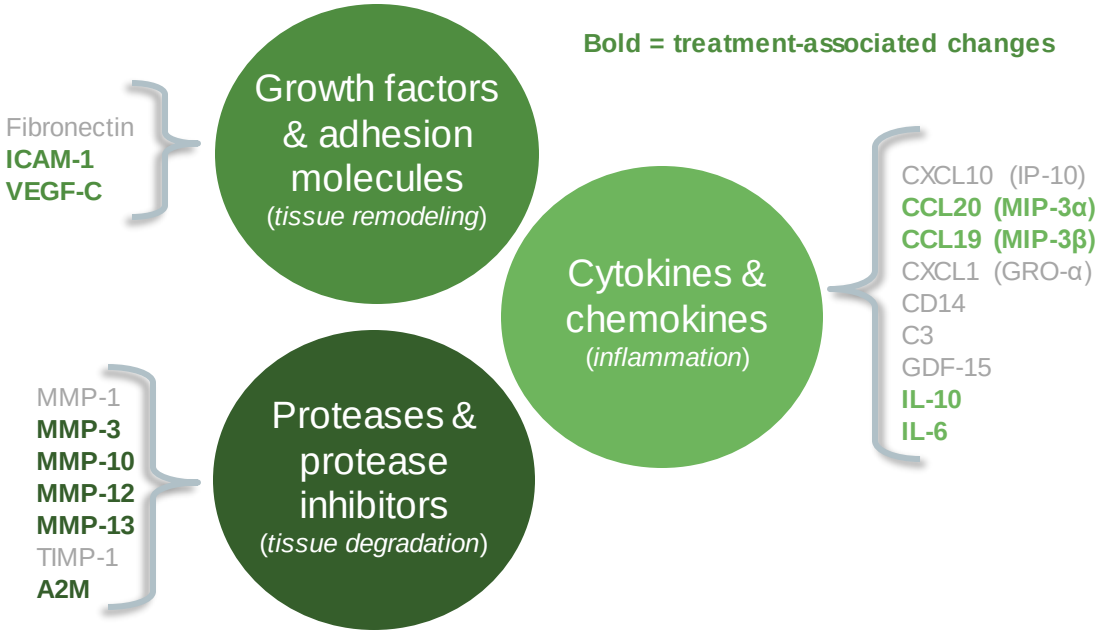
Durable, dose-dependent and substantial effect across NRS, WOMAC-A and WOMAC-C

PHASE 1 MEASUREMENT OF SASP / OA BIOMARKERS

Data is supportive of a senolytic mechanism

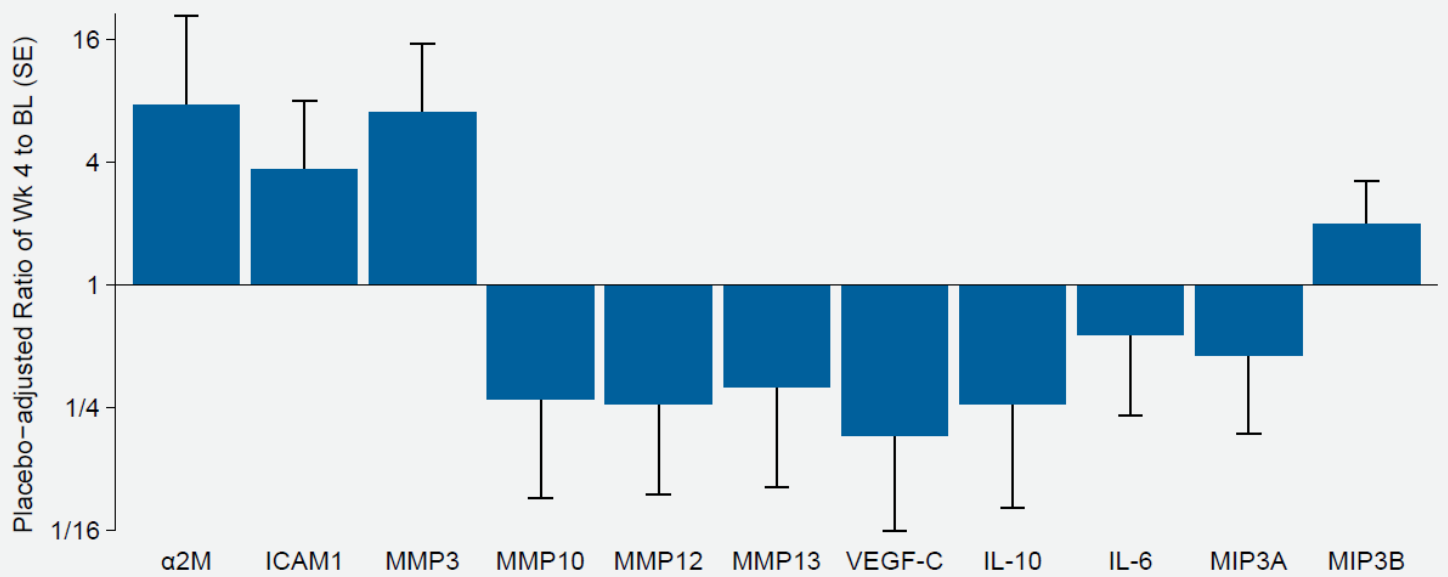
Selection Process

- **Phase 0 OA biomarker study – factors analyzed in synovial fluid/tissue for correlation with disease**
- **Extensive literature review**
- **Pre-clinical data (UNITY and external)**
- **OA and senescence disease state knowledge**



OA is believed to be a heterogeneous and multifactorial disease; UBX0101 injection altered joint environment

4 WEEK SYNOVIAL FLUID BIOMARKER DATA IS SUPPORTIVE OF A SENOLYTIC MECHANISM

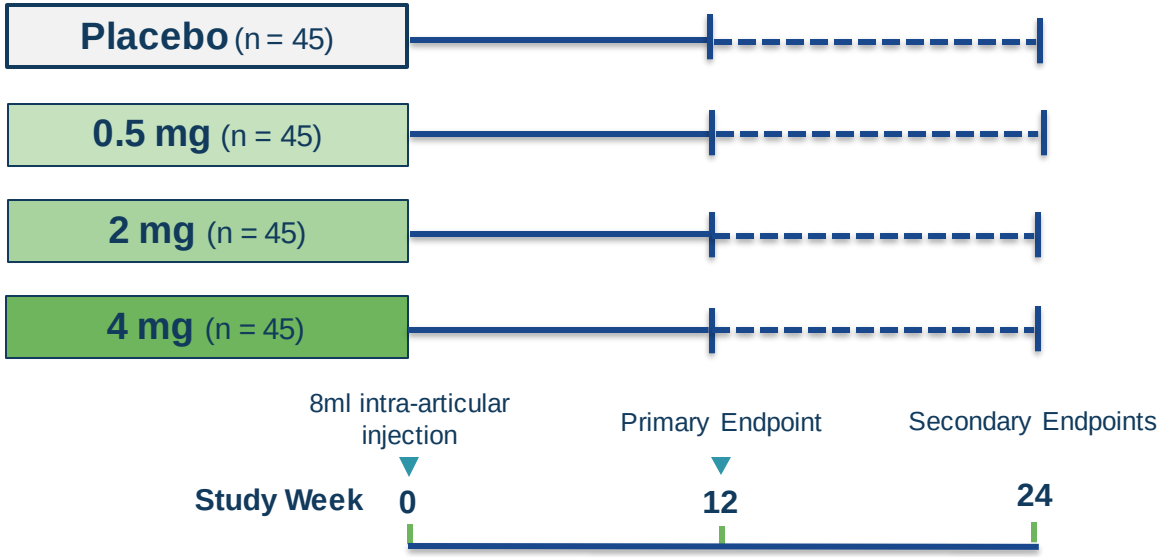


α2M, α2 macroglobulin; ICAM, intercellular adhesion molecule; IL, interleukin; MIP, macrophage inflammatory protein; MMP, matrix metalloproteinase; VEGF, vascular endothelial growth factor. Paired fluid analyses from UBX0101 group, n=14; placebo group, n=6.

UBX0101 PHASE 2 STUDY DESIGN

Designed to substantiate Phase 1 efficacy and explore duration and disease modification

A randomized, double-blind, placebo-controlled study evaluating three doses of UBX0101 administered via a single intra-articular injection



Primary Measure

- Pain by WOMAC-A at 12 weeks

Secondary Measures:

- Pain by WOMAC-A at 24 weeks
- Pain by NRS at 12 & 24 weeks
- Function by WOMAC-C at 12 & 24 weeks
- Safety & tolerability

Additional Phase 1b studies will explore higher and repeat doses

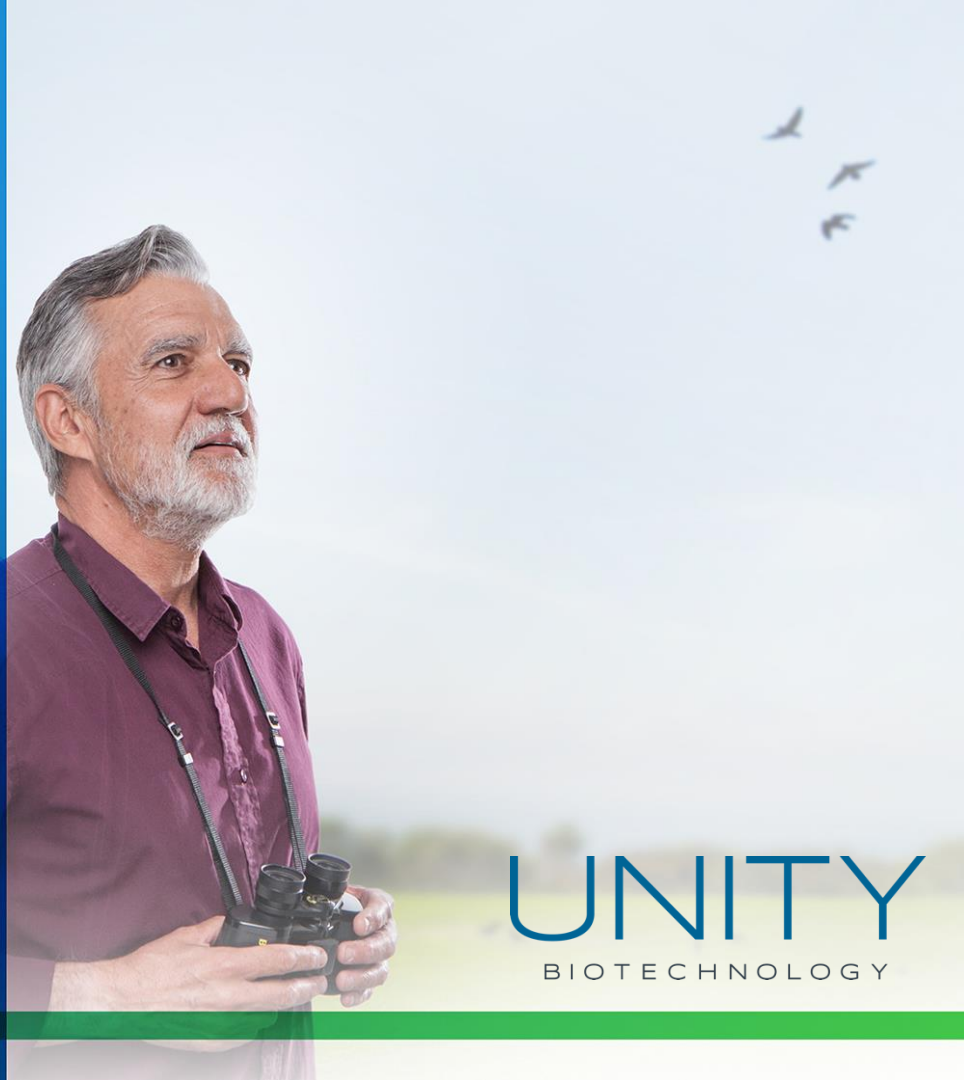
POTENTIAL DIFFERENTIATING FEATURES OF UBX0101 IN OA

1. **Novel MOA**: eliminates SnCs → potential root cause of disease
2. **Large-Magnitude Effect**: Clinically meaningful impact on pain and function
3. **Durability** → sustained effect to 12 weeks in Phase 1 study

Phase 2 study designed to substantiate effects observed in Phase 1

OPHTHALMOLOGY

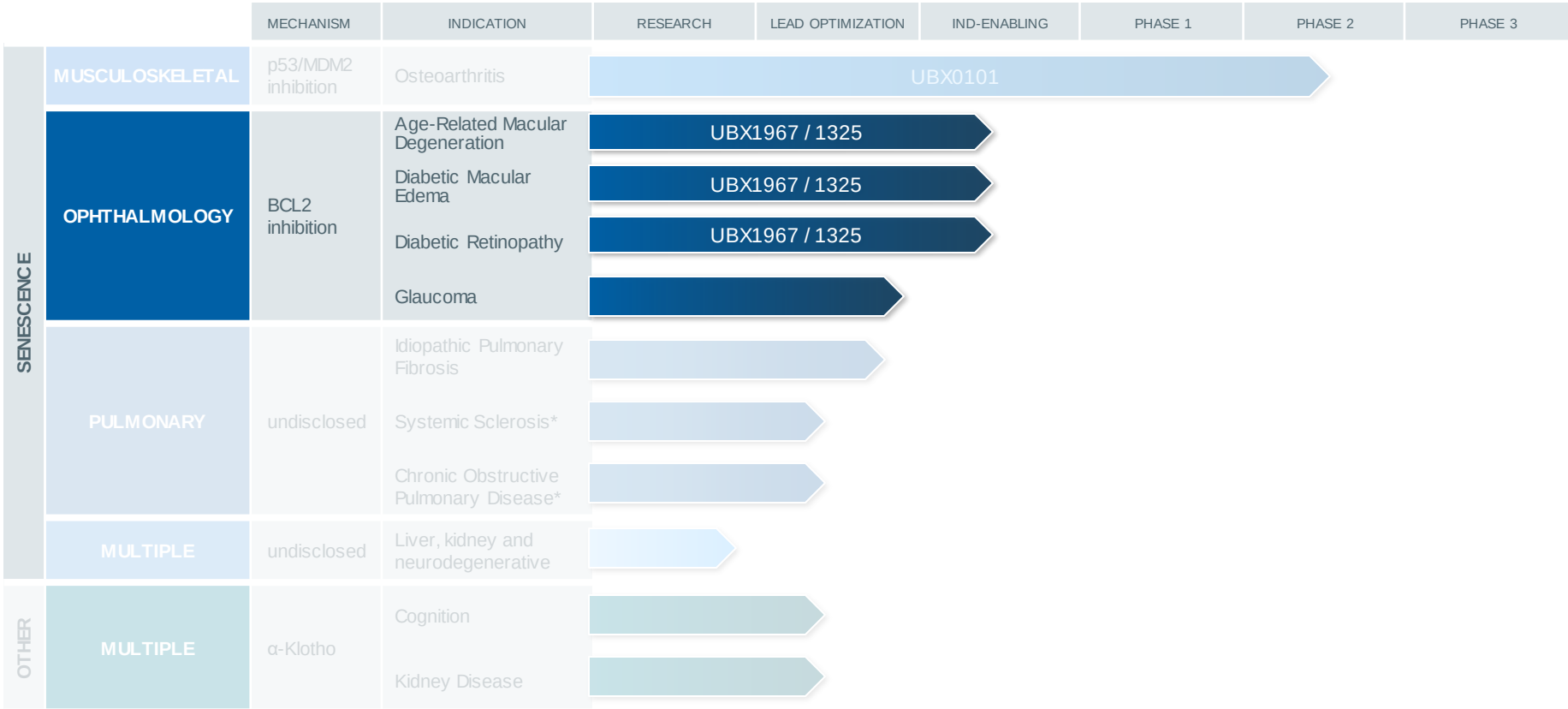
(AGE-RELATED EYE DISEASE INDICATIONS)



UNITY
BIOTECHNOLOGY

UNITY PIPELINE

Pursuing broad range of diseases with established endpoints and regulatory pathways



*Following IPF Outcome

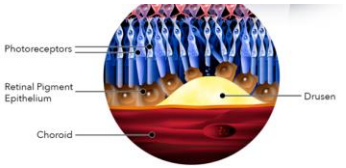
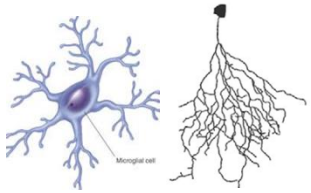
AGE-RELATED EYE DISEASES ARE SIGNIFICANT PUBLIC HEALTH BURDENS

Leading causes of visual disability in aging populations, which may be treatable with a senolytic

	AGE-RELATED MACULAR DEGENERATION (AMD)	DIABETIC RETINOPATHY (DR)	DIABETIC MACULAR EDEMA (DME)
GLOBAL IMPACT	• ***Leading cause of visual disability*** in industrialized world; ***3rd leading cause*** globally • Aging is the greatest risk factor	• ***Leading cause of vision loss*** in middle-aged and elderly • Impacts ***8% of people*** >65 years old	• A manifestation of DR that is the ***primary cause of vision loss for people with diabetes***
GLOBAL PREVALANCE	• **170M** people affected • Expected to increase to **285M+** by 2040	• **90M+** people affected; **28M** with vision-threatening disease stages • ***~33% of people with diabetes*** have signs of DR	• **20M+** people affected
CURRENT TREATMENTS	• Anti-VEGF agents, laser therapy	• Diabetes control, anti-VEGF agents, laser photocoagulation	• Diabetes control, corticosteroids, anti-VEGF agents, laser photocoagulation
GLOBAL MARKET SIZE	• ***>$8B in global annual anti-VEGF sales***		

ROLE OF SENESCENCE IN AGE-RELATED EYE DISEASE

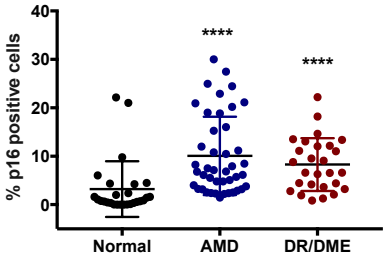
SnCs accumulate in the retina, potentially contributing to disease phenotypes

AMD		DR & DME
SnCs accumulate with with age in the retina retinal pigment epithelium (RPE) layer		**senescent cell**		**SnCs** accumulate in the retina with age & diabetic disease
SASP → choroidal remodeling & RPE dysfunction → atrophy	SASP FACTORS (VEGF-α, IP-10)	**senescence secretome**	SASP FACTORS (PAI-1, IL-6, IL-8, VEGF)	**SASP** → ocular inflammation, abnormal blood vessel growth
Disease → central vision loss		**disease symptoms**		**Disease** → vision loss

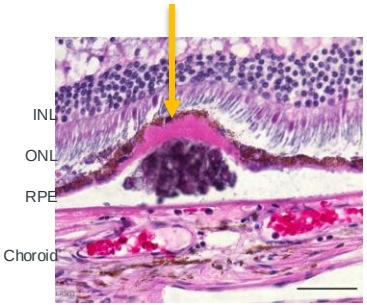
Oubaha et al., Senescence-associated secretory phenotype contributes to pathological angiogenesis in retinopathy, Sci. Transl. Med. 8, 362ra144 (2016)

SENESCENCE BURDEN IN AMD AND DR/DME

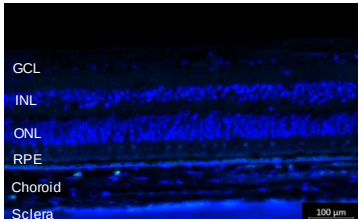
SNC BURDEN



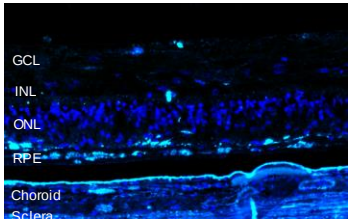
drusen



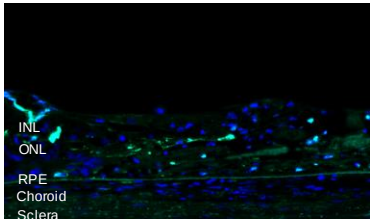
NORMAL (71F)



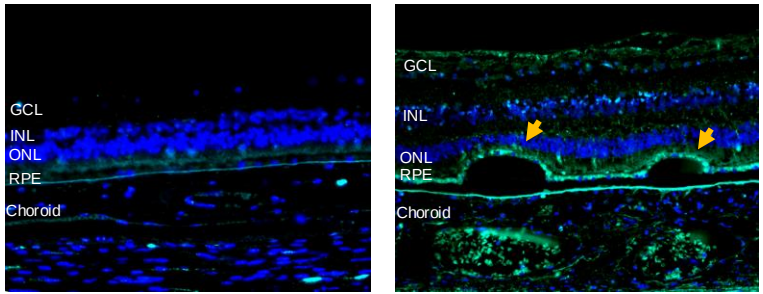
AMD (93M)



DR (64M)



INTERMEDIATE AMD (86F)



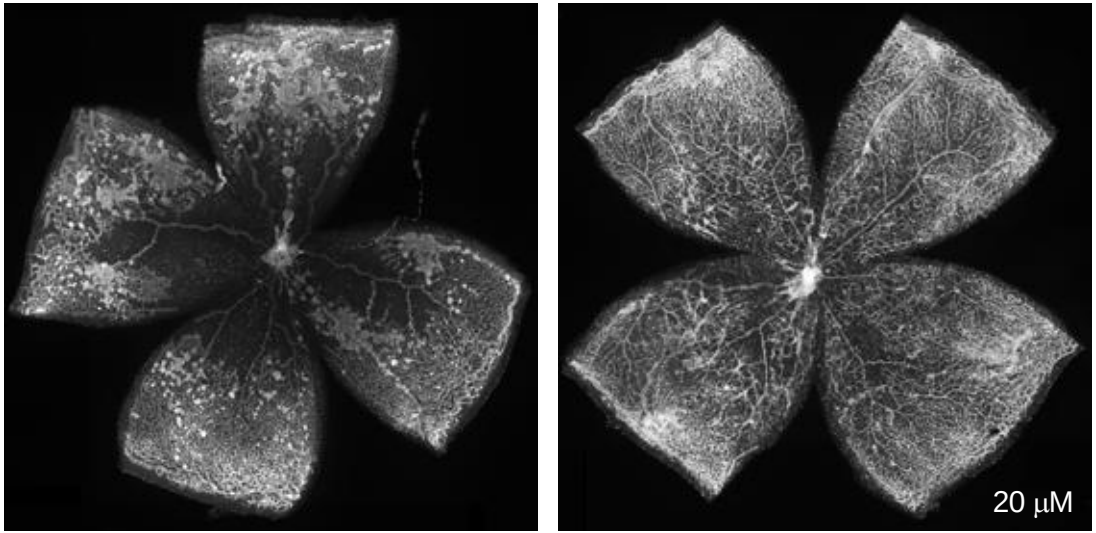
- Age-related eye diseases are multifactorial
- SnC burden increases with disease stage
- DR/DME patients show SnC in the retina and Choroid

Faidley EA (2008)

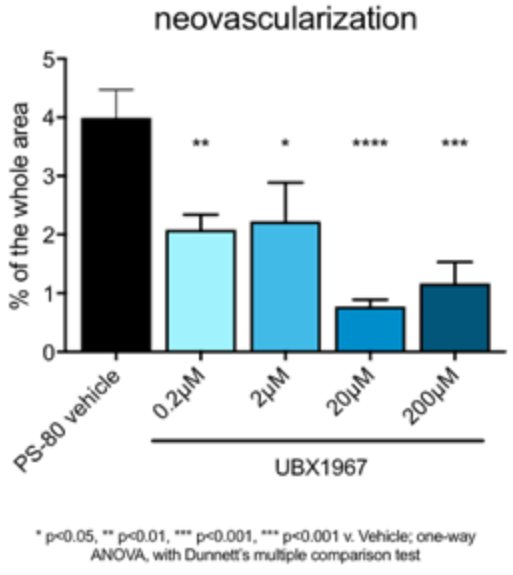
UBX1967 DEMONSTRATES EFFICACY IN MOUSE OIR

Oxygen induced retinopathy (OIR) model

Vehicle	UBX1967



Improves Retinal Vasculature



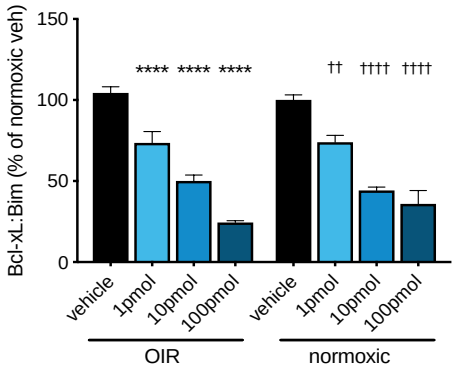
Intravitreal dosing improves retinal vasculature

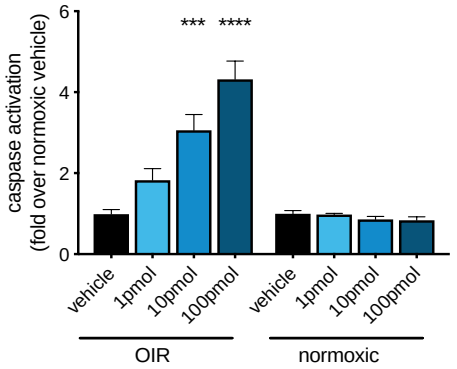
UBX1967 ENGAGES BCL-xL AND SELECTIVELY PROMOTES APOPTOSIS IN HYPEROXIC MOUSE RETINA

TARGET ENGAGEMENT

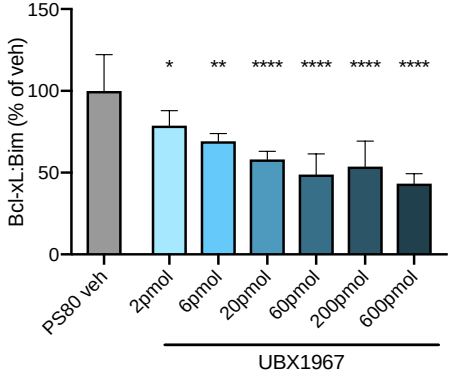
MECHANISM ENGAGEMENT

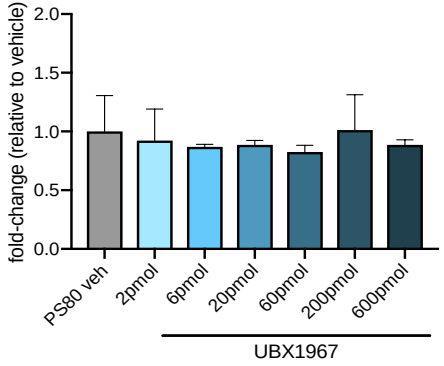
OIR Model





3 mo Healthy





UBX1967 VALUE PROPOSITION IN MULTIPLE AGE-RELATED EYE DISEASES

DIFFERENTIATING PRECLINICAL FEATURES

1. **Bcl senolytic:** Potent inhibitor of Bcl family
2. **Novel MOA**: eliminates SnCs → reduces multicomponent SASP
3. ***in vivo* efficacy** → activity in two preclinical models of retinopathy

PROPOSED CLINICAL BENEFITS

- Potential for improvements in visual function over anti-VEGF therapy
- Potential for efficacy in patients that don't respond to anti-VEGF therapy
- Potential for efficacy in combination with anti-VEGF therapy

Potential to reduce SASP factors across multiple diseases of aging retina

SUMMARY

UNITY
BIOTECHNOLOGY

FINANCIAL METRICS AND MILESTONES

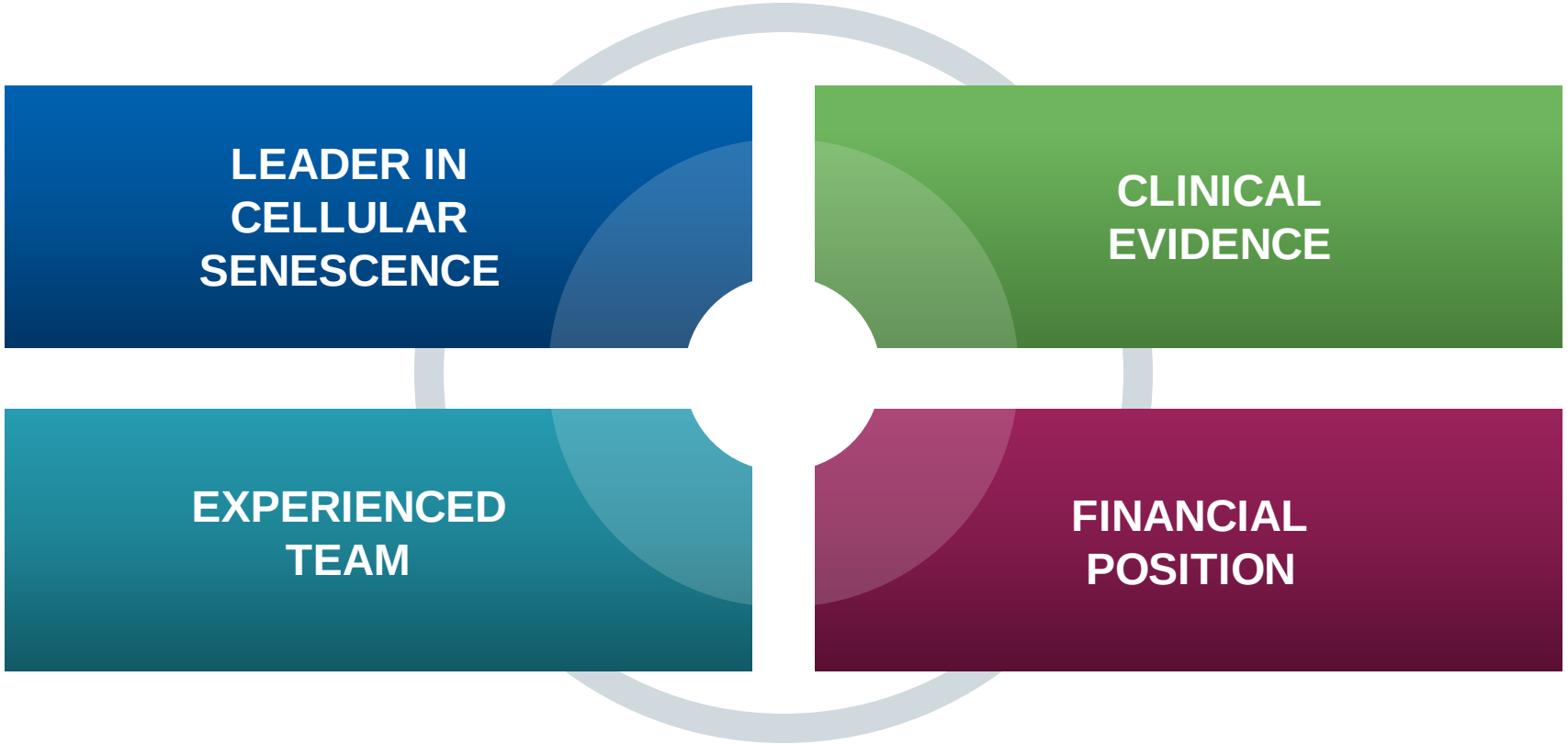
FINANCIAL

- $120.3 million cash and investments at September 30, 2019
- Cash runway into 2nd half of 2021

MILESTONES

- ✓ Q2 2019 – Data from UBX0101 Ph1
- ✓ Q4 2019 – Initiate UBX0101 Ph2
- Early 2020 – Anticipate ophthalmology IND filing
  - To enable multiple indications (e.g., AMD, DR and DME)
  - Safety data expected 2H 2020; Efficacy data expected 1H 2021
- 2H 2020 – Expect 12 week results from UBX0101 Ph2
- 1H 2021 – Expect 24 week results from UBX0101 Ph2

UNITY BIOTECHNOLOGY



At UNITY we are developing medicines designed to:

AGE-RELATED DISEASE

HEALTHSPAN

MANAGEMENT

An experienced team with a track record of success

KEITH LEONARD, MS, MBA
Chief Executive Officer

KYTHERA BIOPHARMACEUTICALS · ANACOR PHARMACEUTICALS · AMGEN

NATHANIEL DAVID, PHD
President

KYTHERA BIOPHARMACEUTICALS · ACHAOGEN · syrrx

DAN MARQUESS, D. PHIL
Chief Scientific Officer

Theravance · gsk GlaxoSmithKline

JAMIE DANANBERG, MD
Chief Medical Officer

Lilly · Takeda

BOB GOELTZ, CPA, MBA
Chief Financial Officer

CYTOMX THERAPEUTICS · ONYX PHARMACEUTICALS · AMGEN · Tularik

TAMMY TOMPKINS, JD
General Counsel

khosla ventures · AMYRIS

SUSIE LUNDEEN
SVP of People

KYTHERA BIOPHARMACEUTICALS · AMGEN

PEDRO BELTRAN, PHD
SVP of Biology

AMGEN

DOUG RICH, MBA
SVP, Operations

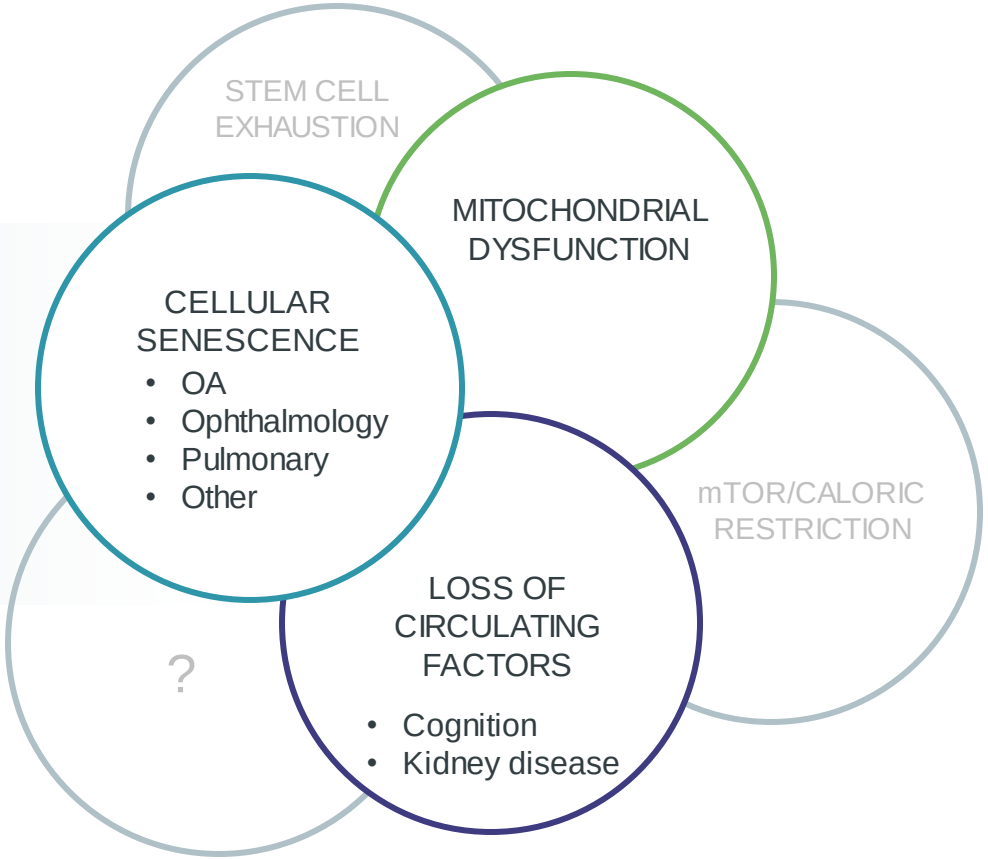
KYTHERA BIOPHARMACEUTICALS · AMGEN · Boehringer Ingelheim

CAMILLE LANDIS, MBA
SVP, Corporate Development

AFFYMAX · NAVIGANT CONSULTING · PiperJaffray. · relypsa

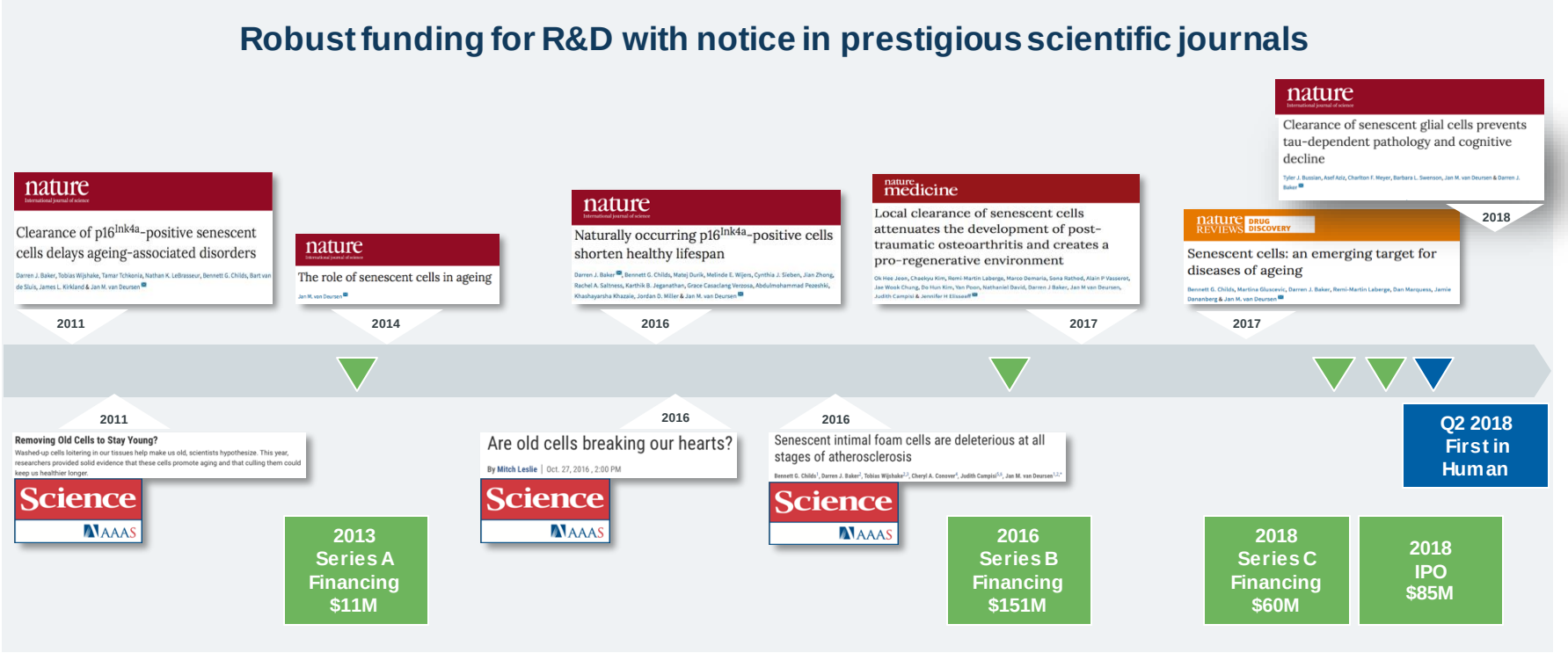
MULTIPLE MECHANISMS DRIVE AGING

UNITY is pursuing multiple pathways to impact the aging process



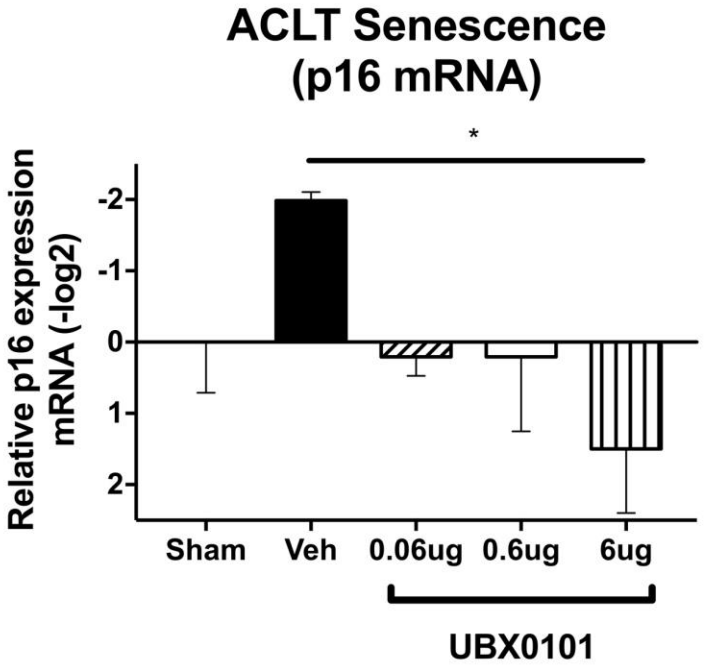
UNITY: ESTABLISHING LEADERSHIP IN HEALTHSPAN

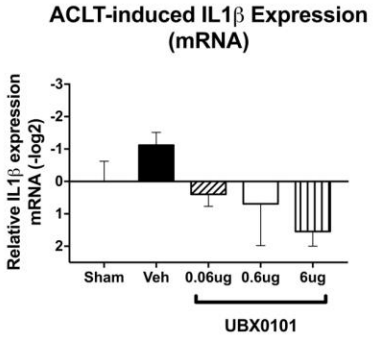
Robust funding for R&D with notice in prestigious scientific journals

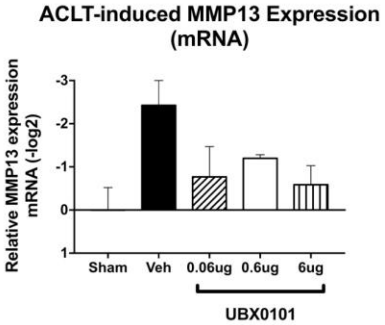


UBX0101 EFFICACY *IN VIVO*

Intra-articular dosing eliminates senescent cells and modulates SASP in mice







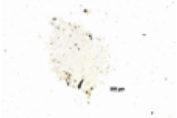
* *p*-value ≤ .05

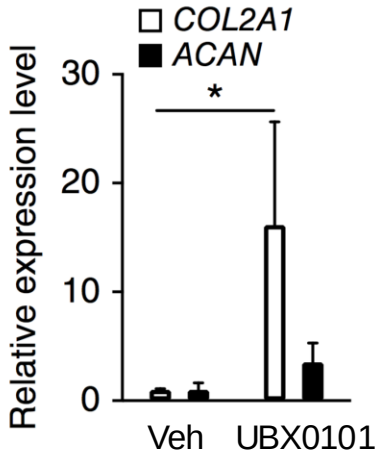
IA dosing of UBX0101 eliminates p16^{+} SnCs and reduces OA-relevant SASP factors

UBX0101 EFFICACY *EX VIVO*

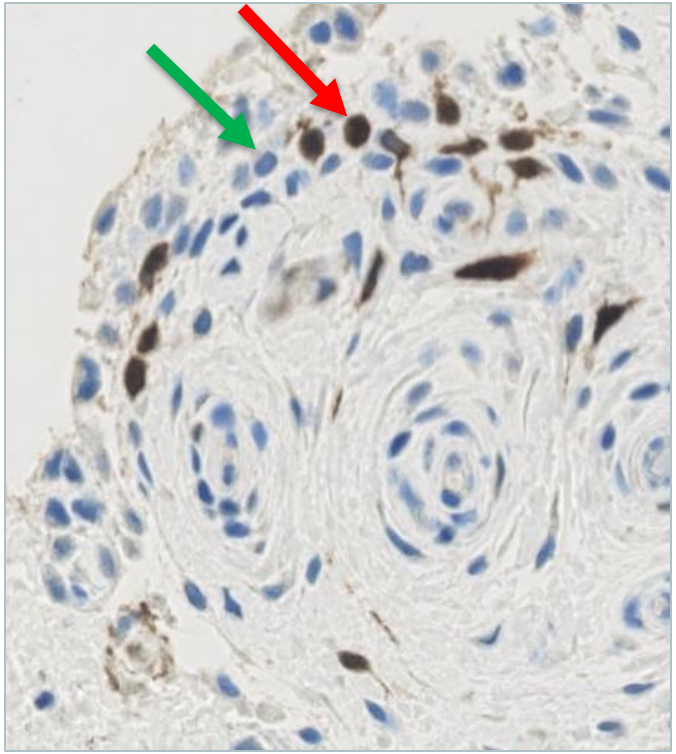
Diseased tissue exposed to UBX0101 upregulates expression of key components of cartilage

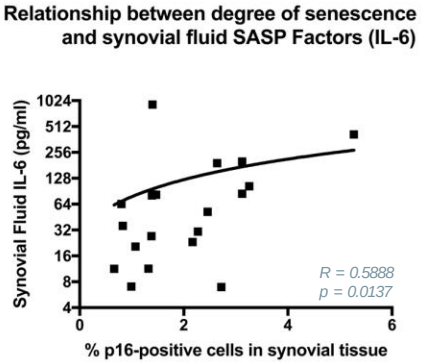
Human OA
chondrocyte isolation

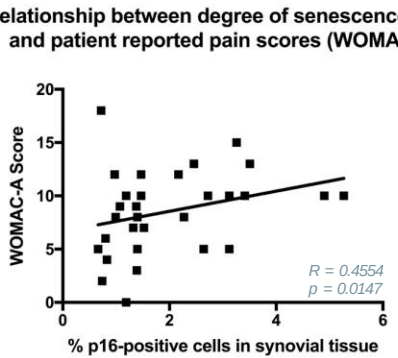
	SnC MARKERS		CARTILAGE MARKERS	
	p16	MMP13	Type 2 collagen	Aggrecan
VEHICLE				
UBX0101				

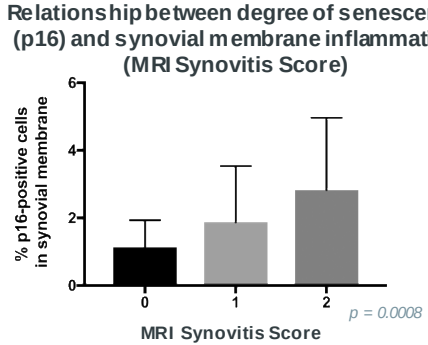


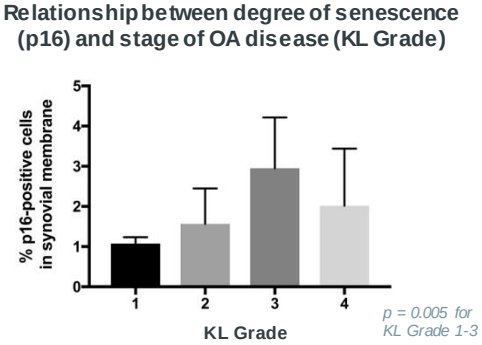
INCREASED SnCs OBSERVED IN KNEES OF OA PATIENTS











In 30 patients, senescence burden correlated directly with pain, arthritic severity, and inflammation

PATIENT DEMOGRAPHICS

	Part A		Part B	
	Total Subjects (n=48)	Cohorts Balanced	Total Subjects (n=30)	Cohorts Balanced
Age (yrs)	62.4	Yes	61.2	Yes
Gender (M:F)	16:32	No	15:15	Yes
Race (%) (Asian/African American/Pacific Islander/White/American Indian)	0 / 6.3 / 0 / 89.6 / 4.2	Yes	0 /16.7 / 0 / 82.3	Yes
Ethnicity (%) (Hispanic/Non-Hispanic/Unknown)	33.3 / 64.6 / 2.1	No	40 / 60 / 0	Yes
Weight (kg)	82.20	Yes	84.50	Yes
Height (cm)	165.0	Yes	167.0	Yes
BMI (kg/m^2)	30.25	Yes	29.10	Yes

BASELINE PATIENT CHARACTERISTICS

Baseline Characteristic	Part A (UBX-0101 Intra- Articular Dose in mg) Mean (SD)							Part B Mean (SD)	
Dose Group (n)	Placebo (n=14)	0.1 (n=6)	0.2 (n=5)	0.4 (n=5)	1.0 (n=6)	2.0 (n=6)	4.0 (n=6)	Placebo (n=10)	4.0 (n=20)
K-L Score	2.58 (0.90)	2.83 (0.41)	3.00 (1.22)	3.00 (1.22)	2.67 (0.52)	2.50 (0.84)	3.17 (0.41)	2.50 (0.85)	2.47 (1.12)
11-pt Synovitis Score	13.36 (5.14)	10.33 (5.79)	16.20 (4.21)	8.25 (5.19)	12.67 (4.80)	12.00 (5.59)	11.17 (5.38)	Not measured in Part B	
Yrs Dx with OA	6.84 (4.04)	15.4 (15.3)	11.3 (4.39)	10.3 (6.57)	13.4 (10.1)	11.6 (8.39)	6.84 (4.05)	10.1 (8.73)	8.64 (6.36)
BL WOMAC total	47.14 (12.96)	54.17 (7.41)	37.60 (11.55)	58.80 (10.08)	50.67 (14.07)	41.67 (11.45)	46.67 (6.44)	52.40 (12.57)	50.45 (16.37)
BL WOMAC A Pain	9.36 (2.21)	11.17 (1.94)	9.00 (0.71)	11.80 (1.48)	9.67 (2.94)	8.50 (3.15)	9.83 (1.60)	11.30 (1.89)	11.10 (3.40)
BL WOMAC B Stiffness	4.93 (1.27)	4.83 (0.98)	4.80 (0.84)	5.00 (1.41)	4.50 (1.52)	4.00 (0.89)	3.67 (0.82)	4.40 (1.35)	4.40 (1.47)
BL WOMAC C Function	32.86 (10.80)	38.17 (5.08)	23.80 (10.94)	42.00 (8.28)	36.50 (10.62)	29.17 (9.43)	33.17 (5.27)	36.70 (10.24)	34.95 (12.70)
BL Weekly Average NRS	6.47 (1.11)	5.90 (1.40)	6.30 (0.53)	6.76 (1.10)	6.49 (1.55)	6.15 (1.18)	6.29 (1.42)	Not measured in Part B	

EFFICACY: PART A – IMPRESSION OF CHANGE - PGIC

Grouped Dose Cohorts – 12 Week

PGIC Binary Variable	Part A (UBX-0101 Intra-Articular Dose in mg) Estimated Probability (95% CI)		
Dose Group (n)	Placebo (n=14)	Low Doses (n=16)	High Doses (n=18)
Much Improved or Better	42.9 % (20.6 – 68.4%)	50.0 % (40.0-60.0%)	61.1 % (50.0- 66.7%)

PGIC score demonstrated dose response

Low Dose - (0.1, 0.2, 0.4 mg)

High Dose - (1.0, 2.0, 4.0 mg)

Consistent efficacy demonstrated in Part A across multiple measurements including NRS, WOMAC-A, WOMAC-C & PGIC

PART B MEASURES OF PAIN & FUNCTION DIRECTIONALLY CONSISTENT WITH PART A RESULTS

WOMAC-A

5 item, 0-4 point scale where a 0.5 point change is considered clinically meaningful.

WOMAC-C

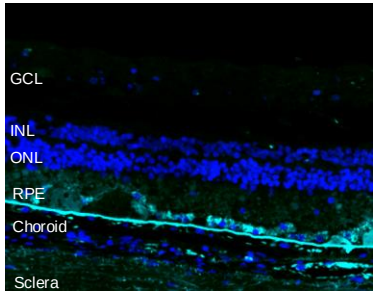
17 item, 0-4 point scale where a 0.3 point change is considered clinically meaningful.

	WOMAC-A		WOMAC-C	
	CFBL	Pbo-Adj (P-value)	CFBL	Pbo-Adj (P-value)
Placebo (n=10)	-0.72		-0.60	
4.0 mg (n=20)	-0.87	-0.15 (0.62)	-0.77	-0.17 (0.60)

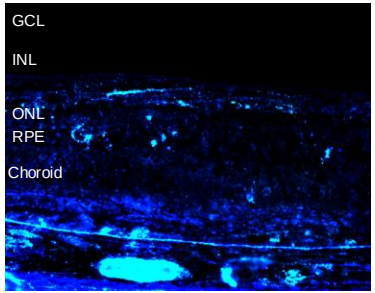
Part B procedure for optimal collection of synovial fluid following treatment included complete drainage of the knee, and if fluid yield is insufficient, introducing saline and then repeating the withdrawal

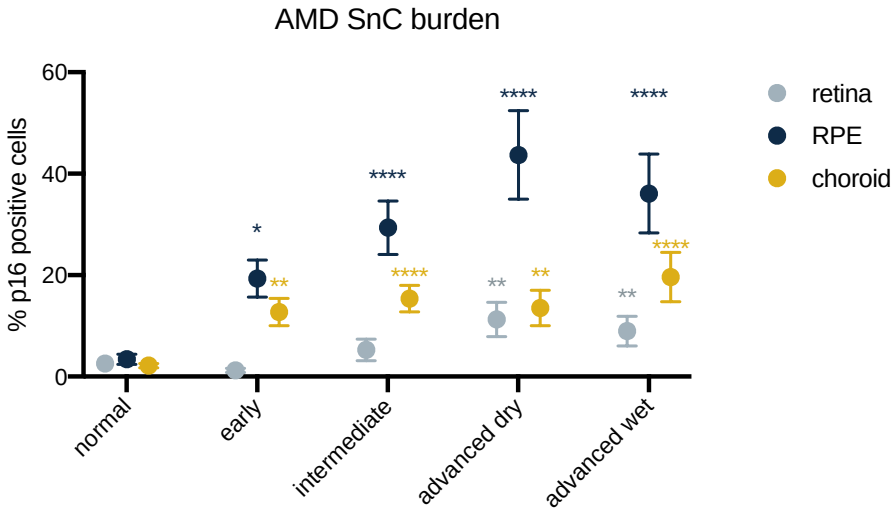
SNC BURDEN INCREASES WITH DISEASE STAGE

INTERMEDIATE (86F)



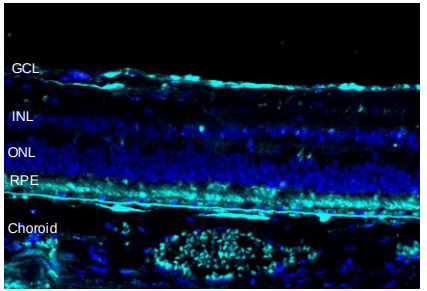
EARLY (89M)



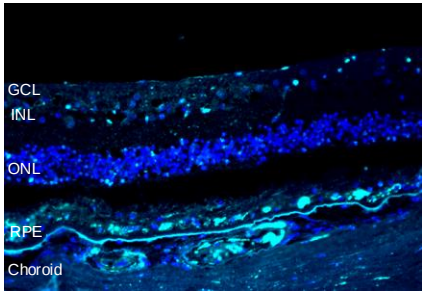


*p<0.05; **p<0.01; ****p<0.0001 v. Normal by Kruskal-Wallis with Dunn's multiple comparisons test

ADVANCED DRY (93F)



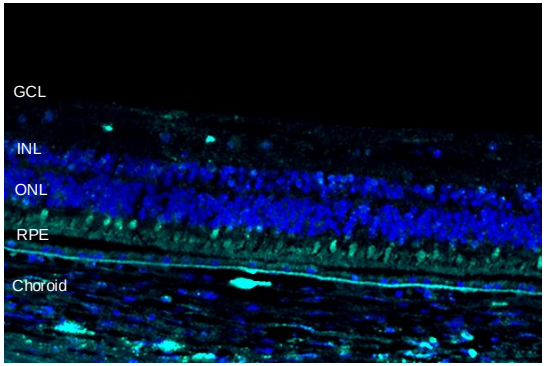
ADVANCED WET (93M)



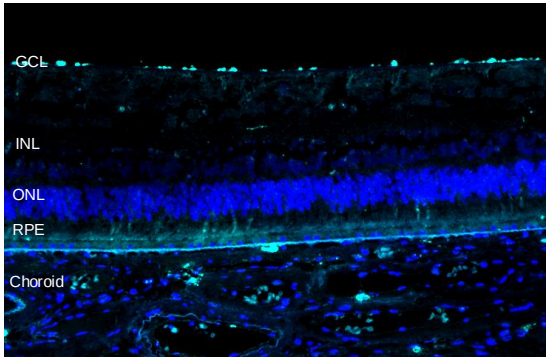
Senolysis has the opportunity to halt disease progression from an early stage

DR/DME PATIENTS SHOW SNC IN THE RETINA AND CHOROID

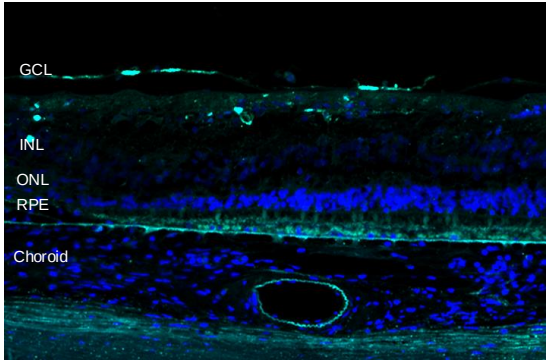
NPDR (78F)



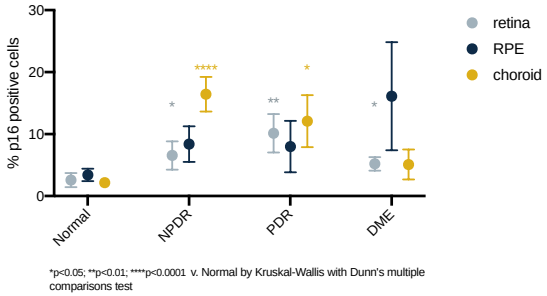
DME (54F)



PDR (64M)

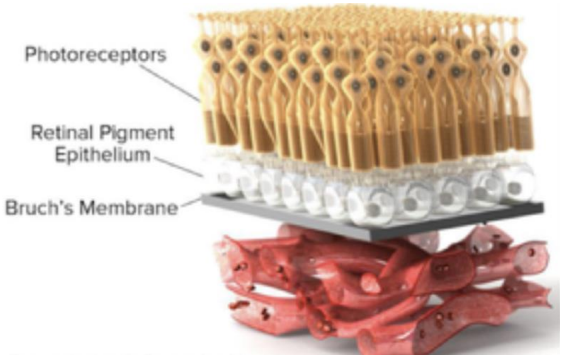


DR SNC BURDEN

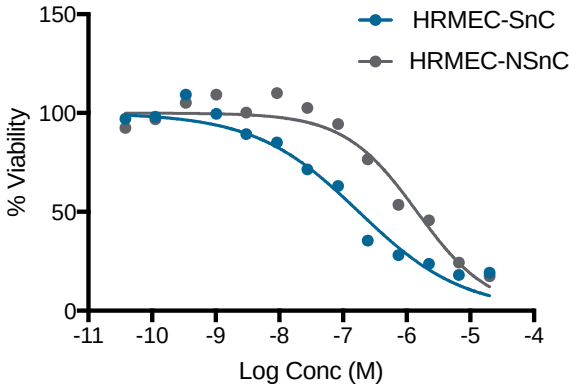


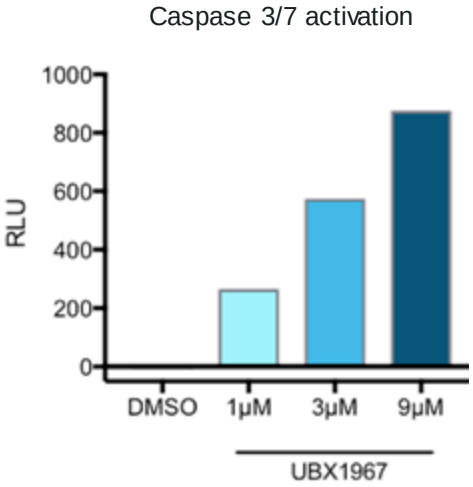
Unpublished UNITY Data

HIGHLY POTENT SENOLYTIC THAT TARGETS BCL-2 FAMILY PROTEINS



Human retinal microvascular endothelial cells (HRMEC)



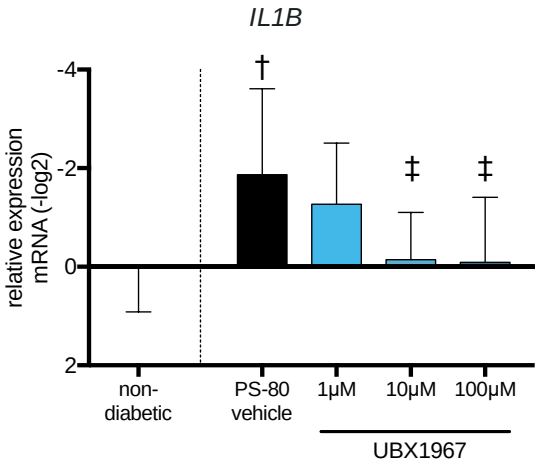


UBX1967 selectively eliminates HRMEC-SnCs over non-SnCs

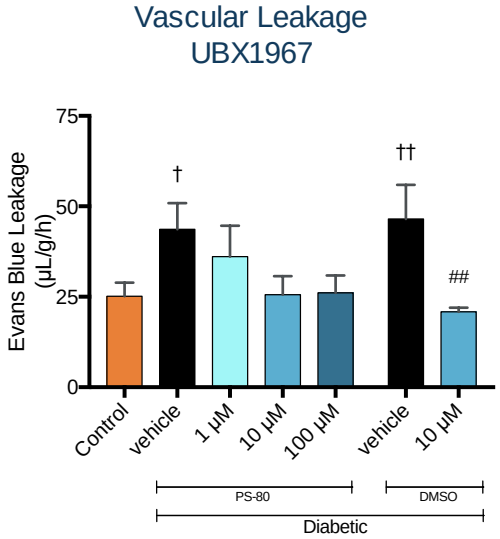
Unpublished UNITY Data

UBX1967 DEMONSTRATES EFFICACY IN MOUSE STZ

Streptozotocin (STZ) diabetic retinopathy model

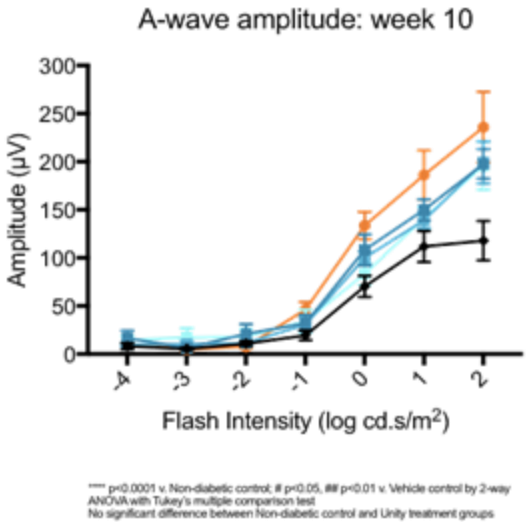


† p<0.05 v. non-diabetic control by two-tailed t-test;
‡ p<0.05 v. Vehicle; one-way ANOVA, with Dunnett's multiple comparison test



† p<0.05 v. Non-diabetic control by two-tailed t-test

†† p<0.01 v. Non-diabetic control by two-tailed t-test
p<0.01 v. DMSO control by two-tailed t-test

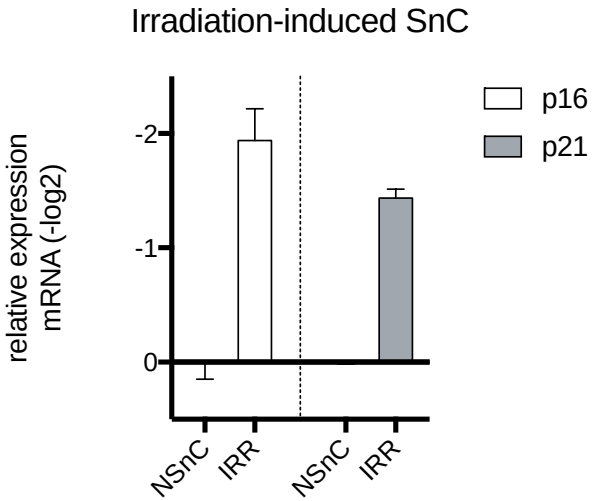


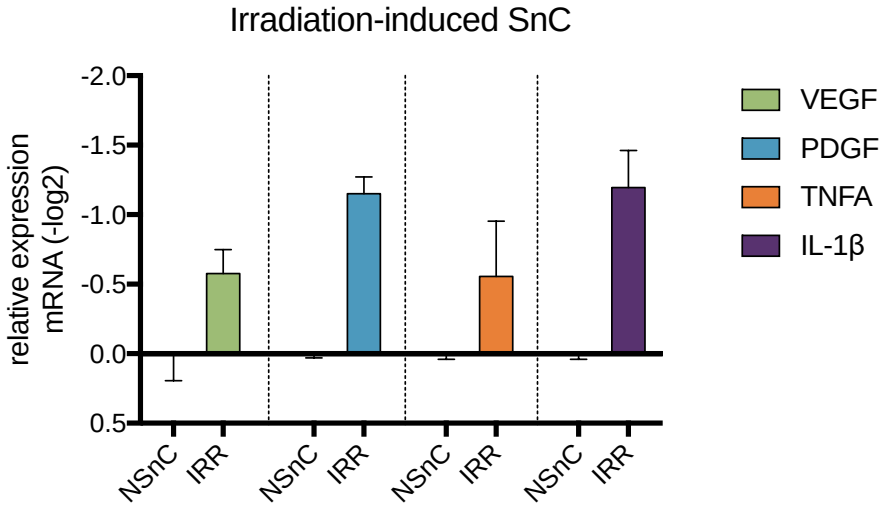
**** p<0.0001 v. Non-diabetic control; # p<0.05, ## p<0.01 v. Vehicle control by 2-way ANOVA with Tukey's multiple comparison test
No significant difference between Non-diabetic control and Unity treatment groups

Intravitreal dosing reduces SASP & vascular leakage and protects retinal function in diabetic mice

1 μM = ~1.7 ng of UBX1967

INDUCTION OF SASP FACTORS IN SENESCENT HRMECS





AGE-RELATED EYE DISEASES ARE MULTIFACTORIAL

Factors beyond VEGF are detected in the vitreous of AMD & DR patients

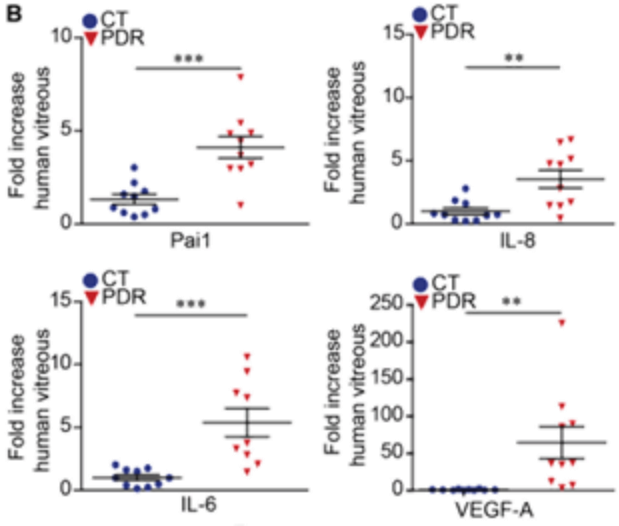
AMD

	nAMD Level mean ± SD	Controls Level mean ± SD	*P* value Pre IVA vs Control
IL-1rα	0	0.77 ± 3.19	0.381
IL-6	6.51 ± 5.24	78.2 ± 100	0.0029
IL-7	10.9 ± 3.95	13.5 ± 12.8	0.465
IL-8	6.00 ± 6.69	6.43 ± 6.84	0.448
IL-9	0.087 ± 0.40	0.10 ± 0.41	0.483
IL-12	12.1 ± 5.79	10.4 ± 9.11	0.268
IL-13	1.97 ± 2.22	2.25 ± 2.65	0.448
Eotaxin	3.76 ± 4.17	1.22 ± 2.39	0.035
βFGF	0	0.71 ± 2.92	0.381
G-CSF	0.28 ± 1.27	0	0.402
IP-10	755 ± 645	273 ± 260	3.00×10^{-4}
MCP-1	229 ± 155	204 ± 112	0.381
MIP-1α	0.41 ± 0.91	0.96 ± 2.35	0.4
MIP-1β	37.2 ± 15.6	37.2 ± 31.1	0.112
VEGF	228 ± 176	132 ± 54.2	0.029

*pg/ml in AH

DR

SASP	Control	DR/PDR	P-value
IL-1b	4.0	12.9	<0.0001
	5.5	34.1	0.0001
IL-6	43.3	212.5	0.0005
	4.72	59.37	0.0003
	6.9	45.2	0.0005
IL-8	18.2	53.6	<0.0001
	7.43	87.89	<0.0001
	12.4	96.2	0.0003
TNFa	63.9	155.8	<0.0001
	12.3	160.7	0.0001
VEGF	18.9	422.6	0.0028
	23.5	1208.1	0.016
	7.36	240.18	0.0031
PDGF (BB)	0.84	15.64	0.0234
	0.7	2.9	0.015
PDGF (AA)	29.42	131.75	<0.0001
	99.1	602.2	0.007



*pg/ml in VH

Oubaha et al., Senescence-associated secretory phenotype contributes to pathological angiogenesis in retinopathy, Sci. Transl. Med. 8, 362ra144 (2016)
Sato (2018)