

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 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 June 30, 2019, filed with the Securities and Exchange Commission on August 7, 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

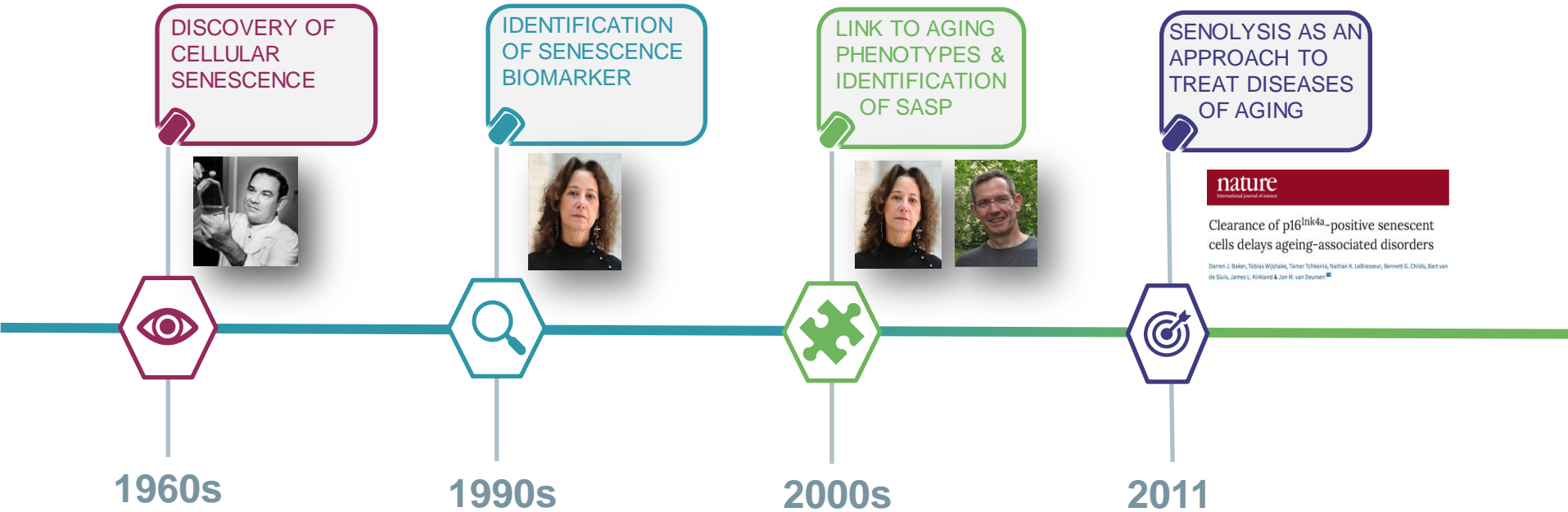
- Seasoned executive team with broad biotech experience
- Strong track record of delivering for patients and investors

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



FROM SCIENTIFIC INSIGHT TO THERAPEUTIC BENEFIT

nature
International weekly journal of science

Letter | Published: 02 November 2011

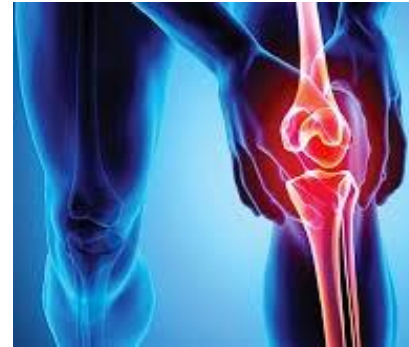
Clearance of p16^{Ink4a}-positive senescent cells delays ageing-associated disorders

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

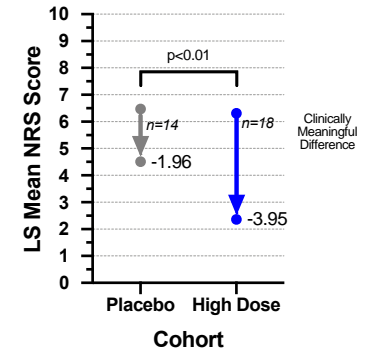


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Phase 1 clinical study in osteoarthritis showed improvements in pain and function



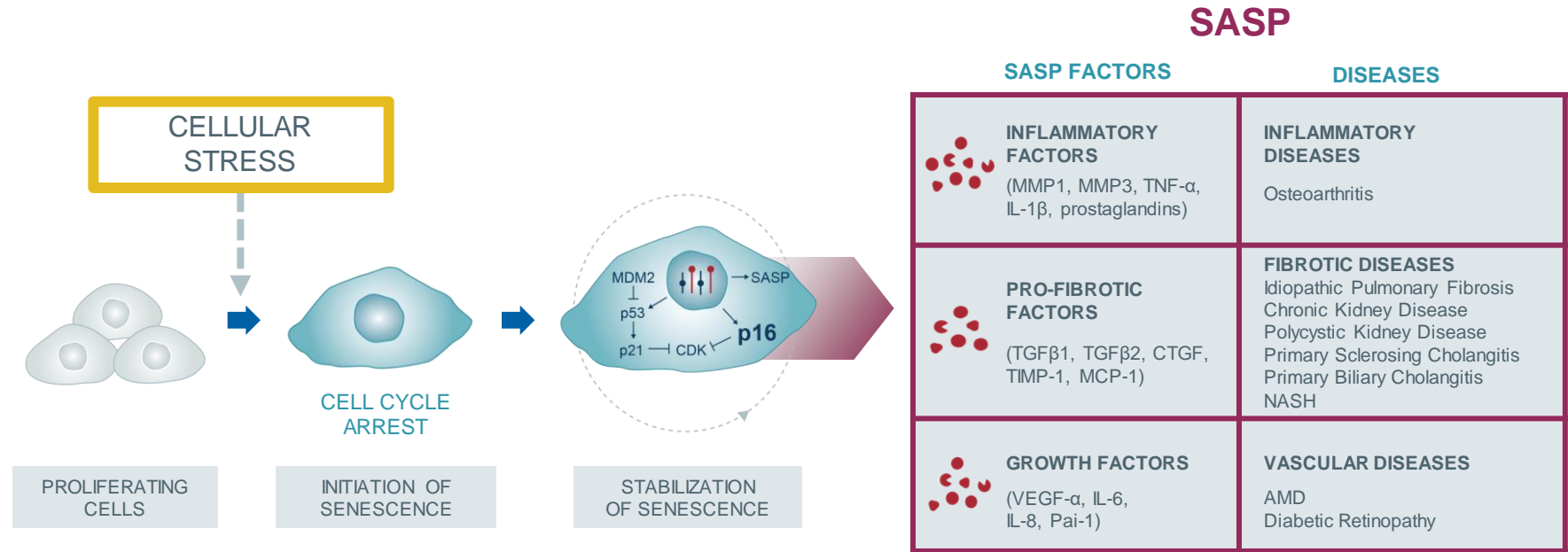
NRS
Change in LS Mean Score
From Baseline to 12 Weeks



2011

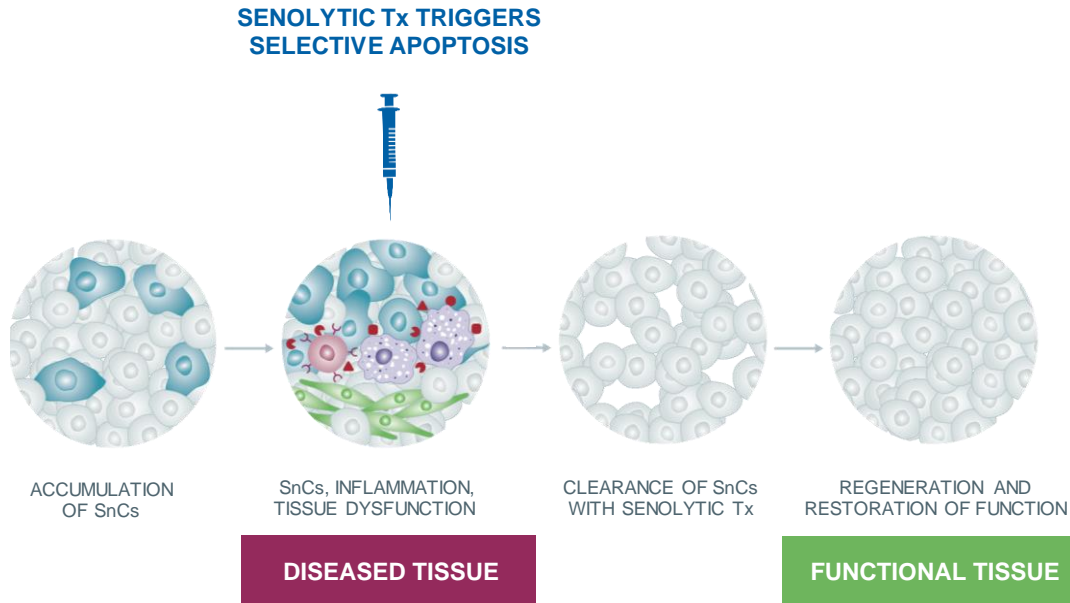
2019

SENESCENT CELLS ARE IMPLICATED IN MULTIPLE DISEASES OF AGING



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

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



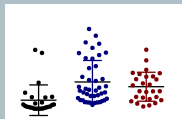
OUR PLATFORM FOR GENERATING POTENT AND SELECTIVE SENOLYTIC DRUG CANDIDATES



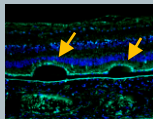
IDENTIFY

Identify SnCs in human disease:

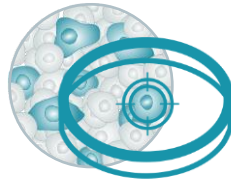
- Cell type(s)
- Tissue location
- SnC burden
- SASP signature



SnC presence



SnC location



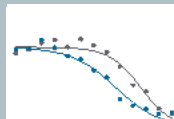
TARGET

Eliminate SnCs selectively:

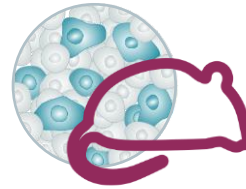
- Survival pathways
- Apoptosis mechanisms:
BCL-2 family, p53/MDM2



Apoptosis



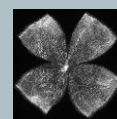
SnC elimination



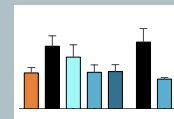
IMPACT

Drive efficacy with senolytic:

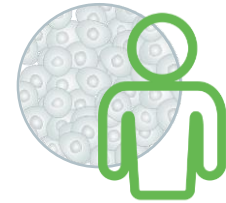
- Efficacy endpoints
- SASP reduction
- Aged *in vivo* models
- Genetic models
- Disease models



Tissue changes



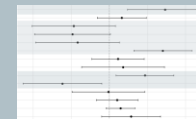
Disease impact



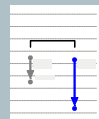
TRANSLATE

Senolytic impacts patient outcomes

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



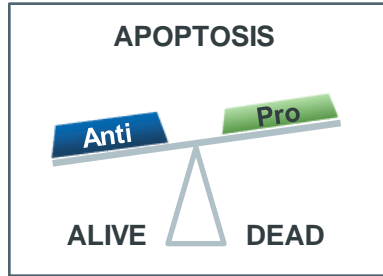
Biomarkers impact



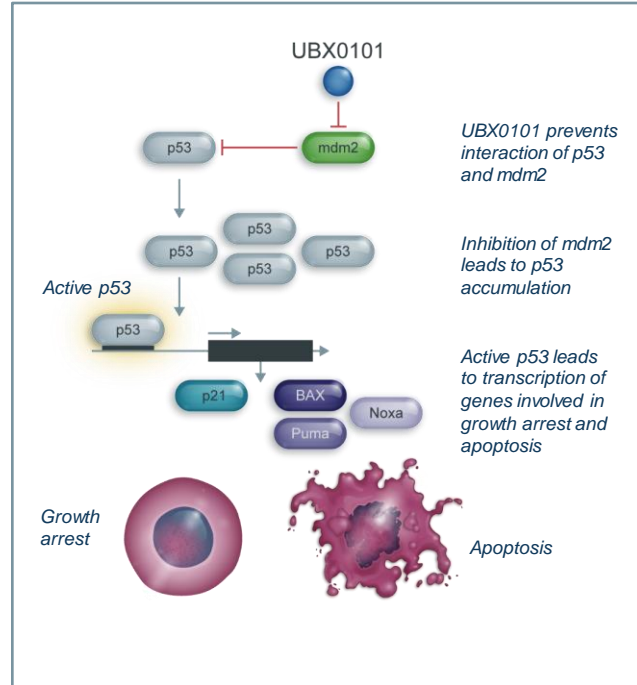
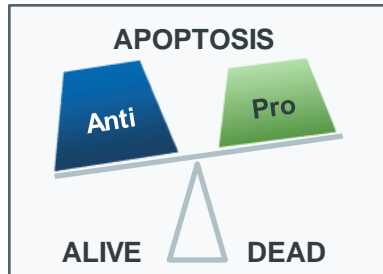
Clinical improvement

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

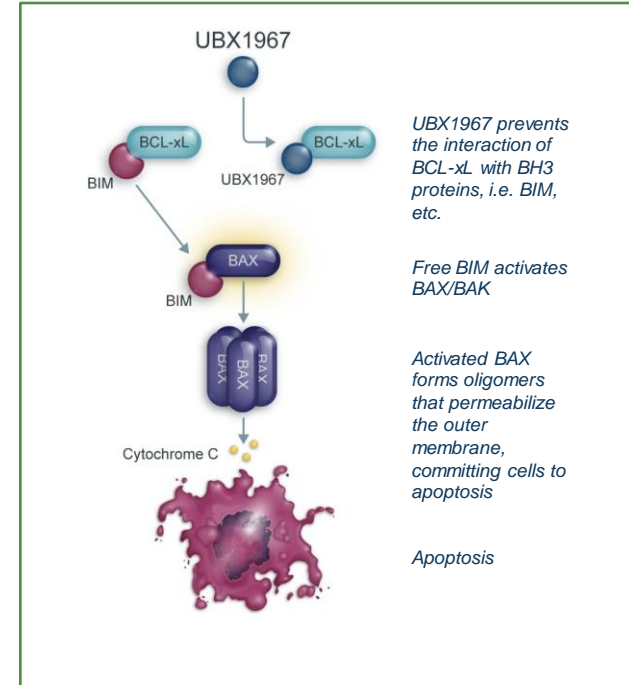
NON-SENESCENT CELL



SENESCENT CELL



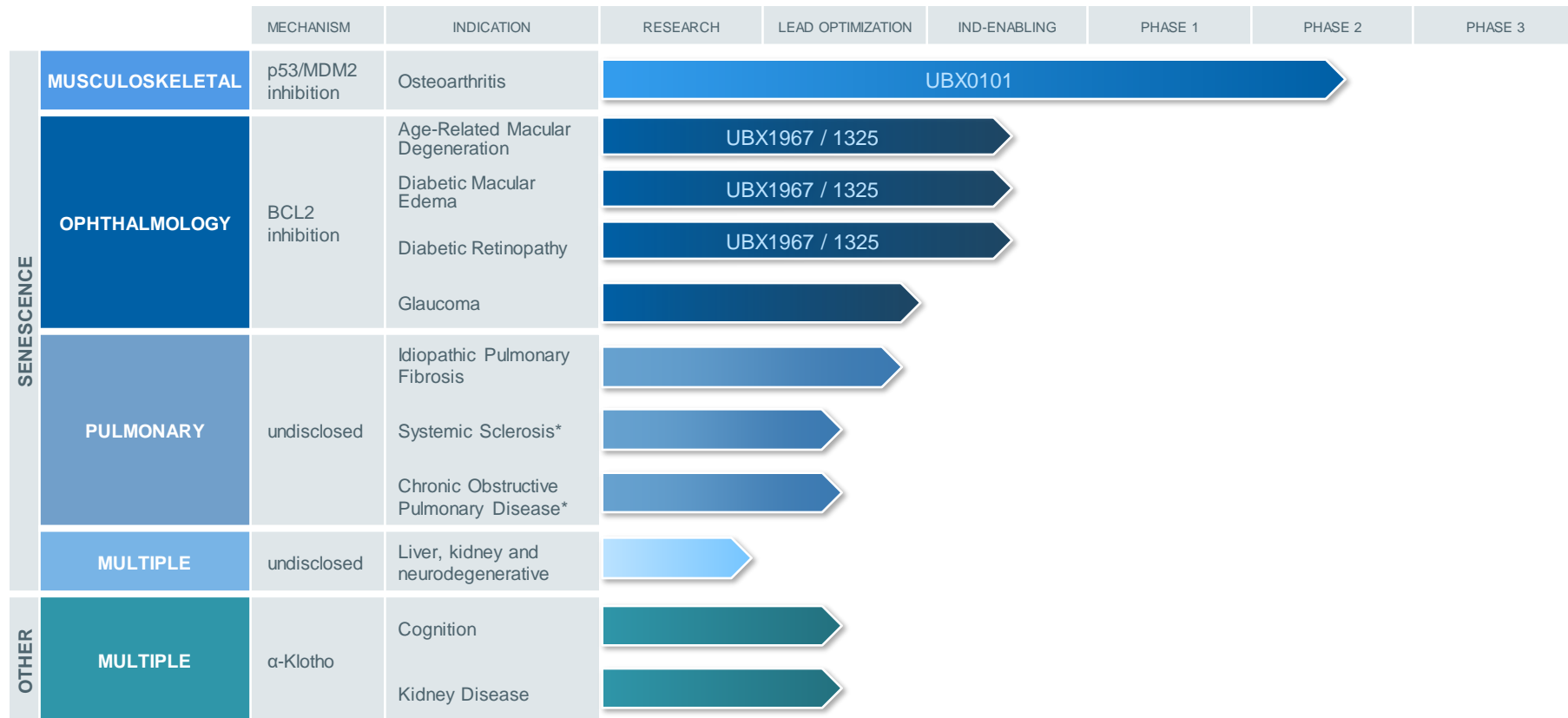
p53/mdm2



Bcl-2

UNITY PIPELINE

Pursuing broad range of diseases with established endpoints and regulatory pathways



OSTEOARTHRITIS

(MUSCULOSKELETAL
INDICATION)



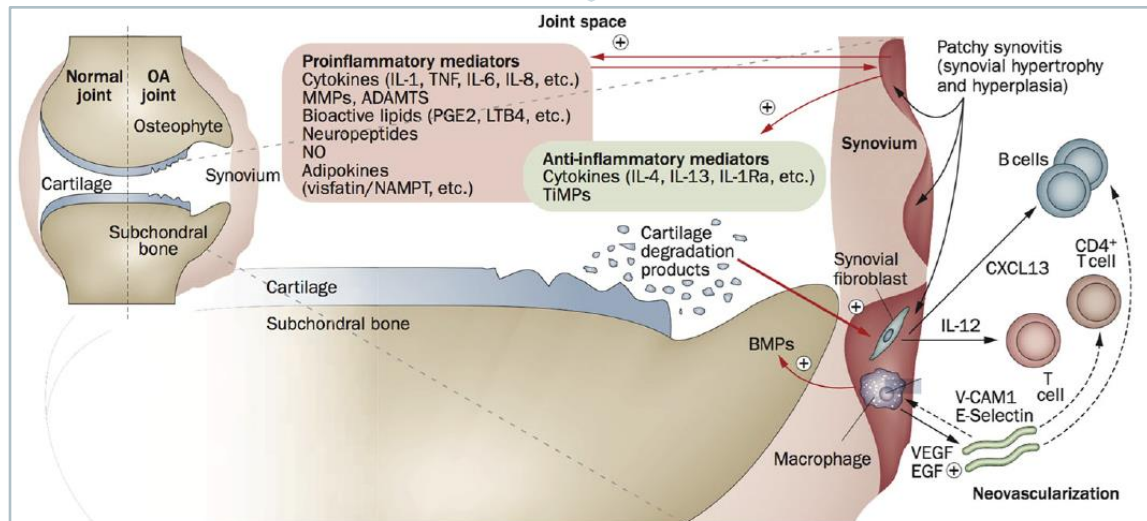
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OSTEOARTHRITIS

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

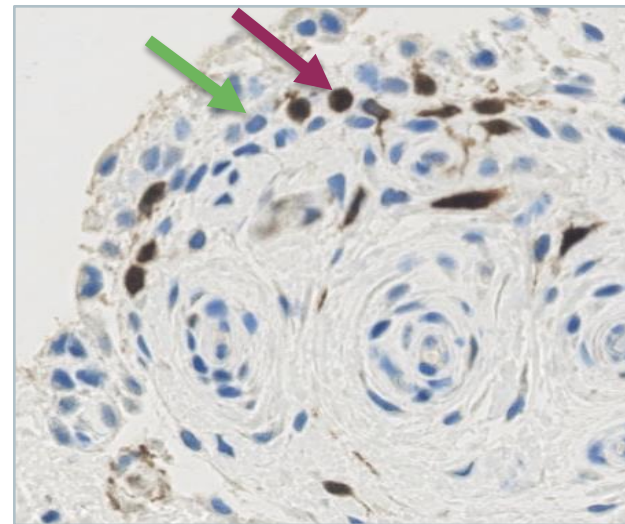


OA IS BELIEVED TO BE A MULTIFACTORIAL DISEASE



Mathiessen et al. *Arthritis Research and Therapy* 2017

In phase 0 study, senescence burden correlated with disease severity



A senolytic may remove source of multiple factors implicated in OA

UBX0101 PHASE 1 PROGRAM

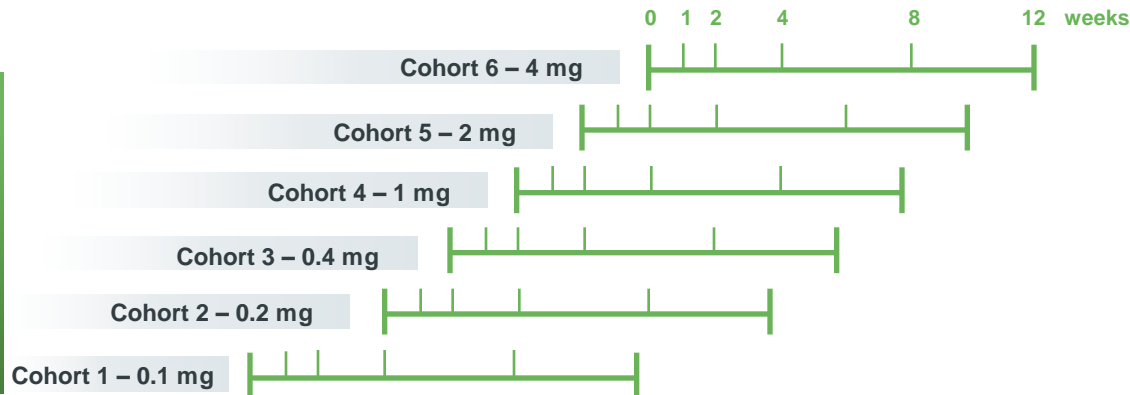


Single Ascending Dose Study (Part A)

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

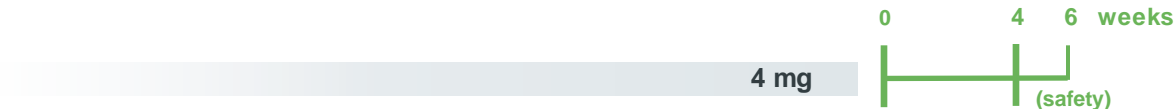
Secondary Objectives: Plasma PK and 11-Point NRS daily pain assessment, WOMAC (Pain, Stiffness, Function and Total), Plasma and Synovial fluid SASP/OA biomarkers to 12 weeks

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

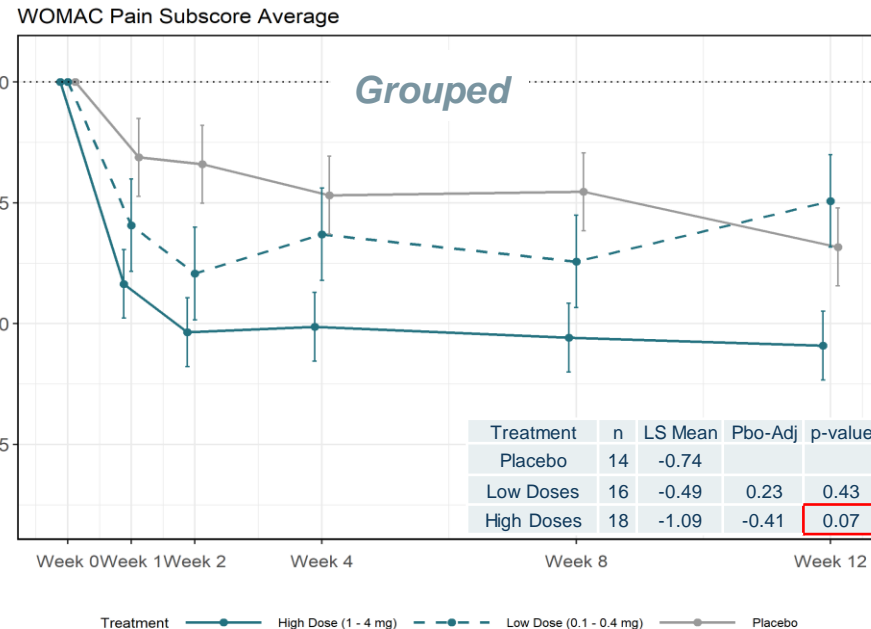
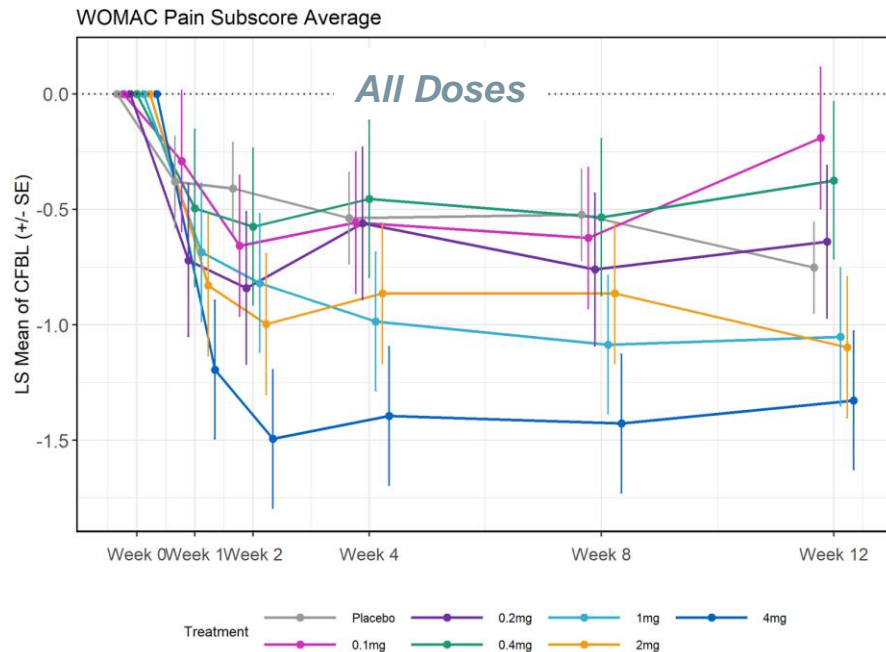
Preferred Term	Part A, 0.1- 4mg (N= 34) n (%)	Placebo (N= 14) n (%)	Part B (4 mg) (N= 20) n (%)	Placebo (N= 10) n (%)
Nasopharyngitis	2 (5.9)	1 (7.1)	0	0
Procedural pain	2 (5.9)	1 (7.1)	2 (10.0)	0
Arthralgia	3 (8.8)	1 (7.1)	0	1 (10.0)
Headache	4 (11.8)	1 (7.1)	0	0

SINGLE DOSE OF UBX0101 DECREASED PAIN



WOMAC-A

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



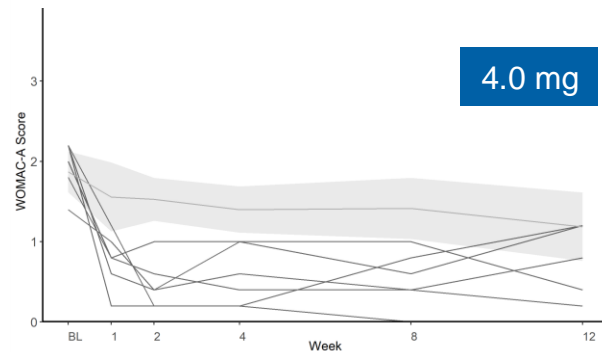
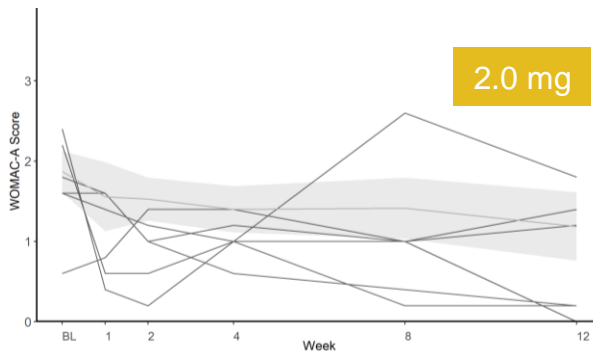
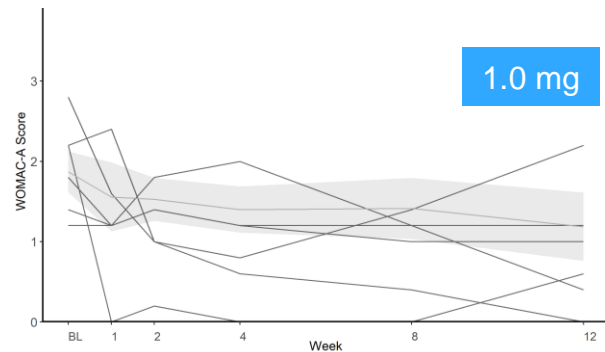
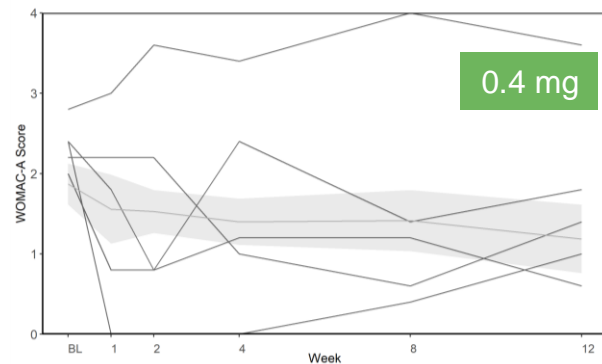
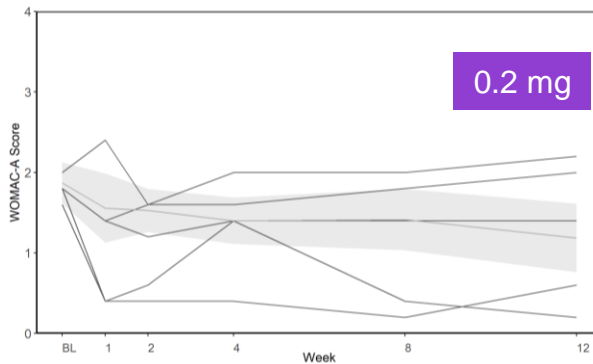
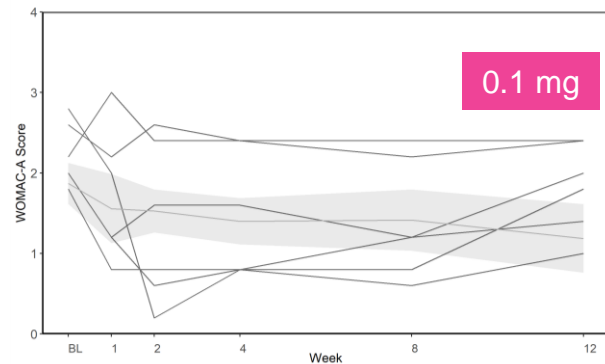
Durable, dose-dependent and substantial effect

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

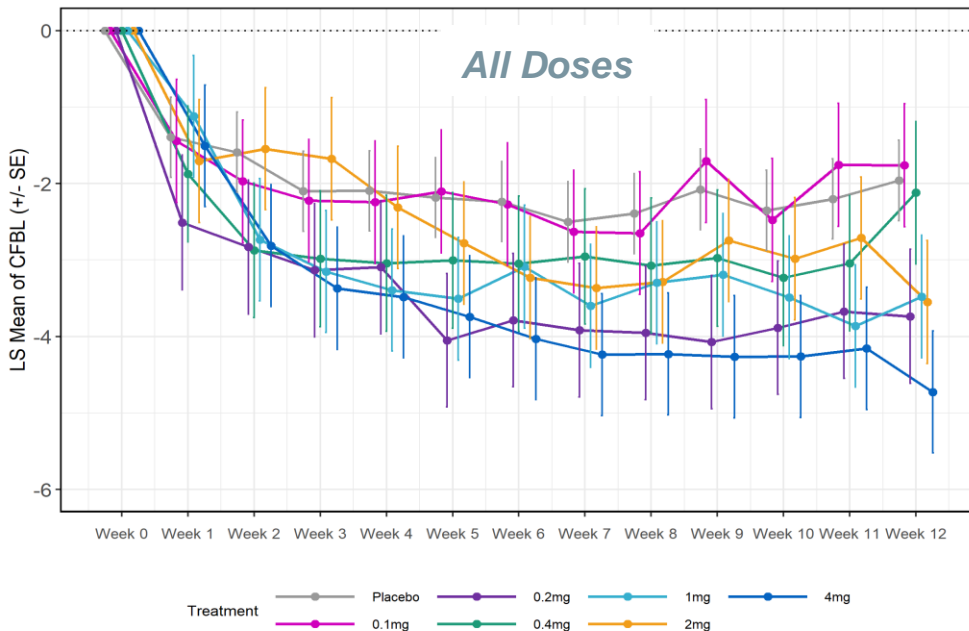
SINGLE DOSE OF UBX0101 DECREASED PAIN



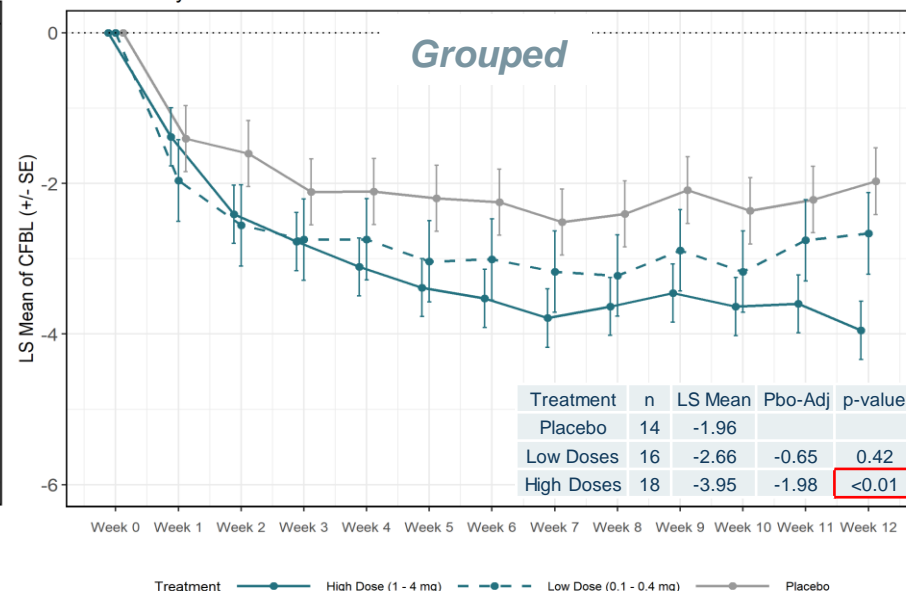
Numerical Rating Scale (NRS)

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

NRS Weekly Score



NRS Weekly Score



Durable, dose-dependent and substantial effect

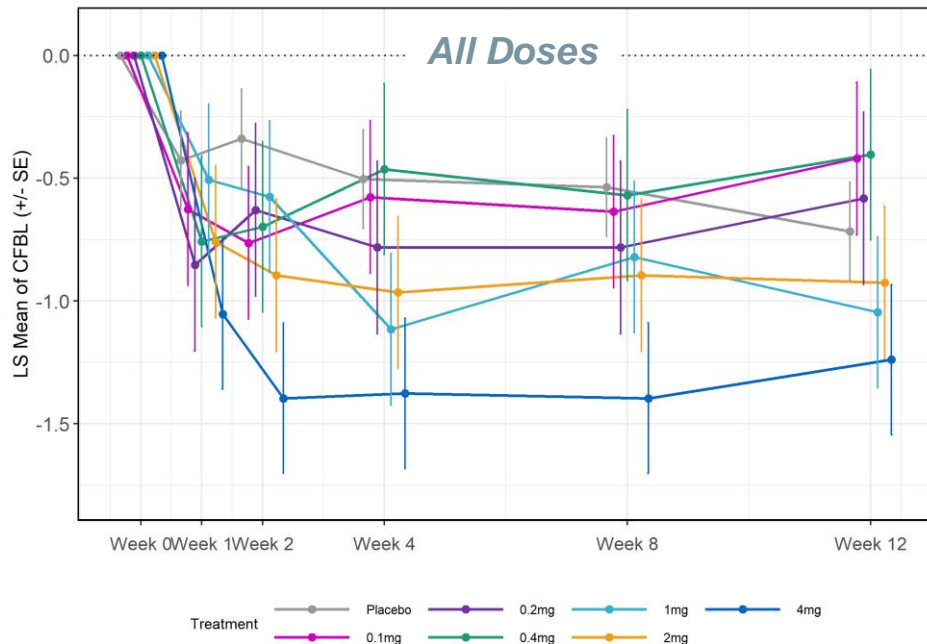
SINGLE DOSE OF UBX0101 IMPROVED FUNCTION



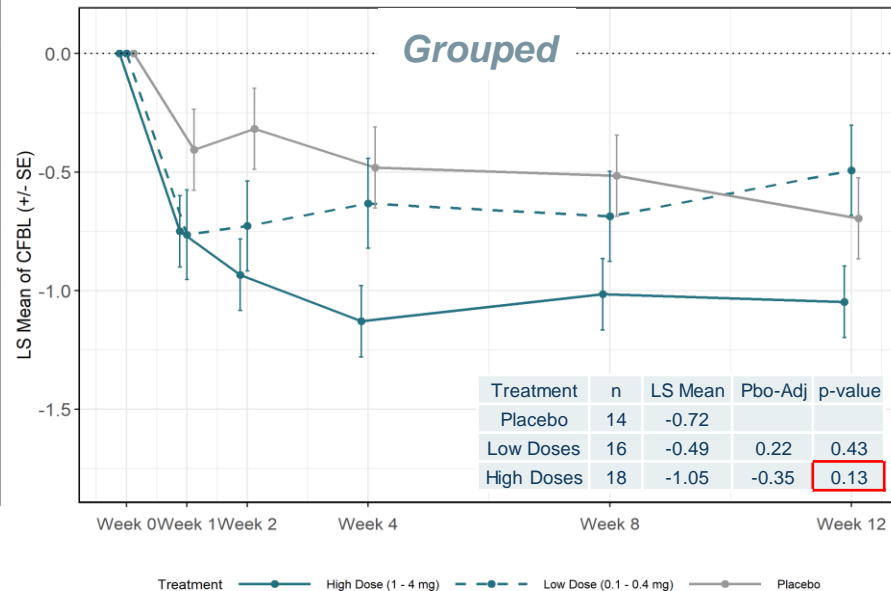
WOMAC-C

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

WOMAC Physical Function Subscore Average



WOMAC Physical Function Subscore Average

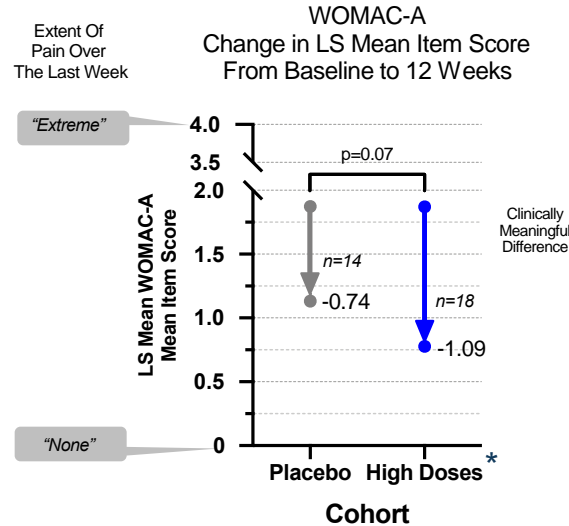
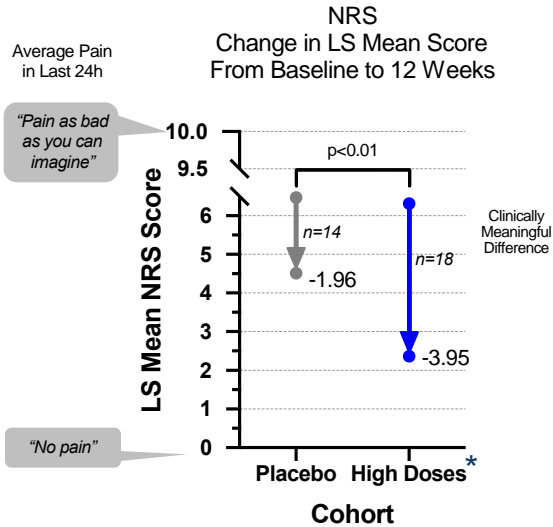


Durable, dose-dependent and substantial effect

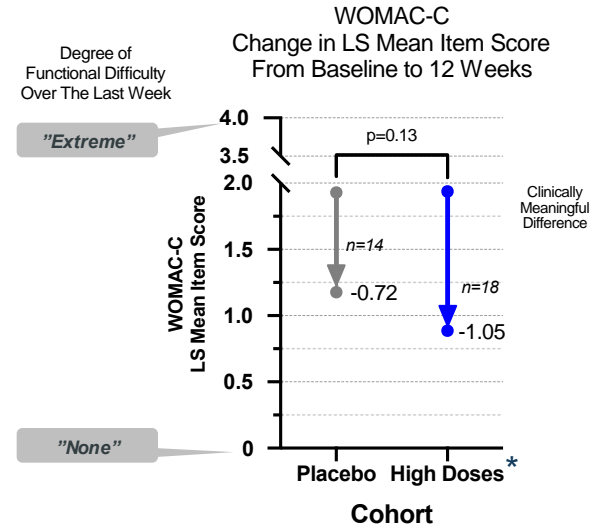
SINGLE DOSE OF UBX0101 IMPACTED PAIN AND FUNCTION AT 12 WEEKS



Pain Instruments



Physical Function Instrument



* High Doses = 1, 2, and 4mg

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

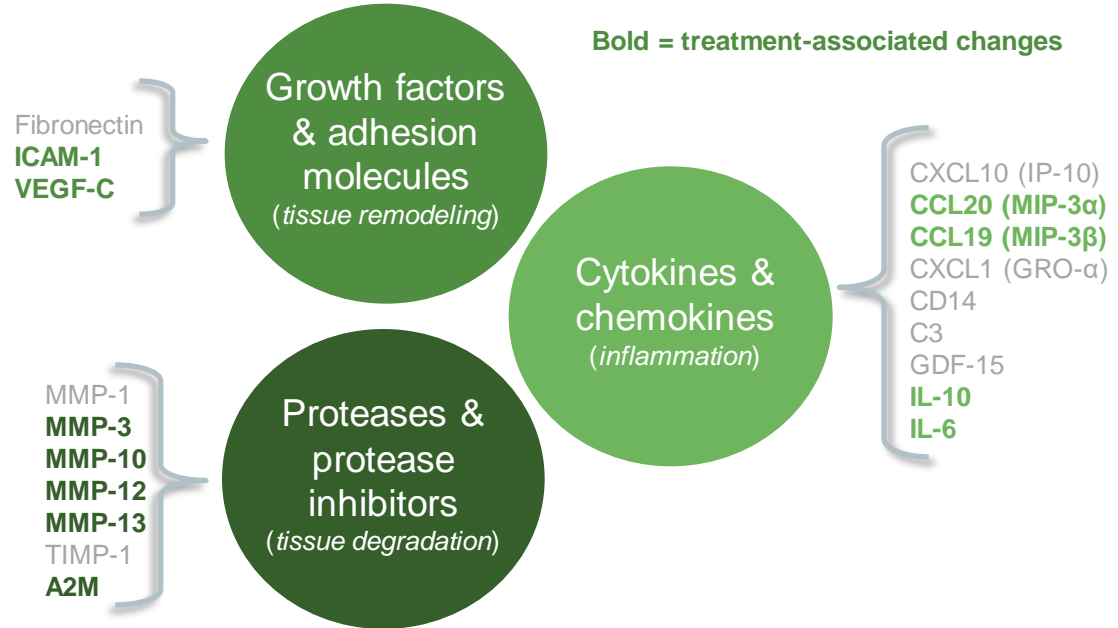
PHASE 1 MEASUREMENT OF SASP / OA BIOMARKERS

Data is supportive of a senolytic mechanism



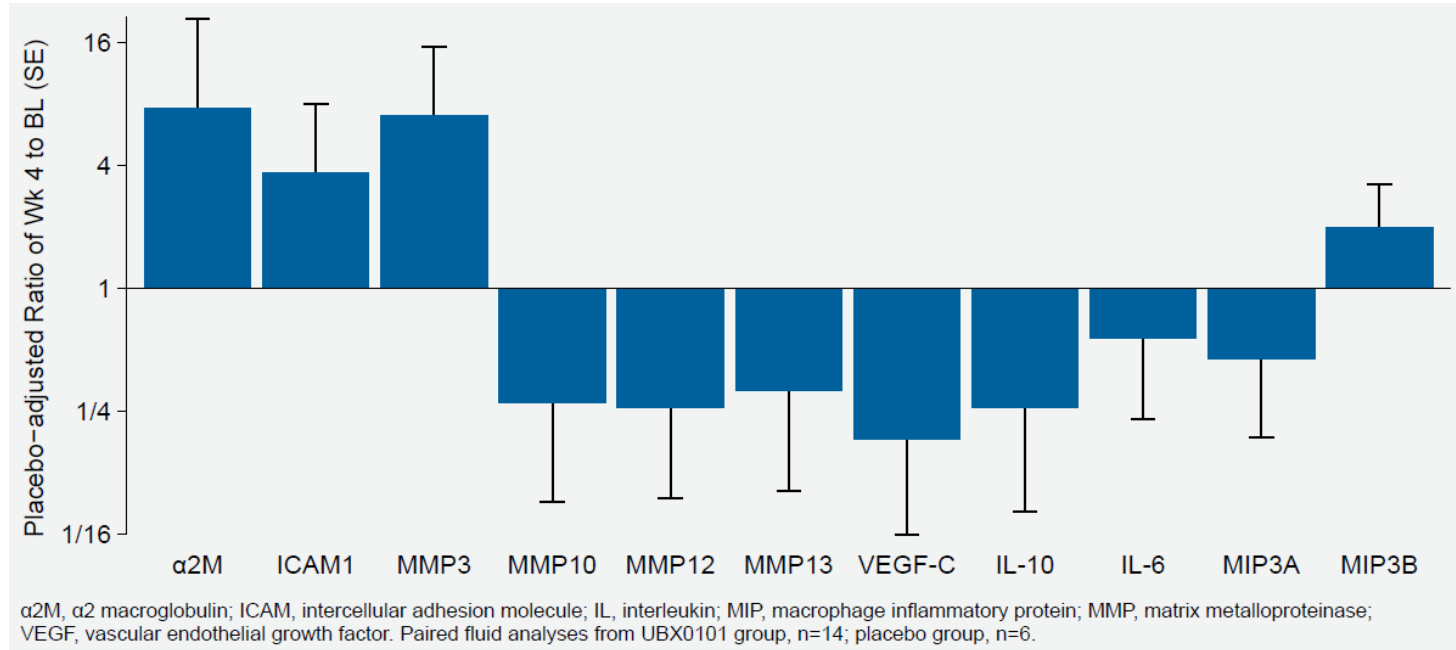
Selection Process

- 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

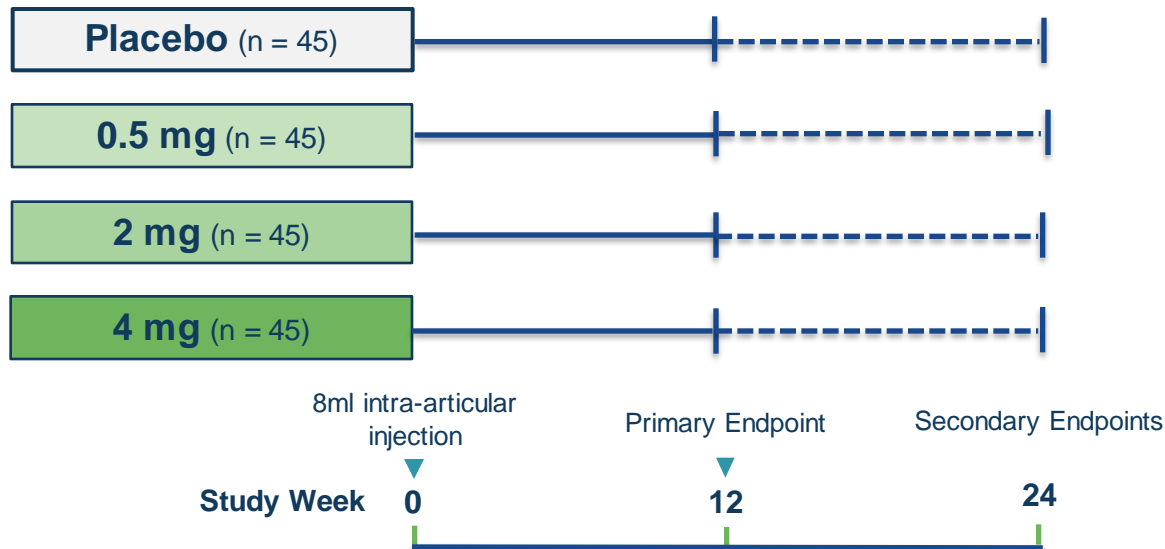


UBX0101 PHASE 2 STUDY DESIGN

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

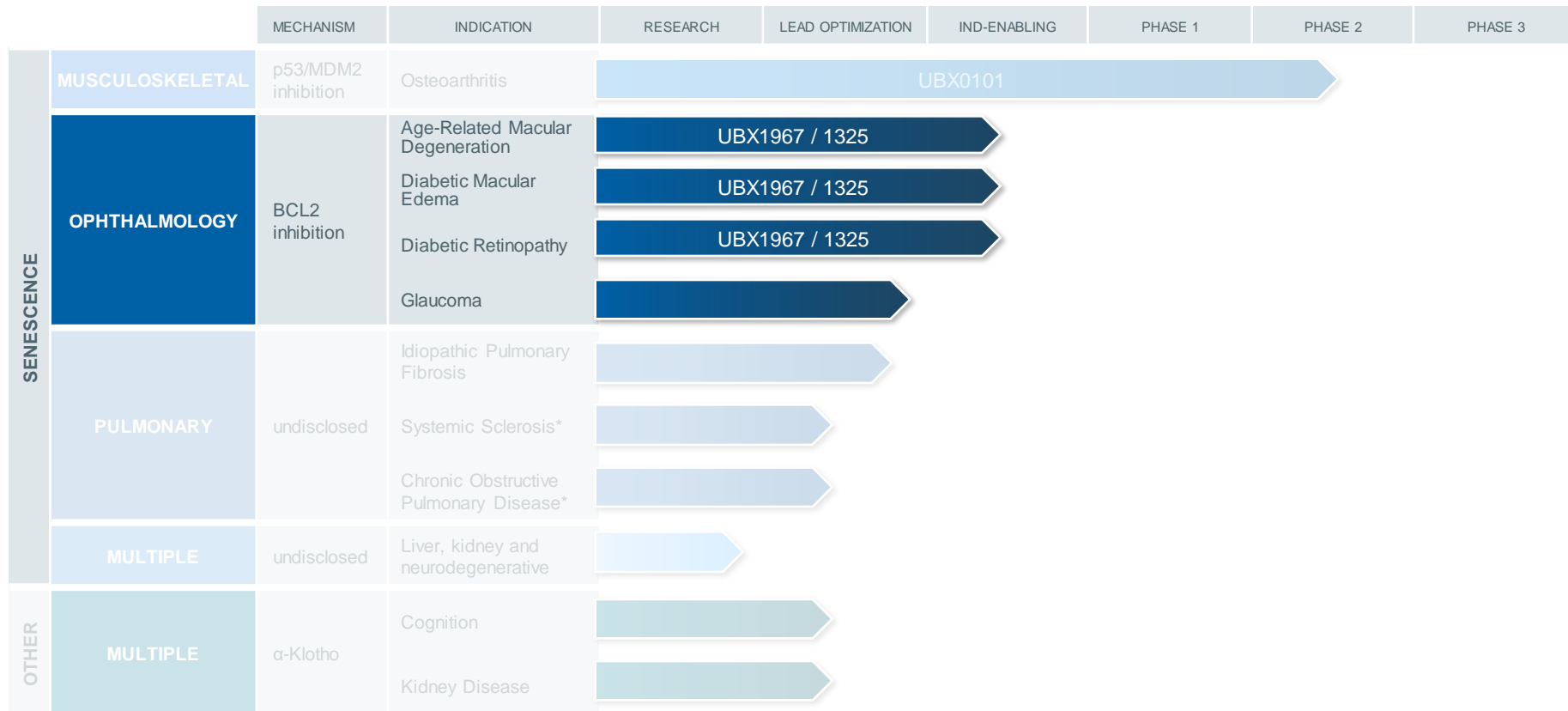
(AGE-RELATED EYE
DISEASE INDICATIONS)



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UNITY PIPELINE

Pursuing broad range of diseases with established endpoints and regulatory pathways



AGE-RELATED EYE DISEASES ARE SIGNIFICANT PUBLIC HEALTH BURDENS

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

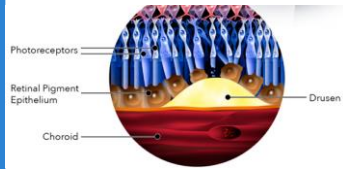
	AGE-RELATED MACULAR DEGENERATION (AMD)	DIABETIC RETINOPATHY (DR)	DIABETIC MACULAR EDEMA (DME)
GLOBAL IMPACT	<ul style="list-style-type: none"> Leading cause of visual disability in industrialized world; 3rd leading cause globally Aging is the greatest risk factor 	<ul style="list-style-type: none"> Leading cause of vision loss in middle-aged and elderly Impacts 8% of people >65 years old 	<ul style="list-style-type: none"> A manifestation of DR that is the primary cause of vision loss for people with diabetes
GLOBAL PREVALENCE	<ul style="list-style-type: none"> 170M people affected Expected to increase to 285M+ by 2040 	<ul style="list-style-type: none"> 90M+ people affected; 28M with vision-threatening disease stages ~33% of people with diabetes have signs of DR 	<ul style="list-style-type: none"> 20M+ people affected
CURRENT TREATMENTS	<ul style="list-style-type: none"> Anti-VEGF agents, laser therapy 	<ul style="list-style-type: none"> Diabetes control, anti-VEGF agents, laser photocoagulation 	<ul style="list-style-type: none"> Diabetes control, corticosteroids, anti-VEGF agents, laser photocoagulation
GLOBAL MARKET SIZE	<ul style="list-style-type: none"> >\$8B in global annual anti-VEGF sales 		

ROLE OF SENESENCE IN AGE-RELATED EYE DISEASE

SnCs accumulate in the retina, potentially contributing to disease phenotypes

AMD

SnCs accumulate with with age in the retina retinal pigment epithelium (RPE) layer

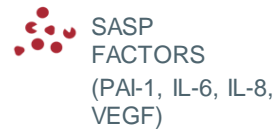


senescent cell

SASP → choroidal remodeling & RPE dysfunction → atrophy



senescence secretome



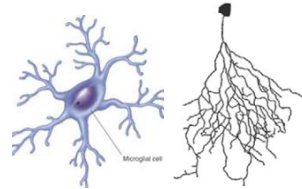
Disease → central vision loss



disease symptoms

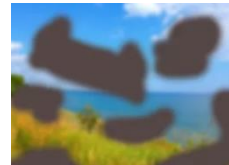
DR & DME

SnCs accumulate in the retina with age & diabetic disease

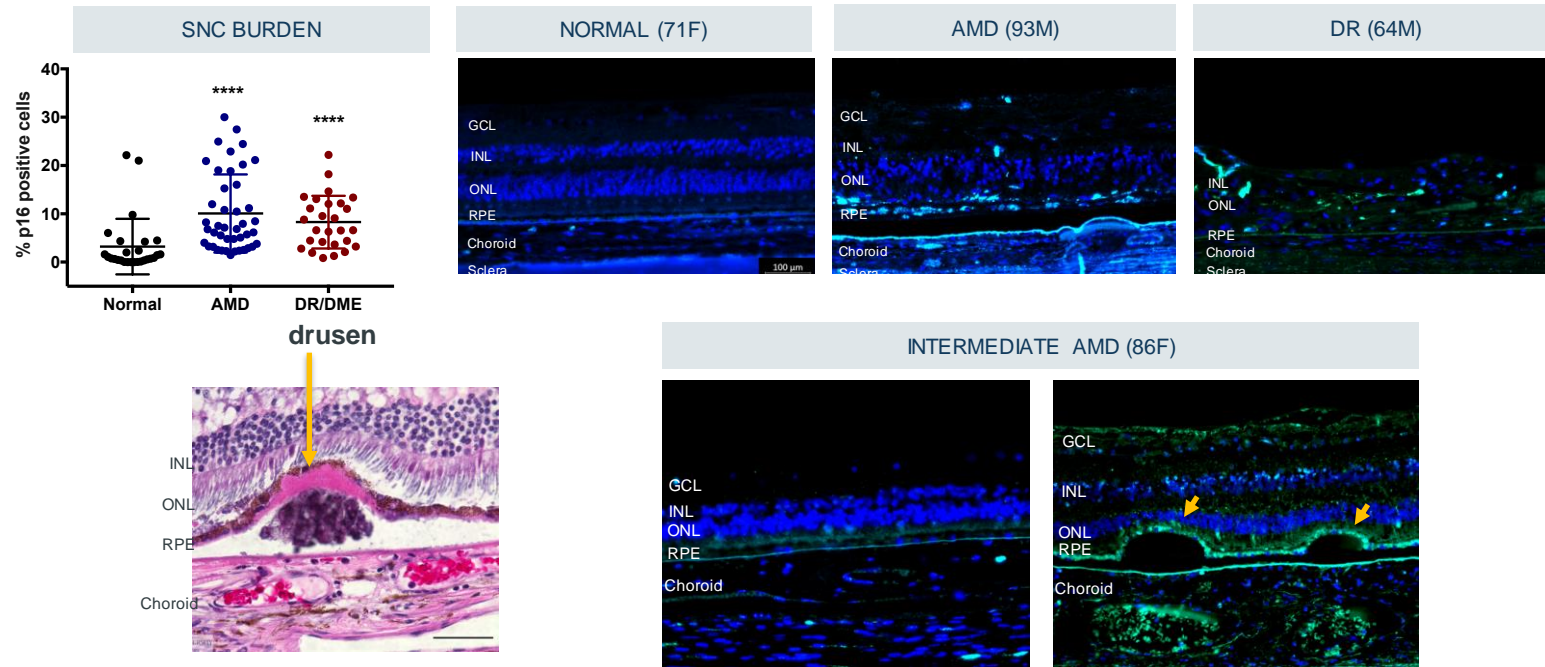


SASP → ocular inflammation, abnormal blood vessel growth

Disease → vision loss



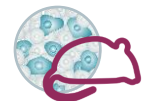
SENESCENCE BURDEN IN AMD AND DR/DME



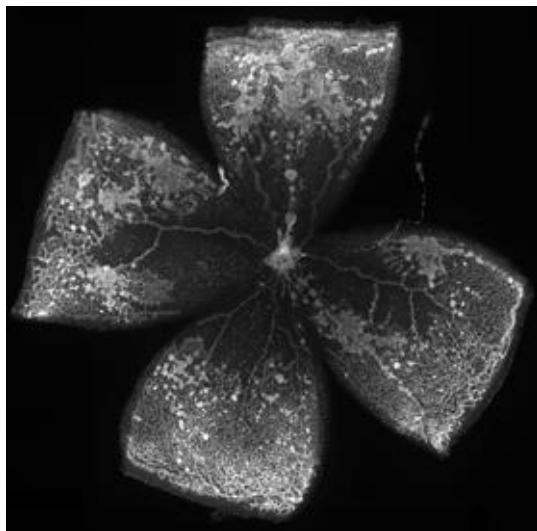
- Age-related eye diseases are multifactorial
- SnC burden increases with disease stage
- DR/DME patients show SnC in the retina and Choroid

UBX1967 DEMONSTRATES EFFICACY IN MOUSE OIR

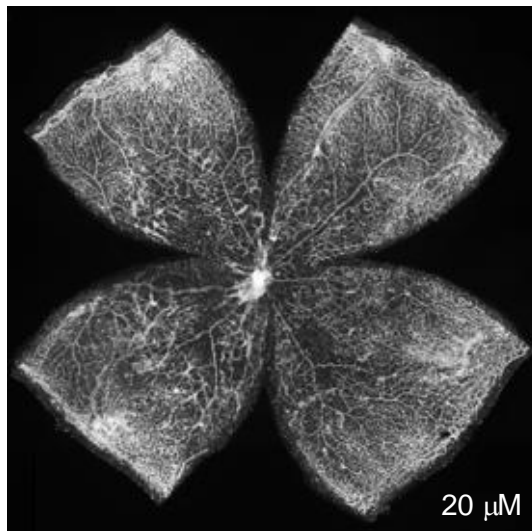
Oxygen induced retinopathy (OIR) model



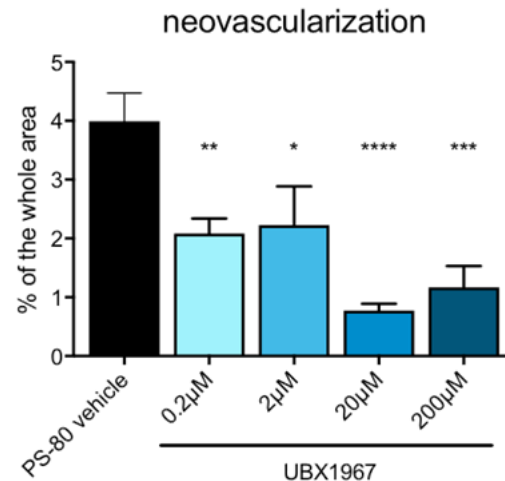
Vehicle



UBX1967



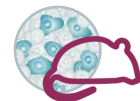
Improves Retinal Vasculature



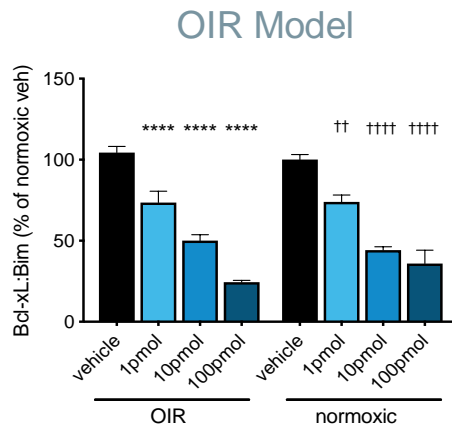
* p<0.05, ** p<0.01, *** p<0.001, **** p<0.0001 v. Vehicle; one-way ANOVA, with Dunnett's multiple comparison test

Intravitreal dosing improves retinal vasculature

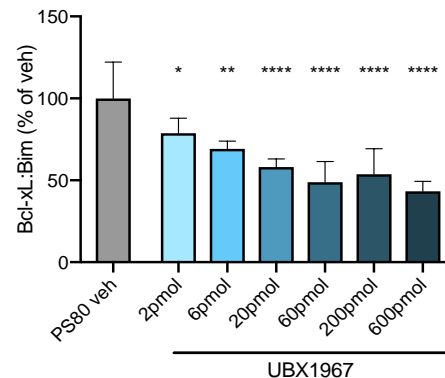
UBX1967 ENGAGES BCL-xL AND SELECTIVELY PROMOTES APOPTOSIS IN HYPEROXIC MOUSE RETINA



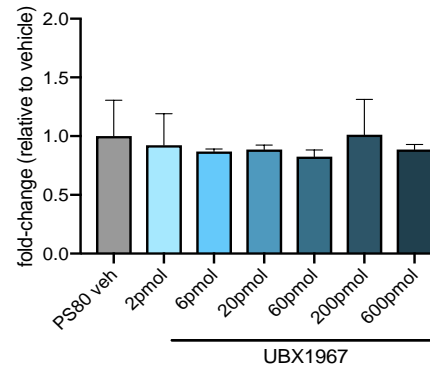
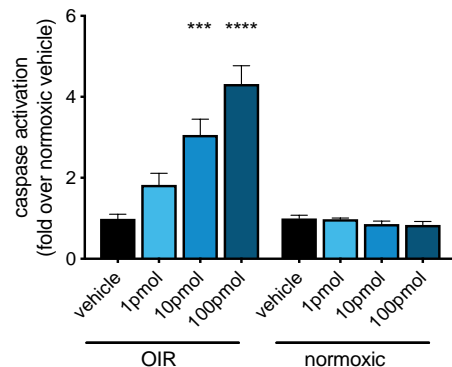
TARGET ENGAGEMENT



3 mo Healthy



MECHANISM ENGAGEMENT



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

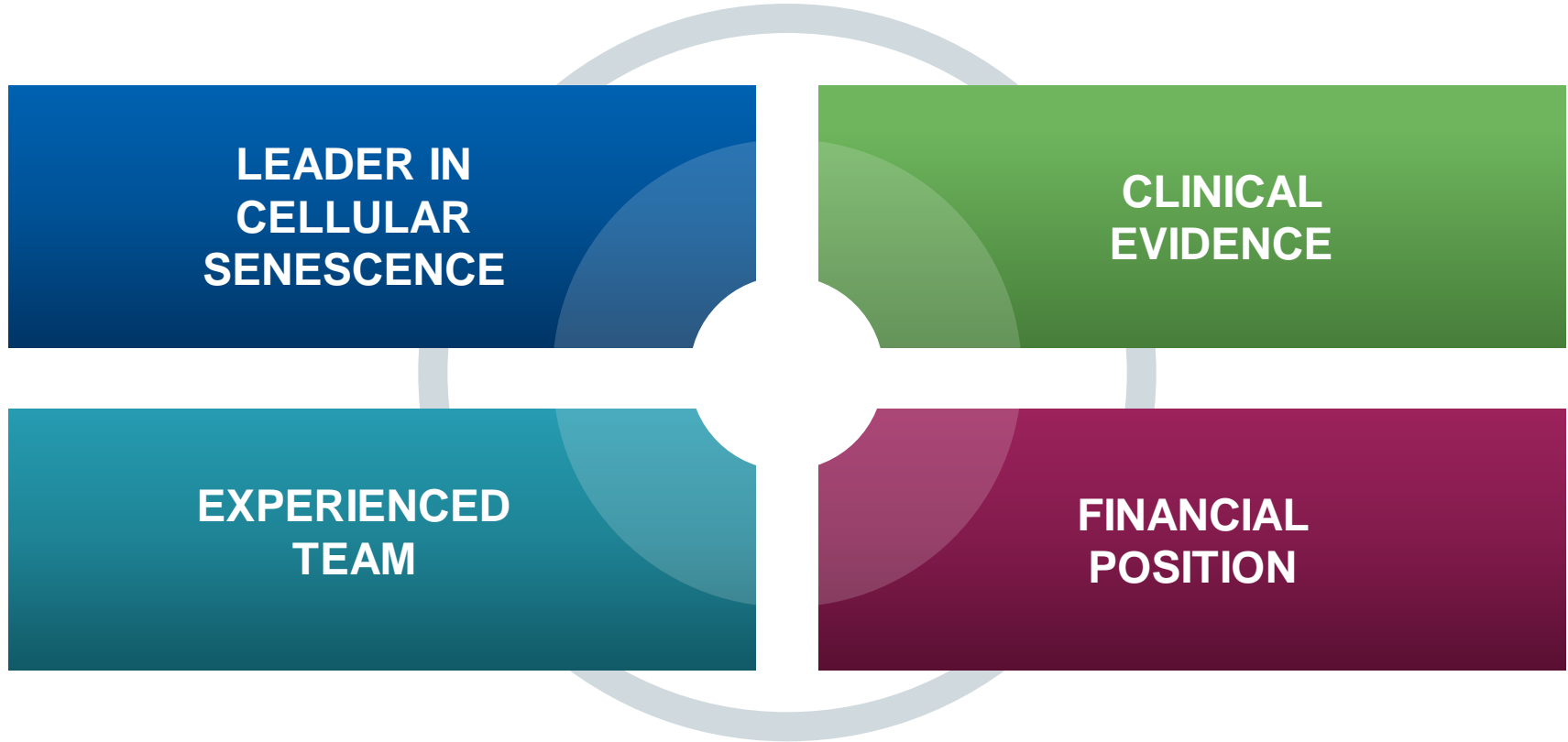
FINANCIAL

- \$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



NATHANIEL DAVID, PHD
President



DAN MARQUESS, D. PHIL
Chief Scientific Officer



JAMIE DANANBERG, MD
Chief Medical Officer



BOB GOELTZ, CPA, MBA
Chief Financial Officer



TAMMY TOMPKINS, JD
General Counsel



SUSIE LUNDEEN
SVP of People



PEDRO BELTRAN, PHD
SVP of Biology



DOUG RICH, MBA
SVP, Operations

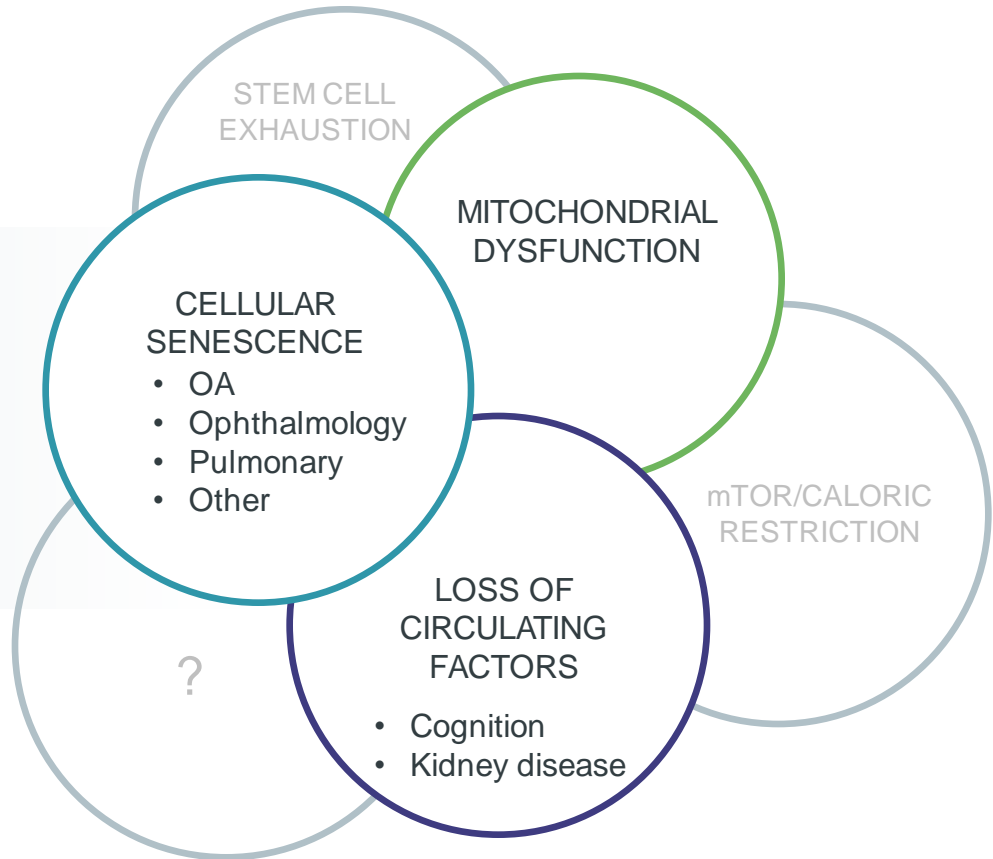


CAMILLE LANDIS, MBA
SVP, Corporate Development



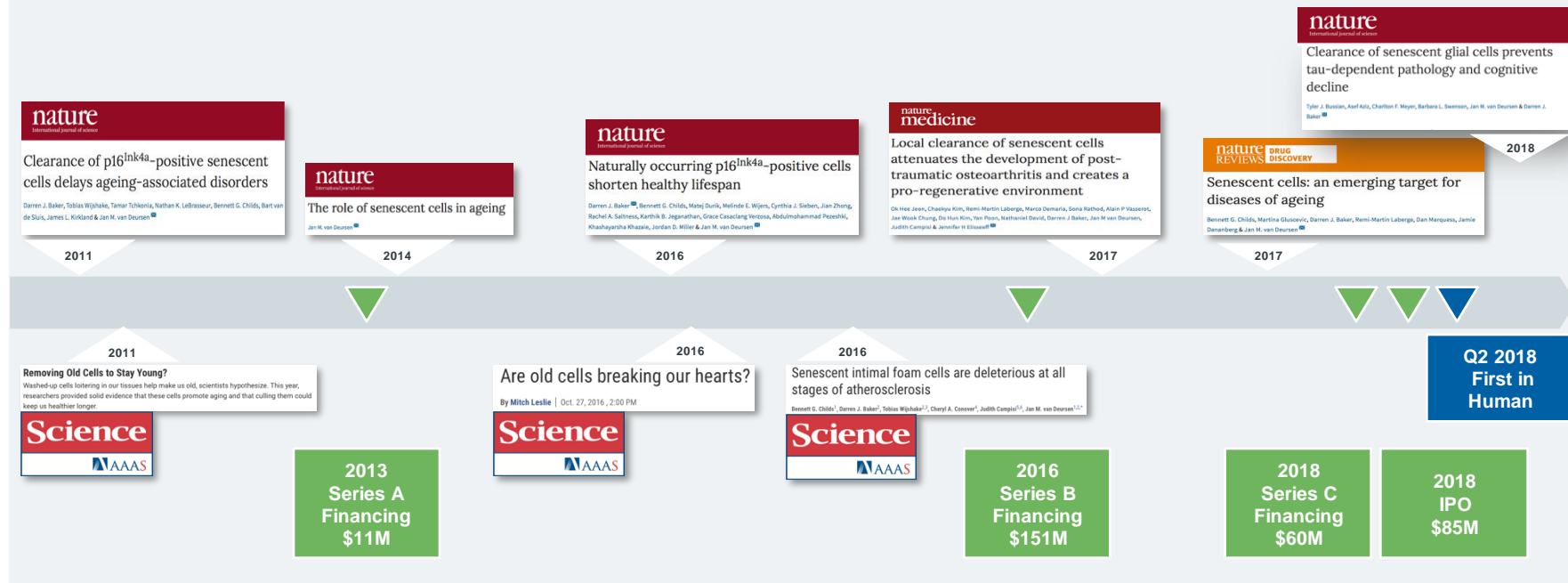
MULTIPLE MECHANISMS DRIVE AGING

UNITY is pursuing multiple pathways to impact the aging process



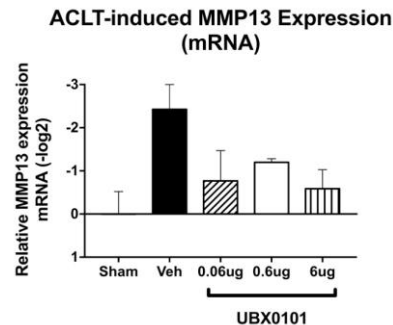
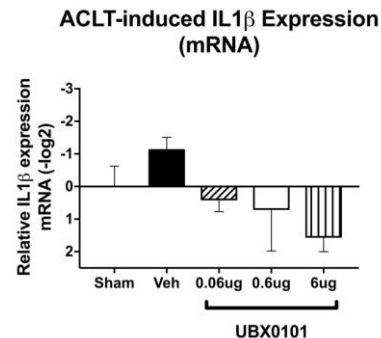
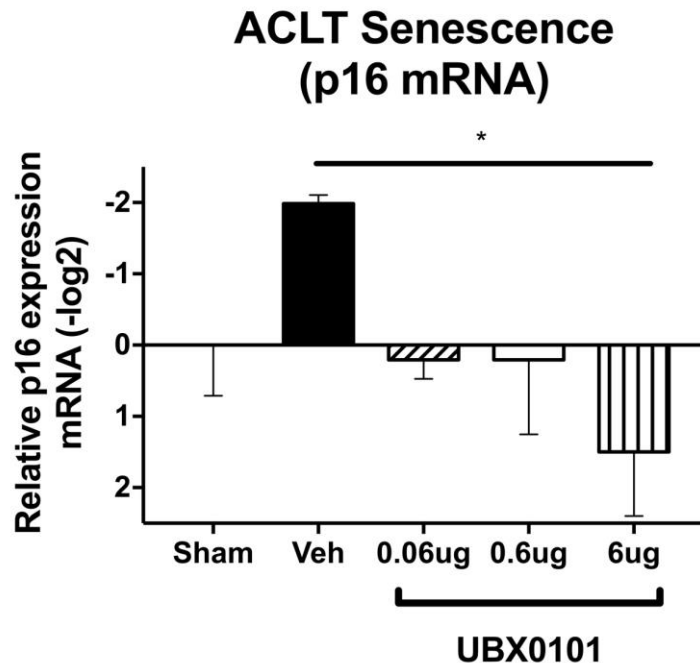
UNITY: ESTABLISHING LEADERSHIP IN HEALTHSPAN

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



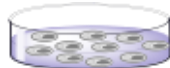
* p -value $\leq .05$

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

p16

MMP13

VEHICLE



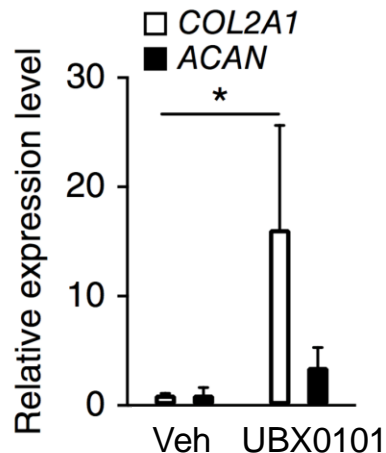
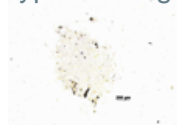
UBX0101



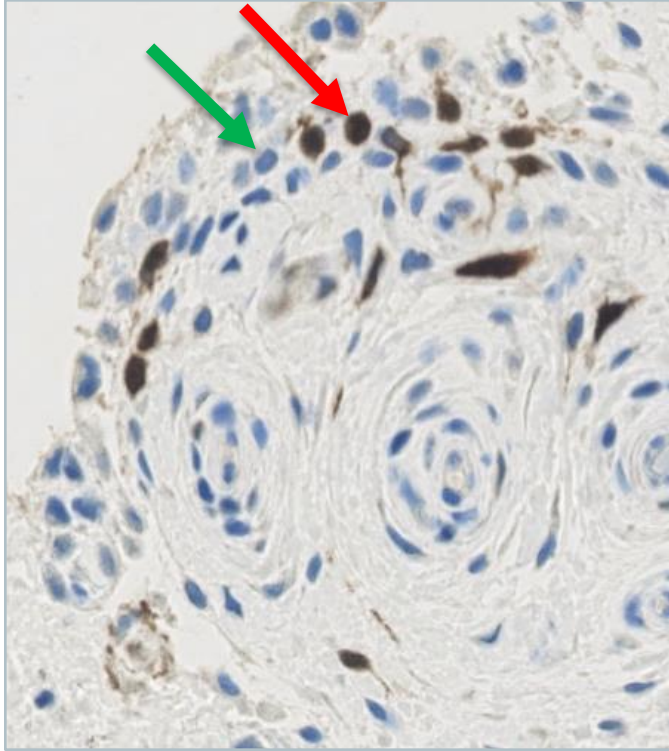
CARTILAGE MARKERS

Type 2 collagen

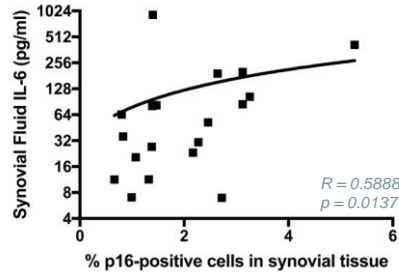
Aggrecan



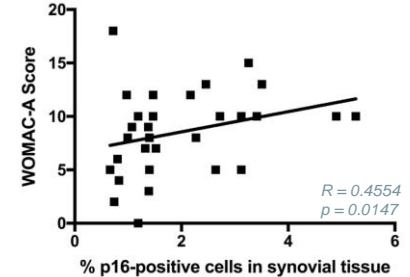
INCREASED SnCs OBSERVED IN KNEES OF OA PATIENTS



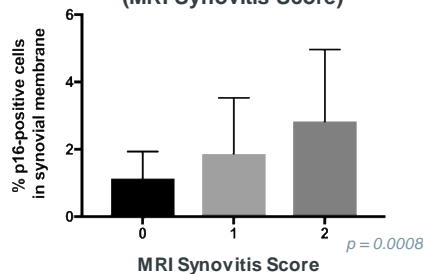
Relationship between degree of senescence (p16) and synovial fluid SASP Factors (IL-6)



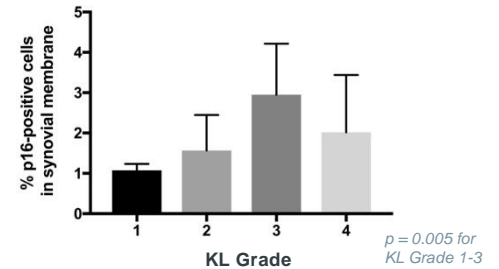
Relationship between degree of senescence (p16) and patient reported pain scores (WOMAC-A)



Relationship between degree of senescence (p16) and synovial membrane inflammation (MRI Synovitis Score)



Relationship between degree of senescence (p16) and stage of OA disease (KL Grade)



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)							Part B	
	Mean (SD)							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.

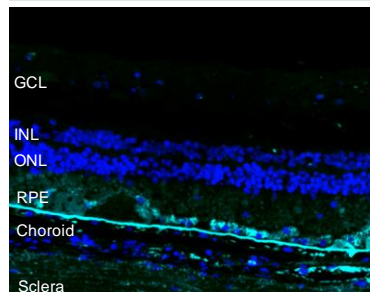
	WOMAC-A		WOMAC-C	
	CFBL	Pbo-Adj (P-value)	CFBL	Pbo-Adj (P-value)
Placebo (n=10)	-0.72		-0.60	
4.0 mg (n=20)	-0.87	-0.15 (0.62)	-0.77	-0.17 (0.60)

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

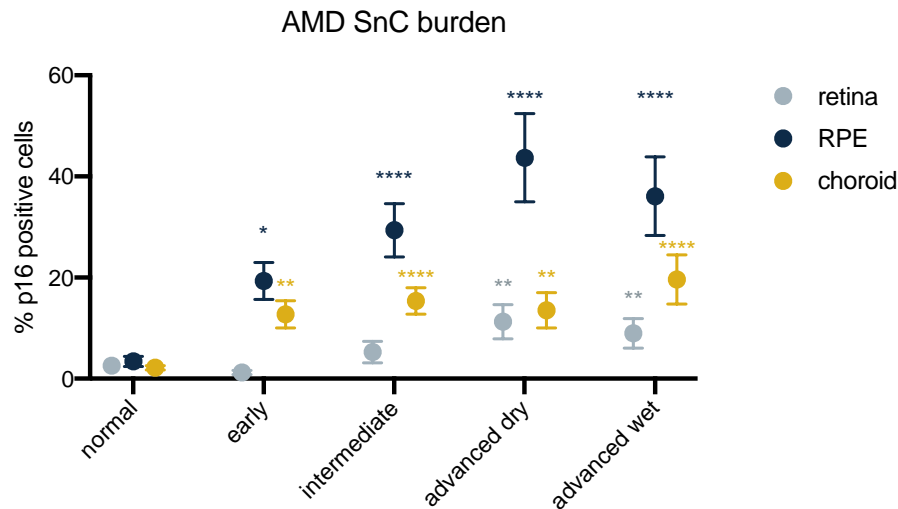
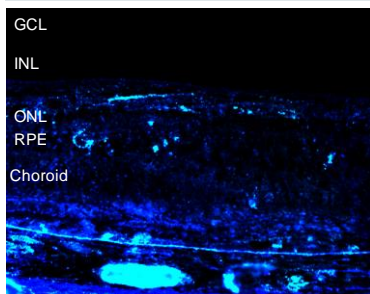
SNC BURDEN INCREASES WITH DISEASE STAGE



INTERMEDIATE (86F)

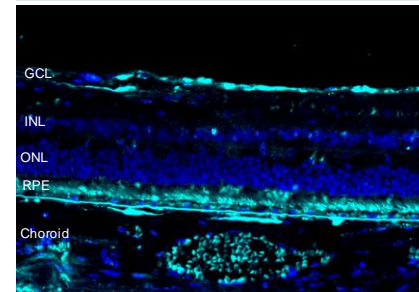


EARLY (89M)

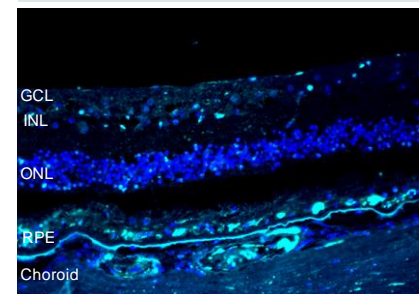


*p<0.05; **p<0.01; ****p<0.0001 v. Normal by Kruskal-Wallis with Dunn's multiple comparisons test

ADVANCED DRY (93F)

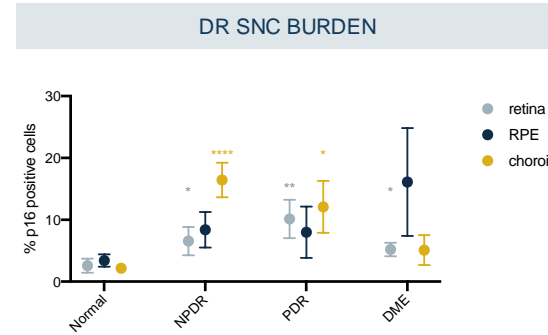
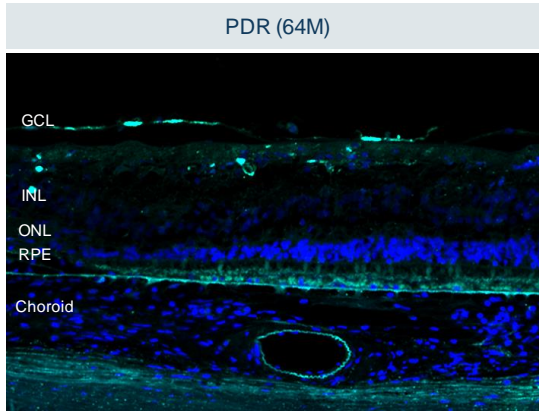
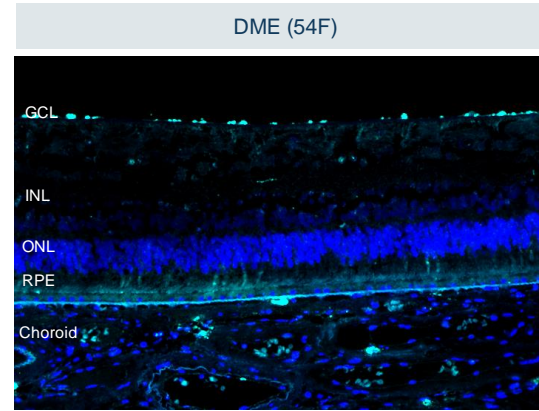
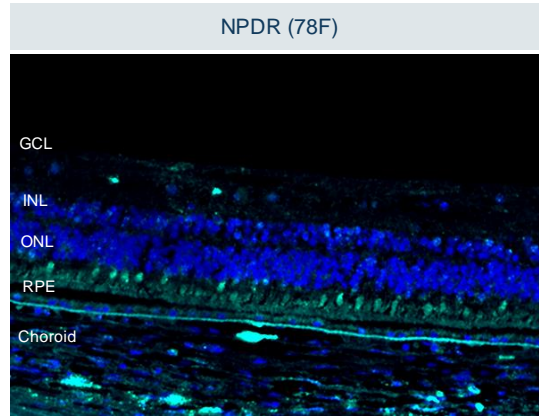


ADVANCED WET (93M)

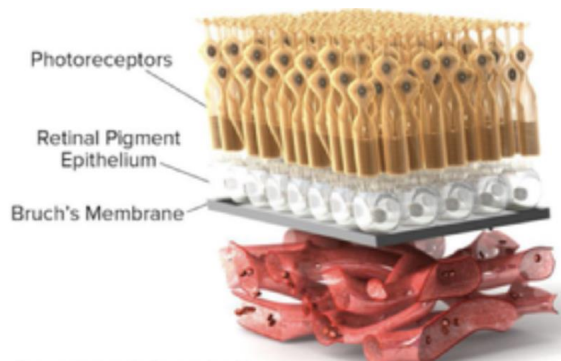


Senolysis has the opportunity to halt disease progression from an early stage

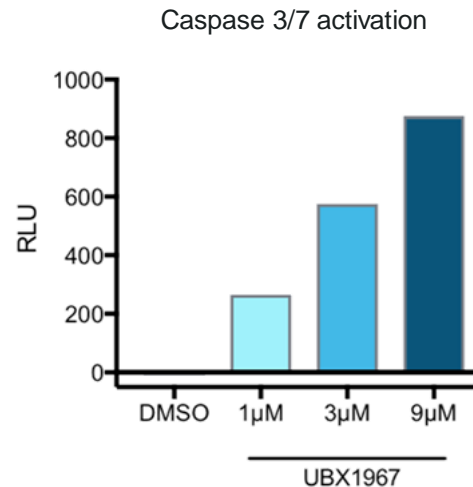
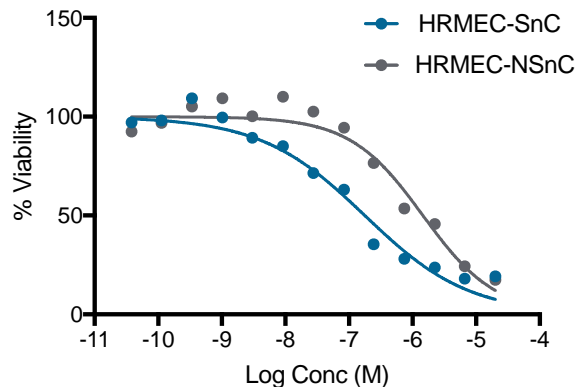
DR/DME PATIENTS SHOW SNC IN THE RETINA AND CHOROID



HIGHLY POTENT SENOLYTIC THAT TARGETS BCL-2 FAMILY PROTEINS



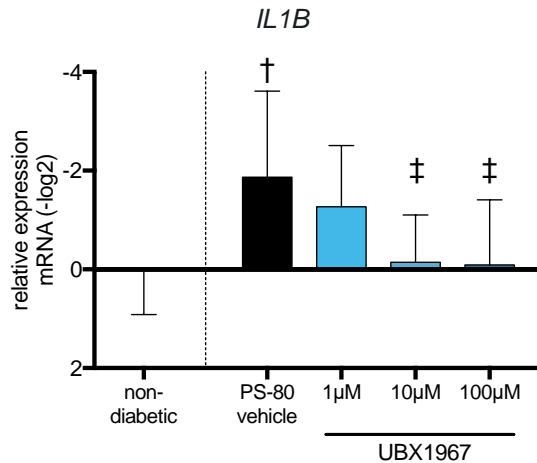
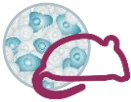
Human retinal microvascular endothelial cells (HRMEC)



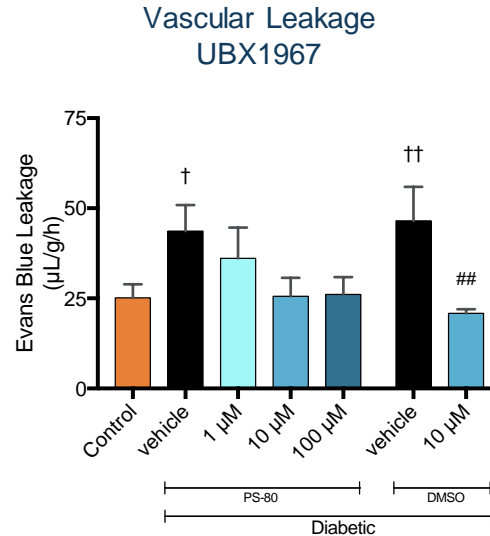
UBX1967 selectively eliminates HRMEC-SnCs over non-SnCs

UBX1967 DEMONSTRATES EFFICACY IN MOUSE STZ

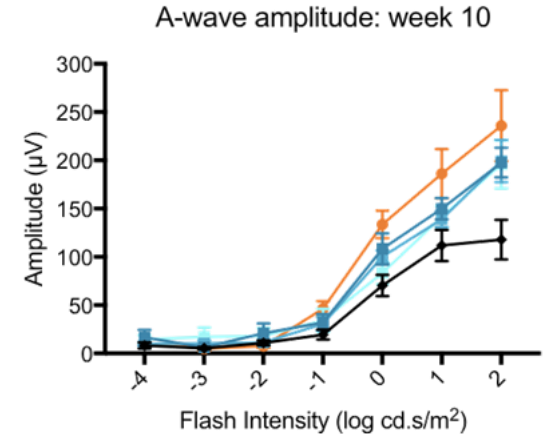
Streptozotocin (STZ) diabetic retinopathy model



† 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



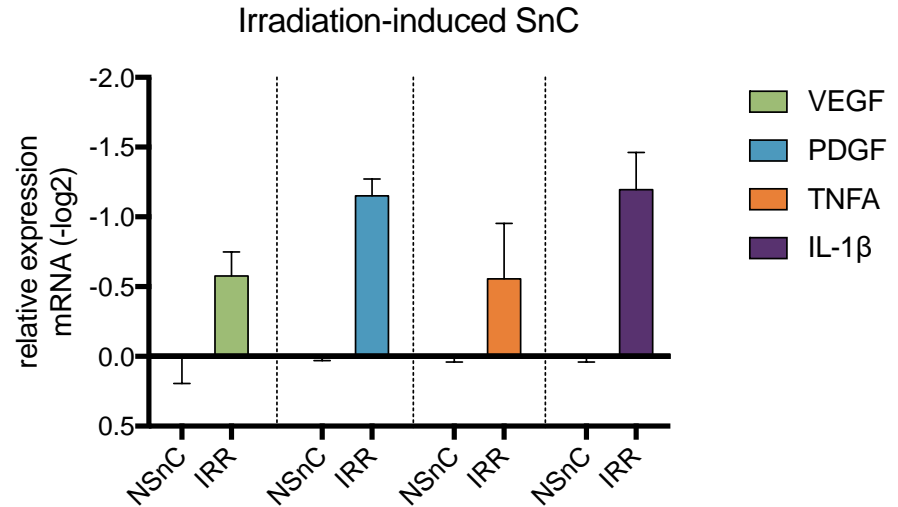
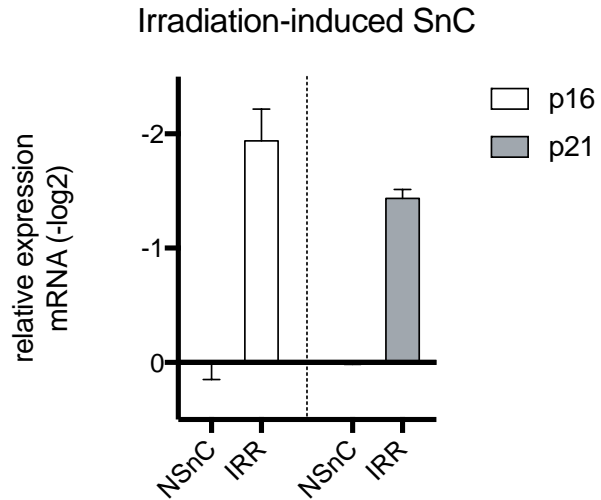
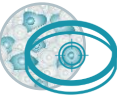
† p<0.05 v. Non-diabetic control by two-tailed t-test
†† p<0.01 v. Non-diabetic control by two-tailed t-test
p<0.01 v. DMSO control by two-tailed t-test



**** 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

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

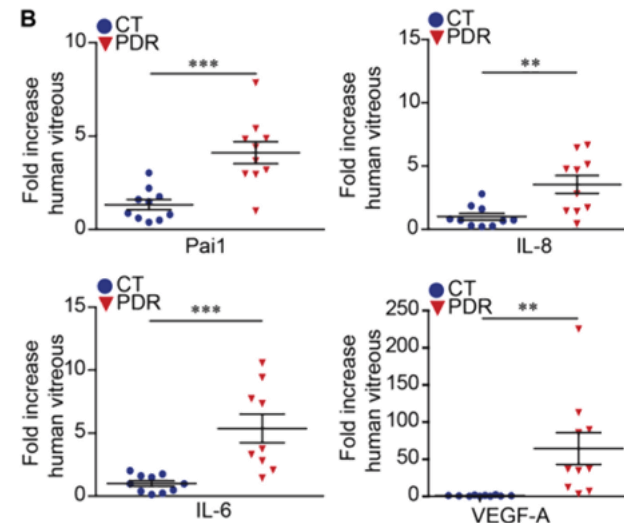
	nAMD	Controls	P value
	Level	Level	Pre IVA
	mean \pm SD	mean \pm SD	vs Control
IL-1 α	0	0.77 \pm 3.19	0.381
IL-6	6.51 \pm 5.24	78.2 \pm 100	0.0029
IL-7	10.9 \pm 3.95	13.5 \pm 12.8	0.465
IL-8	6.00 \pm 6.69	6.43 \pm 6.84	0.448
IL-9	0.087 \pm 0.40	0.10 \pm 0.41	0.483
IL-12	12.1 \pm 5.79	10.4 \pm 9.11	0.268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