EXTEND HEALTHSPAN

March 2019

UNITY
BIOTECHNOLOGY

As of March 6, 2019

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 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 Annual Report on Form 10-K for the year ended December 31, 2018, filed with the Securities and Exchange Commission on March 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.

Health·span |helth' span| *noun*

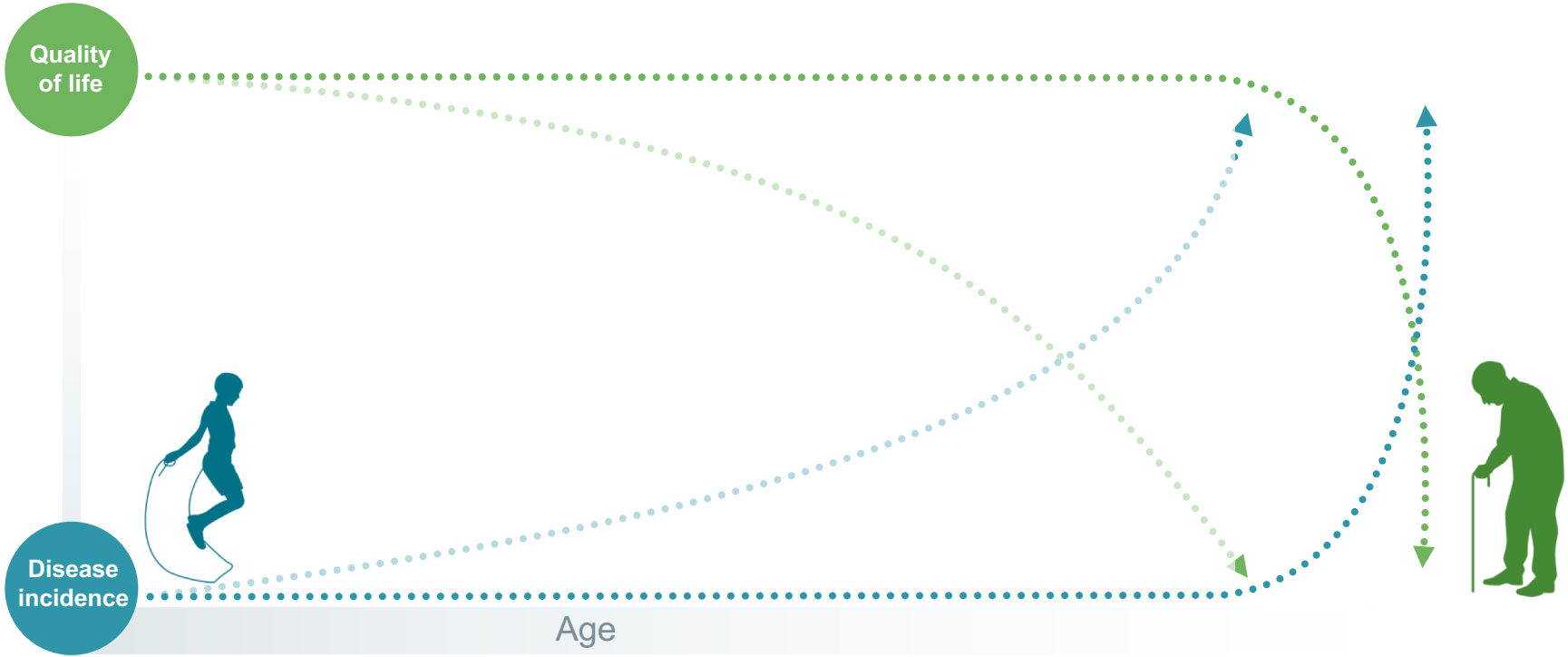
The period of one's life unburdened by the diseases of aging

See also: anti-aging, healthy longevity

HEALTHSPAN

UNITY BIOTECHNOLOGY

UNITY IS ADVANCING THERAPIES TO EXTEND 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 · 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

UNITY BIOTECHNOLOGY

A NEW HEALTH PARADIGM

UNITY is committed to reshaping human healthspan

BREAKTHROUGH SCIENCE	• Broad approach to healthspan with initial efforts focused on cellular senescence • Cutting edge science published in *Science* and *Nature* • Tractable molecular targets with a clear tie to disease phenotypes • IP portfolio covering senolytic approach, key pathways, target indications and molecules
COMPELLING OPPORTUNITY	• Many disease phenotypes with large unmet need • Phase 1 study of UBX0101 in patients with osteoarthritis ongoing; data expected Q2 2019 • Ophthalmology IND expected in early 2020 enabling multiple indications
EXPERIENCED TEAM	• Seasoned executive team with broad biotech experience • Strong track record of delivering for patients and investors
STRONG FINANCIAL POSITION	• Cash equivalents and investments balance of $171.1 million as of December 31, 2018

UNITY PIPELINE

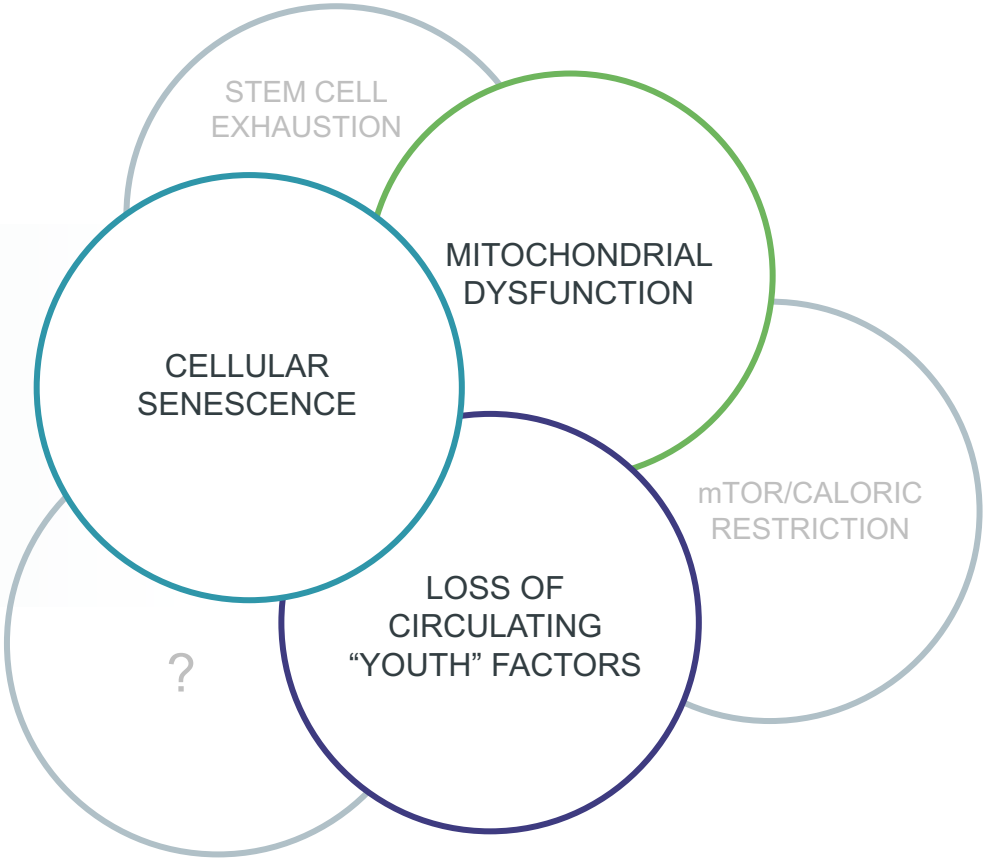
Broad therapeutic potential, addressing multiple mechanisms of aging

*Following IPF Outcome

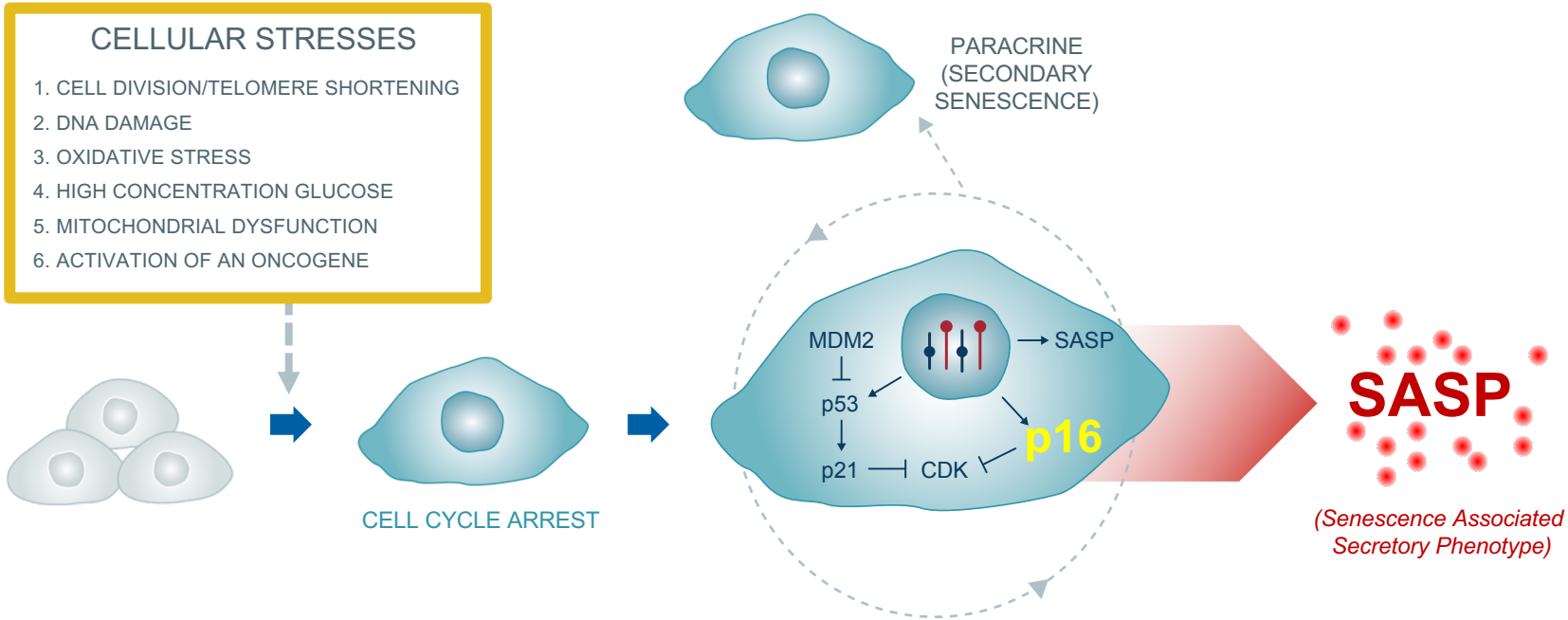
 As of March 6, 2019

MULTIPLE MECHANISMS DRIVE AGING

UNITY is pursuing multiple pathways to impact the aging process

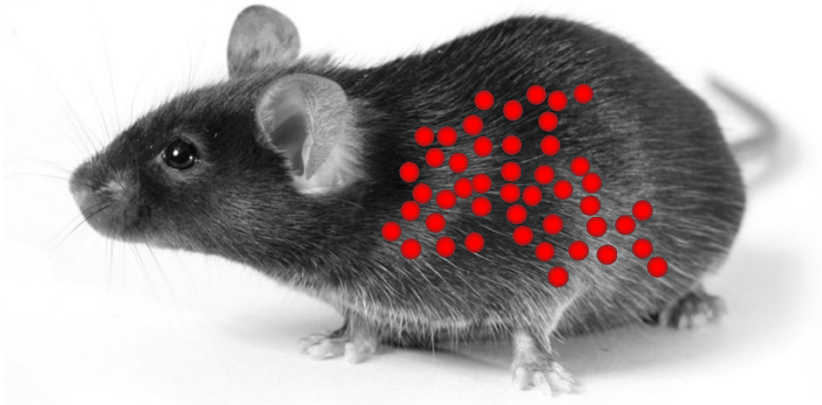


CELLULAR STRESSES TRIGGER SENESCENCE



THE BREAKTHROUGH THAT CREATED A FIELD

UNITY's proprietary models revealed the relevance of SnC clearance to the aging process



nature
International weekly journal of science

LETTER

doi:10.1038/nature10600

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

Darren J. Baker[1,2,3], Tobias Wijshake[1,4], Tamar Tchkonia[3], Nathan K. LeBrasseur[3,5], Bennett G. Childs[3], Bart van de Sluis[4], James L. Kirkland[3] & Jan M. van Deursen[1,2,3]

Advanced age is the main risk factor for most chronic diseases and functional deficits in humans, but the fundamental mechanisms that drive ageing remain largely unknown, impeding the development of interventions that might delay or prevent age-related disorders and maximize healthy lifespan. Cellular senescence, which halts the proliferation of damaged or dysfunctional cells, is an important mechanism to constrain the malignant progression of tumour cells[1,2]. Senescent cells accumulate in various tissues and organs with ageing[3] and have been hypothesized to disrupt tissue structure and function because of the components they secrete[4,5]. However, whether senescent cells are causally implicated in age-related dysfunction and whether their removal is beneficial has remained unknown. To address these fundamental questions, we made use of a biomarker for senescence, p16^{Ink4a}, to design a novel transgene, *INK-ATTAC*, for inducible elimination of p16^{Ink4a}-positive senescent cells upon administration of a drug. Here we show that in the BubR1 progeroid mouse background, *INK-ATTAC* removes p16^{Ink4a}-positive senescent cells upon drug treatment. In tissues—such as adipose tissue, skeletal muscle and eye—

we bred each of the founder lines onto a *BubR1* hypomorphic (*BubR1*$^{H/H}$) genetic background. *BubR1* encodes a key member of the mitotic checkpoint, a surveillance mechanism that ensures accurate chromosome segregation in mitosis by inhibiting the ubiquitin ligase activity of Cdc20-activated anaphase-promoting complex (APC^{Cdc20}) in the presence of unattached chromosomes[10,11]. *BubR1*$^{H/H}$ mice have a markedly shortened lifespan and exhibit a variety of age-related phenotypes, including infertility, lordokyphosis, sarcopenia, cataracts, fat loss, cardiac arrhythmias, arterial wall stiffening, impaired wound healing and dermal thinning[12–14]. It has been proposed that BubR1 is a determinant of natural ageing, because levels of BubR1 decline markedly with age[12–14]. *BubR1*$^{H/H}$ mice selectively accumulate p16^{Ink4a}-positive cells in certain tissues in which age-associated pathologies develop, including adipose tissue, skeletal muscle and eye[15]. Inactivation of *p16*^{Ink4a} in these mice is known to delay the onset of age-related phenotypes selectively in these tissues[15]. To screen for *INK-ATTAC* transgene activity in p16^{Ink4a}-positive cells, we collected samples of inguinal adipose tissue (IAT) from each of the nine *BubR1*$^{H/H}$;*INK-ATTAC* strains at 5 months of age and analysed them for GFP expression by fluorescence microscopy. We

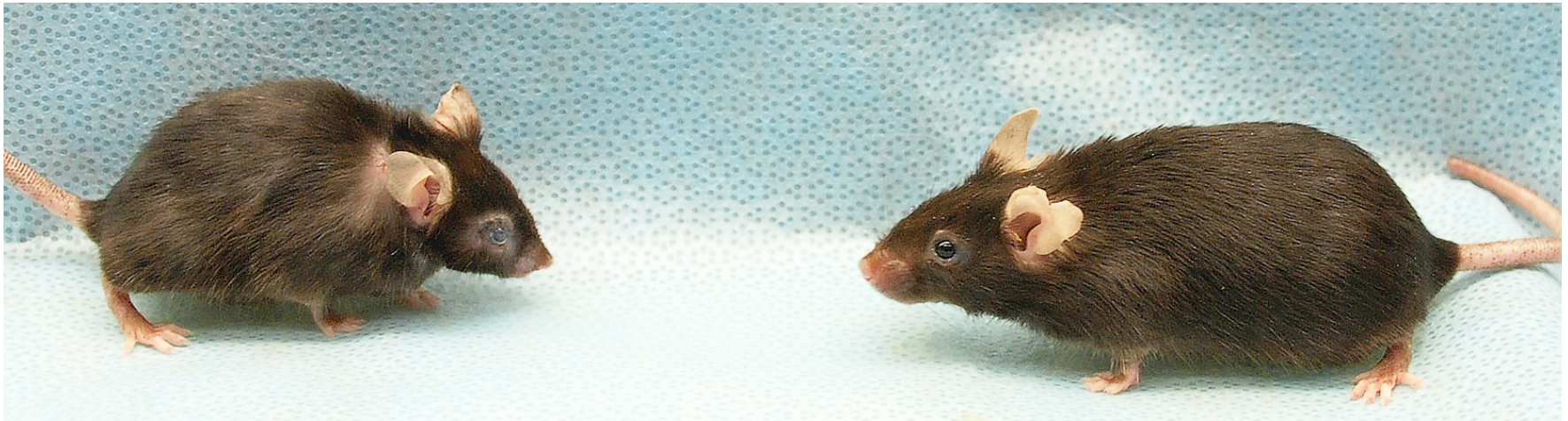
MAYO CLINIC

BUCK INSTITUTE FOR RESEARCH ON AGING

UNITY's founding scientific team was the first group to demonstrate that the removal of senescent cells significantly slowed aging

UNITY BIOTECHNOLOGY

CLEARING SnCs: SIGNIFICANT IMPACT ON AGING



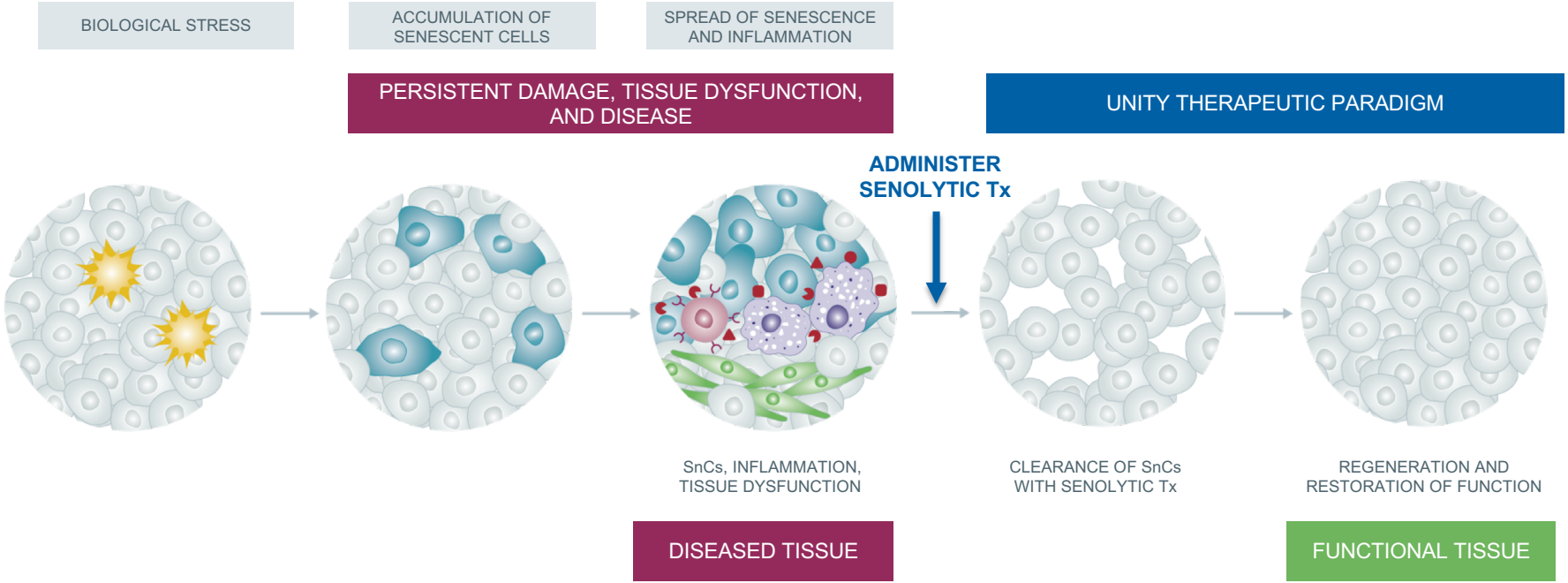
- Kidney dysfunction
- Cardiac dysfunction
- Cardiac hypertrophy
- Frailty
- Cataracts
- Kyphosis
- Loss of subcutaneous fat
- Sarcopenia
- Reduced locomotion
- Cancer

SIGNIFICANT EXTENSION OF HEALTHSPAN

(AND UP TO 35% INCREASE IN MEDIAN LIFESPAN)

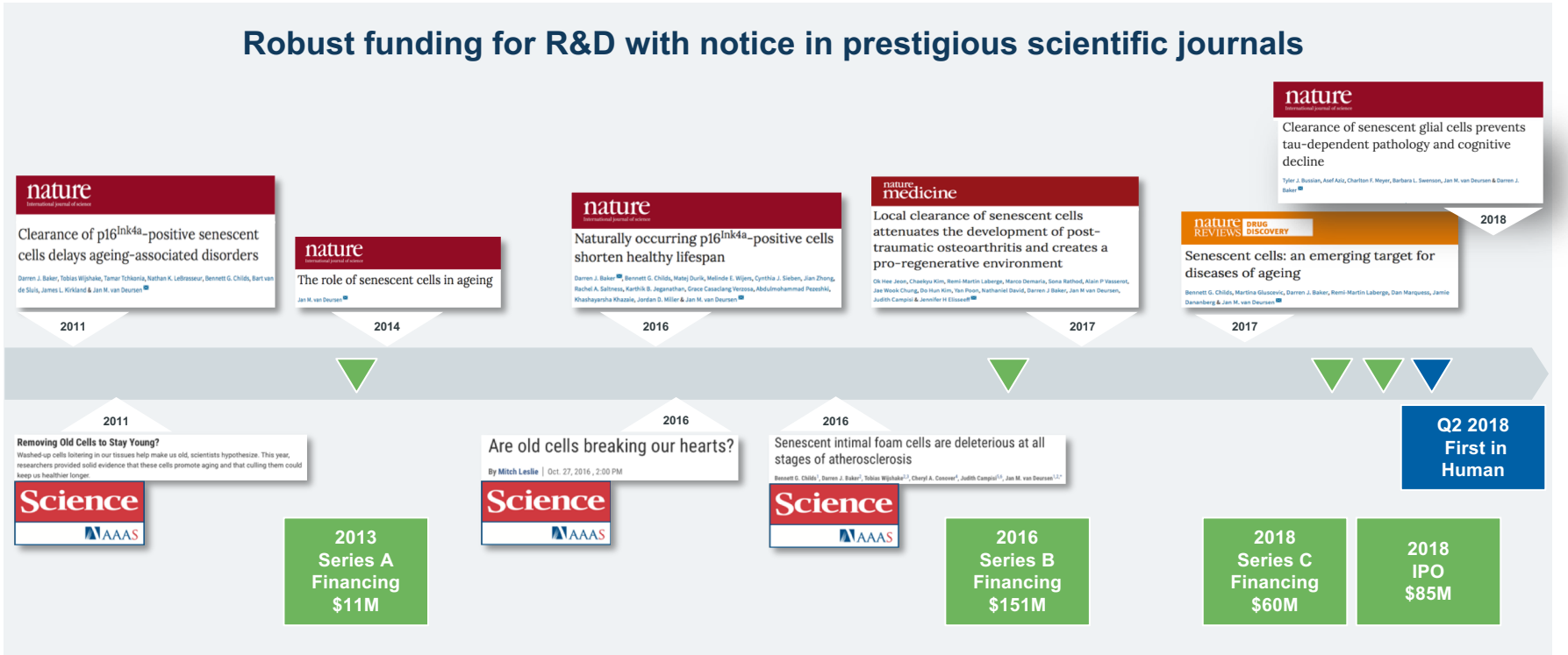
Baker *et al*., Clearance of p16Ink4a-positive senescent cells delays ageing-associated disorders. *Nature* 2011, 2011;479:232-236.
Baker *et al*., Naturally occurring p16Ink4a-positive cells shorten healthy lifespan. *Nature* 2016, 2016;530:184-190.

THE UNITY THERAPEUTIC PARADIGM



UNITY: ESTABLISHING LEADERSHIP IN HEALTHSPAN

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



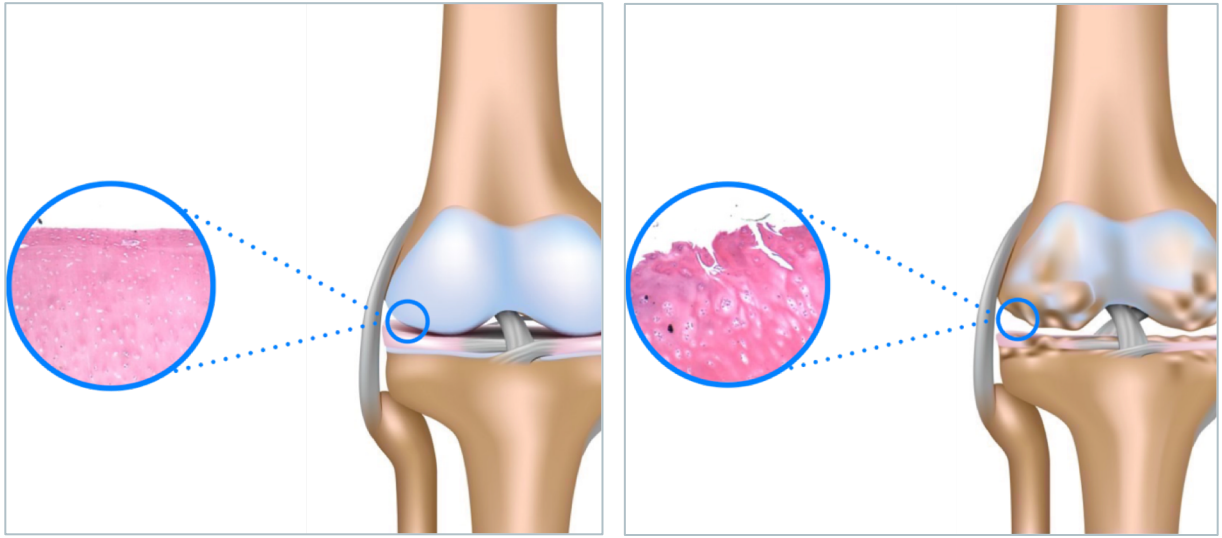
OSTEOARTHRITIS

(MUSCULOSKELETAL INDICATION)

UNITY BIOTECHNOLOGY

OSTEOARTHRITIS

A widespread disease; standard of care is pain mitigation or joint replacement



~10-15% of global population over 60 years of age

Aggregate annual expense associated with OA estimated at >$150B per year in US

Current treatments are NSAIDs, steroids, knee replacement, and acquiescence

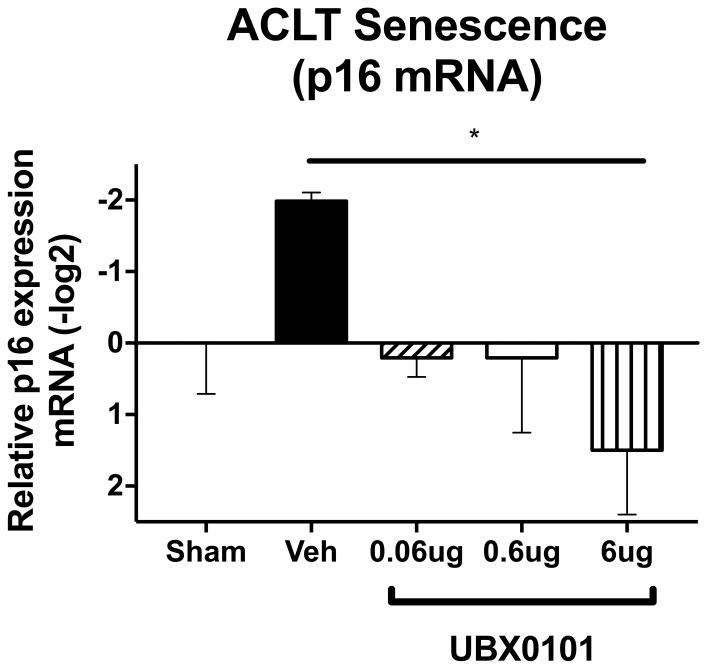
OPPORTUNITY FOR OA DISEASE MODIFICATION

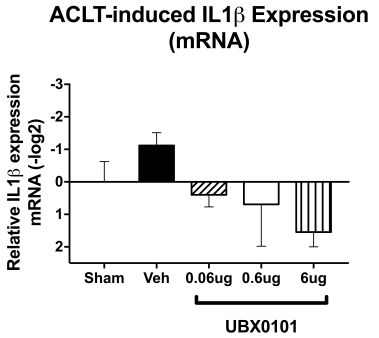
The promise of a senolytic therapy

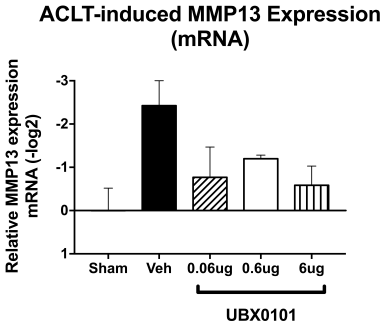
MECHANISM	Novel mechanism to address root cause of OA, ***remove source of multiple SASP factors driving disease***
Tx IMPACT	Potential to be ***1st disease-modifying therapy for OA***; slow, halt or reverse disease, decrease pain and improve function
Tx FREQUENCY	Senolytic treatment likely requires ***infrequent, intermittent dosing***

UBX0101 EFFICACY *IN VIVO*

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







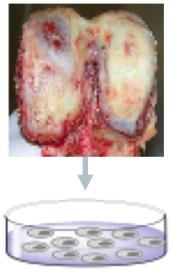
* *p*-value ≤ .05

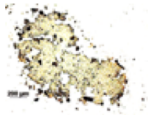
IA dosing of UBX0101 eliminates p16^{+} SnCs and reduces OA-relevant SASP factors

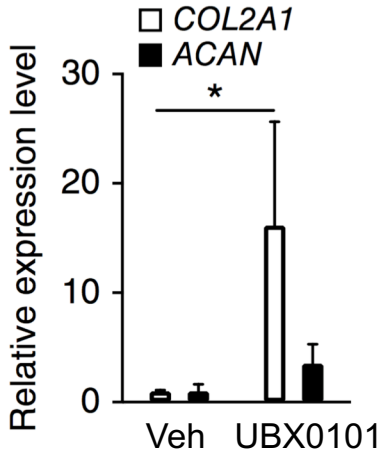
UBX0101 EFFICACY *EX VIVO*

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

Human OA
chondrocyte isolation

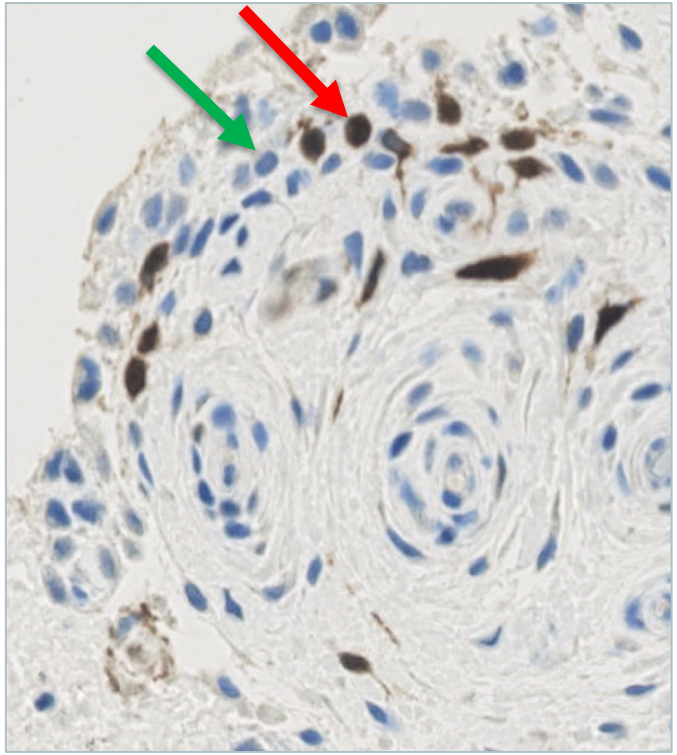


	SnC MARKERS		CARTILAGE MARKERS	
	p16	MMP13	Type 2 collagen	Aggrecan
VEHICLE				
UBX0101				

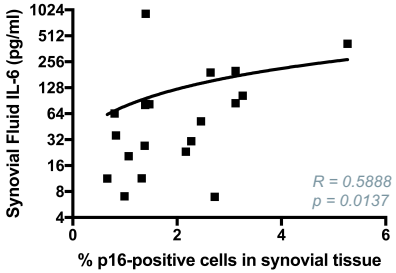


As of March 6, 2019

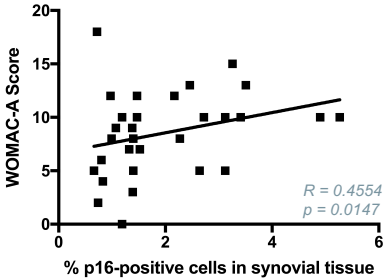
INCREASED SnCs OBSERVED IN KNEES OF OA PATIENTS



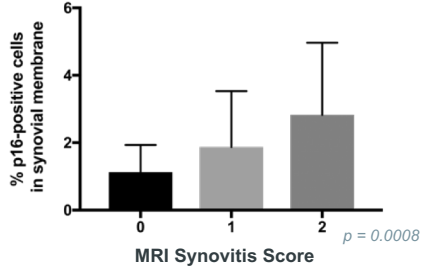
Relationship between degree of senescence (p16) and synovial fluid SASP Factors (IL-6)



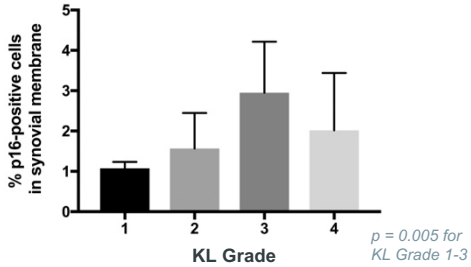
Relationship between degree of senescence (p16) and patient reported pain scores (WOMAC-A)



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

UBX0101 PHASE 1 PROGRAM

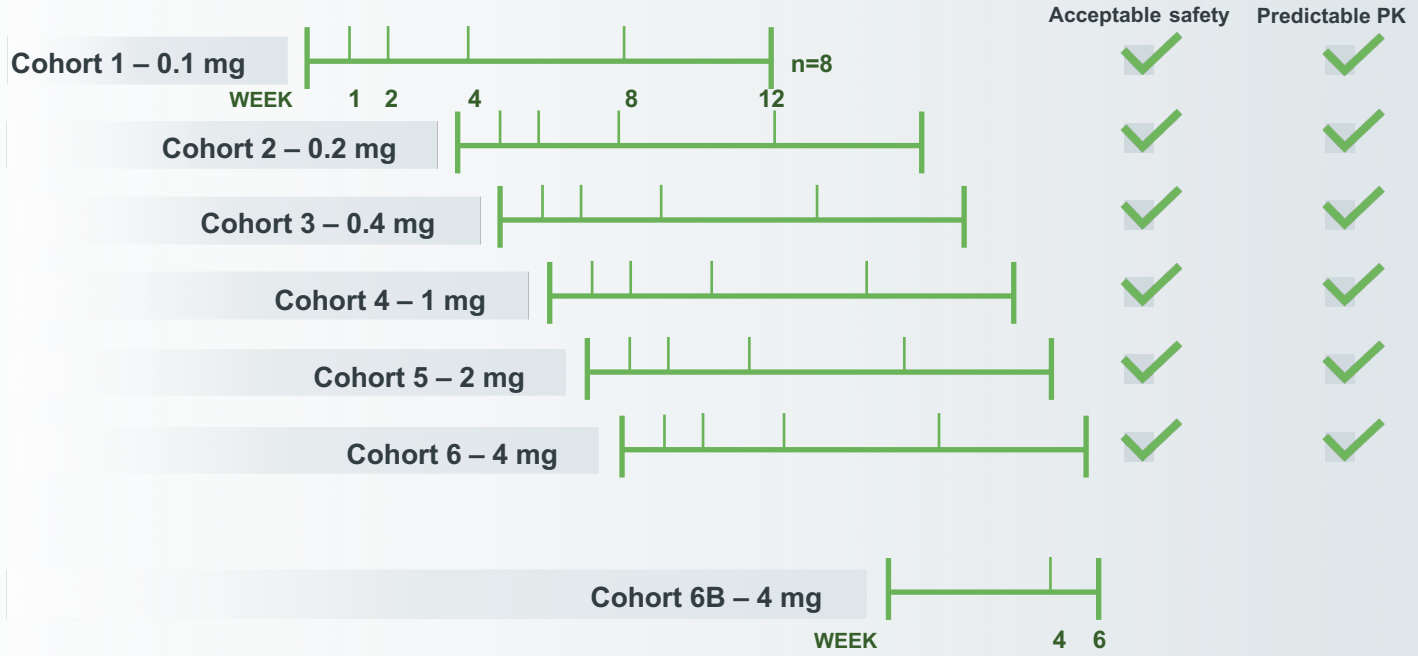
PART A:
Single Ascending Dose

- Patients with OA, KL 1-4 + active synovial inflammation by MRI
- N=48
- Active to Placebo: 3:1

OPTIONS
- Explore higher dose ✓
- Multiple ascending dose
- Expansion cohorts

PART B:
SASP Expansion

- Patients with OA, KL 1-4
- N=24
- Active to Placebo: 2:1



Primary Measure (Part A & B): Safety

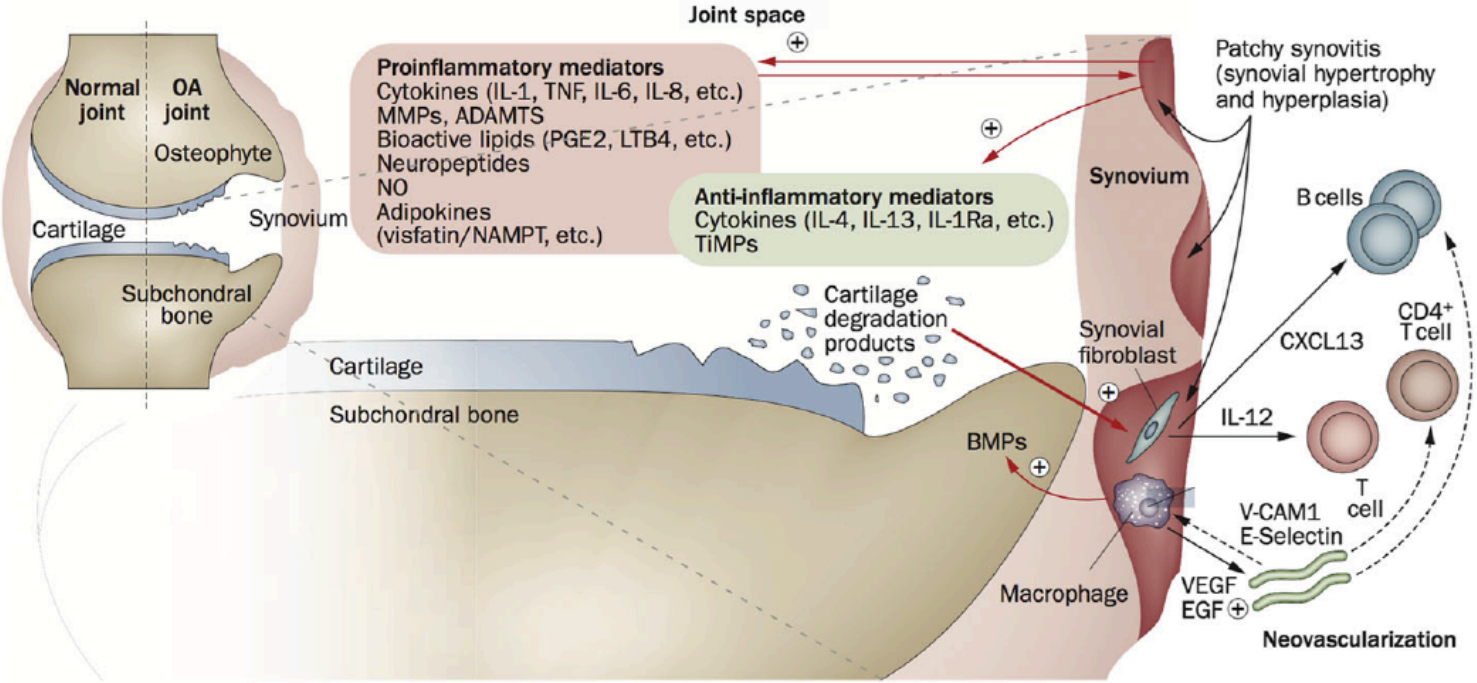
Secondary Measures (Part A): Plasma PK, Semi-quantitative assessment of synovitis by MRI,11-Point NRS pain assessment, WOMAC-A (pain), WOMAC-B (function), & total WOMAC, Synovial fluid SASP factors

Secondary Measures (Part B): Synovial fluid SASP factors, Plasma PK, WOMAC-A (pain)

MEASURING PHASE 1 OA STUDY SUCCESS

MEASURE	"SUCCESS"	IMPLICATION
Safety	**Acceptable safety**	Viable candidate
Tolerability	**Acceptable tolerability**	Viable candidate
Pharmacokinetics	**Predictable pharmacokinetics**	Viable candidate
SASP	**Reduction in factors**	Proof-of-biology
UPSIDE		
Synovitis	**Reduction in synovitis**	Proof-of-concept
Pain	**"Believable" reduction in pain**	Proof-of-concept

OA IS BELIEVED TO BE A MULTIFACTORIAL DISEASE



SASP factors may impact synovial fluid and lining, cartilage and subchondral bone

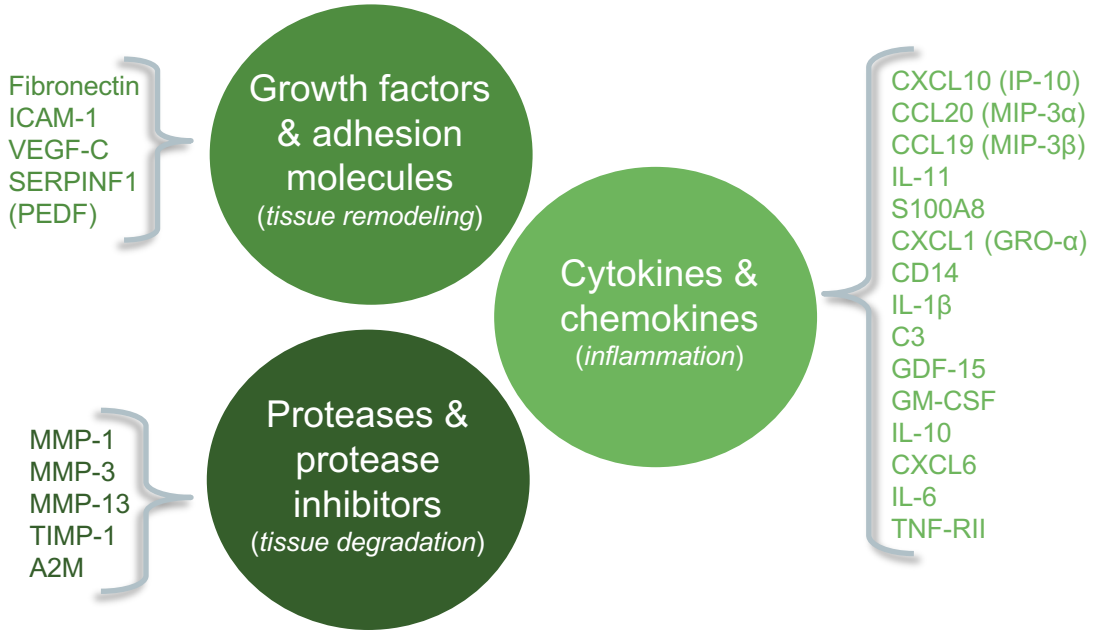
Mathiessen et al., *Arthritis Research and Therapy* 2017

PHASE 1 MEASUREMENT OF POTENTIAL SASP FACTORS

Study will assess up to 24 factors in synovial fluid believed to be relevant to human OA

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**

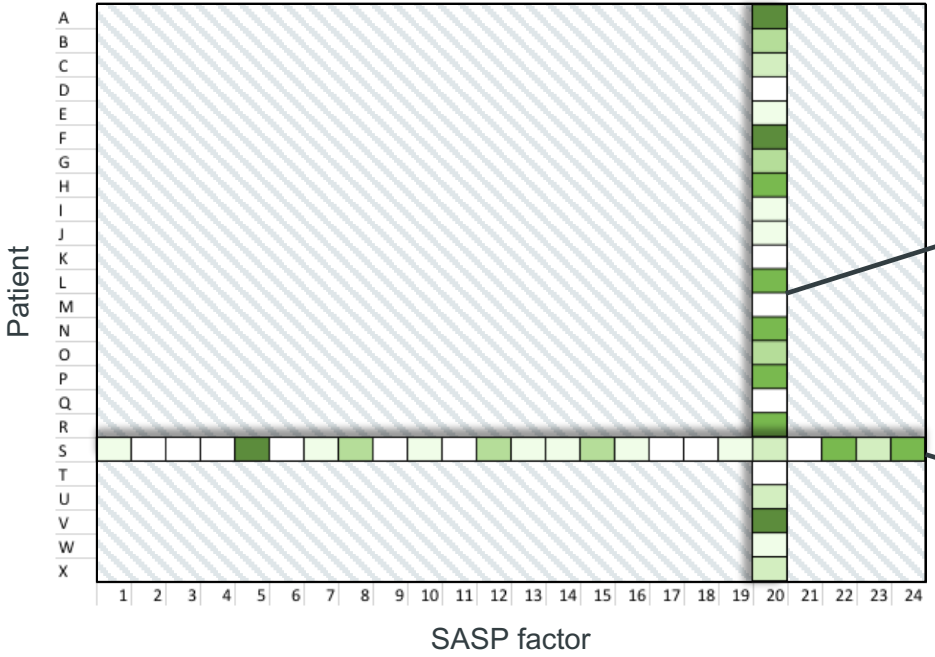


OA is believed to be a heterogeneous and multifactorial disease; Ph 1 will assess multiple implicated SASP factors

PHASE 1 SASP MEASUREMENT

Example SASP trend analysis: baseline vs 4 week post dose

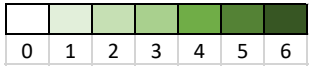
For illustrative purposes only



Does a single SASP factor move in response to treatment across multiple patients?

Do multiple SASP factors move in response to treatment in a single patient?

α= (Week 4 value - Baseline)/ √(Week 4 value + Baseline)



Analysis designed to identify relationship between SASP factor(s) and treatment

PHASE 1 OA SASP MEASUREMENT EXPECTATIONS

1

Heterogeneous: **variability between patients in SASP factor presence/levels is likely**

2

Multifactorial, some SASP factors may play a more dominant role in disease than others: **not all SASP factors are likely to demonstrate the same magnitude of change**

3

Change in a subset of SASP factors has the potential to impact disease pathology, inform OA disease state and senescence biology

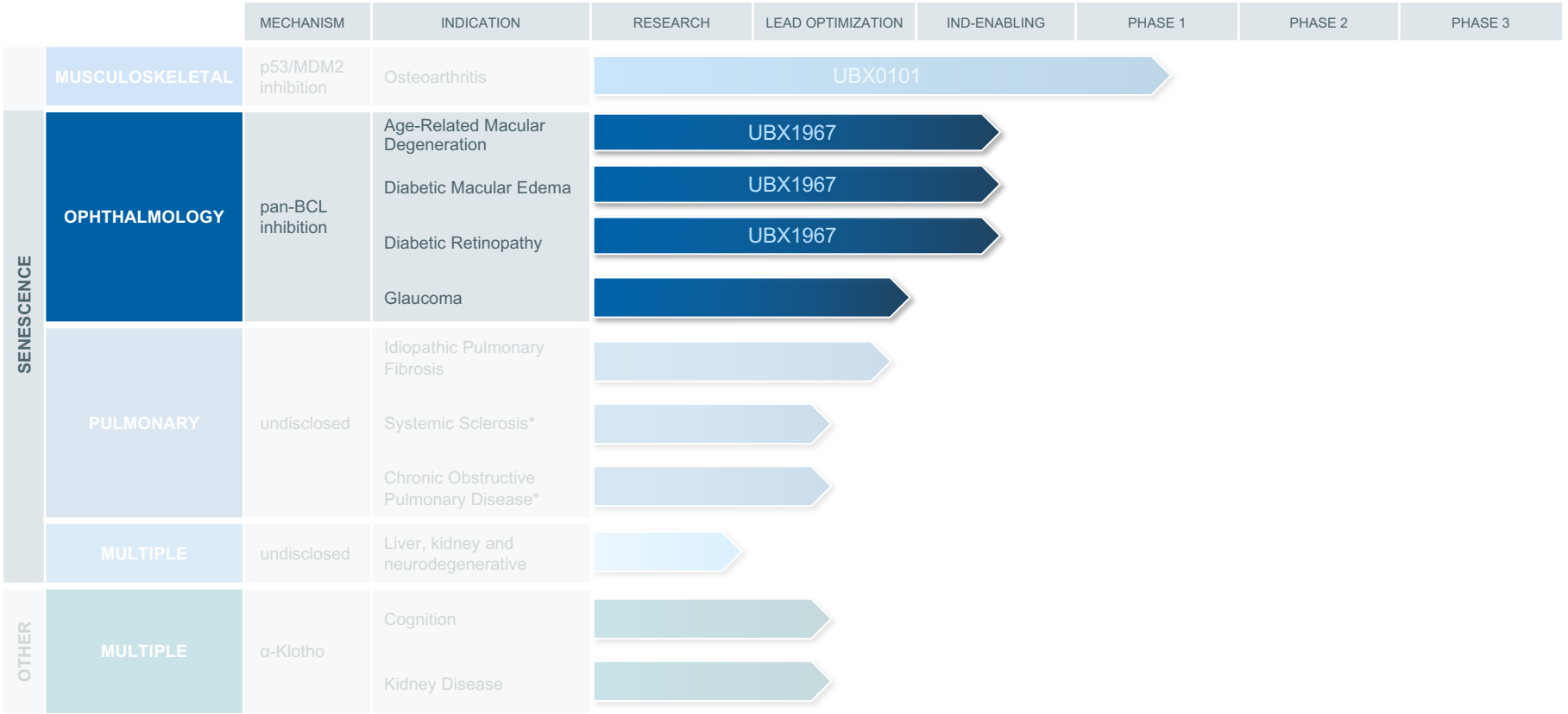
OPTHALMOLOGY

(AGE-RELATED EYE DISEASE INDICATIONS)

UNITY BIOTECHNOLOGY

UNITY PIPELINE

Broad therapeutic potential, addressing multiple mechanisms of aging



*Following IPF Outcome

 As of March 6, 2019

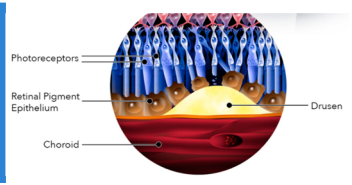
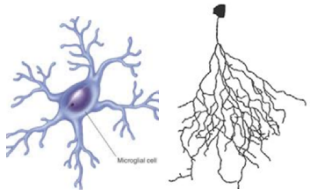
AGE-RELATED EYE DISEASES ARE SIGNIFICANT PUBLIC HEALTH BURDENS

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***		

SENESCENCE AGE-RELATED EYE DISEASE HYPOTHESES

SnCs accumulate in the retina, contributing to disease phenotypes

AMD		DR & DME
SnCs accumulate with with age in the retina retinal pigment epithelium (RPE) layer		**senescent cell**		**SnCs** accumulate with with age & diabetic disease in the retina
SASP → choroidal remodeling & RPE dysfunction → atrophy	SASP FACTORS (VEGF-α, IL-6)	**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)

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AGE-RELATED EYE DISEASES ARE MULTIFACTORIAL

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

AMD

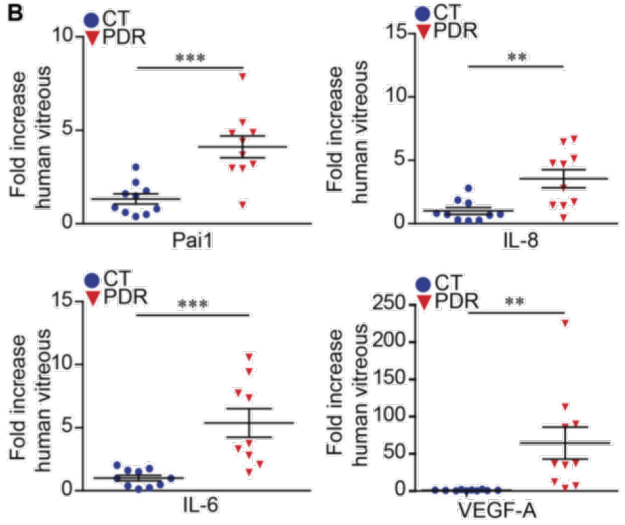
	nAMD Level mean ± SD	Controls Level mean ± SD	*P* value Pre IVA vs Control
IL-1rα	0	0.77 ± 3.19	0.381
IL-6	6.51 ± 5.24	78.2 ± 100	0.0029
IL-7	10.9 ± 3.95	13.5 ± 12.8	0.465
IL-8	6.00 ± 6.69	6.43 ± 6.84	0.448
IL-9	0.087 ± 0.40	0.10 ± 0.41	0.483
IL-12	12.1 ± 5.79	10.4 ± 9.11	0.268
IL-13	1.97 ± 2.22	2.25 ± 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)

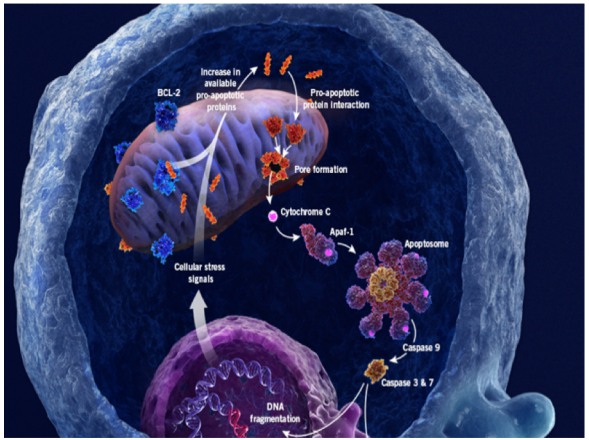
OPPORTUNITY TO HALT AGE-RELATED EYE DISEASES

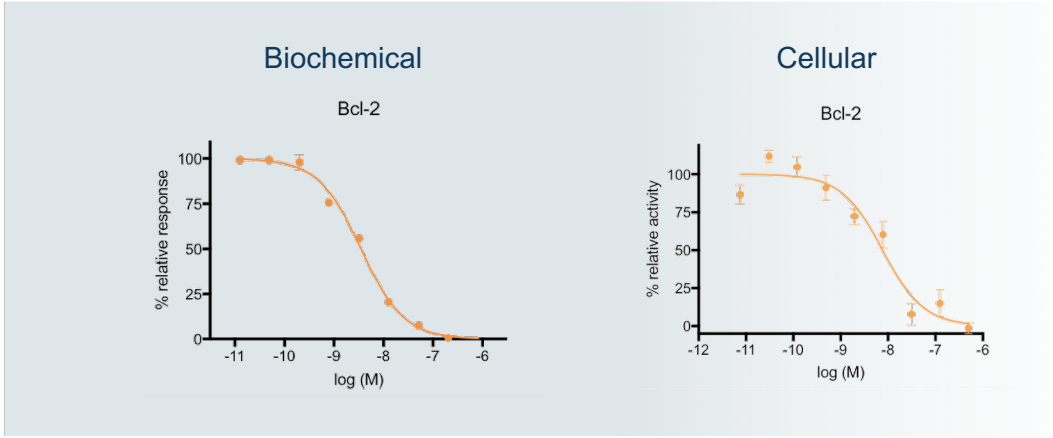
	Current SoC (a-VEGF)	**Promise** (senolytic)
MECHANISM	A-VEGF therapies are designed to remove VEGF, ***only one of numerous SASP factors*** implicated in age-related eye diseases	Utilize novel mechanism to ***remove source of multiple SASP factors*** (root cause of diseases)
Tx IMPACT	Some patients ***do not respond or become refractory*** over time (>20%); long-term use ***can cause complications***	***Improve efficacy*** over a-VEGF therapies, provide options to anti-VEGF non-responders
Tx FREQUENCY	***Require frequent*** (monthly/bi-monthly) ***injections***	***Significantly improve duration of response*** over anti-VEGF therapies, require less frequent dosing
MARKET	***>$8B in global annual sales***	***Increase number of treatable patients*** by improving treatment impact and duration of response

As of March 6, 2019

UBX1967: HIGHLY POTENT PAN-BCL2 SENOLYTIC

Lead development candidate for age-related diseases of the eye



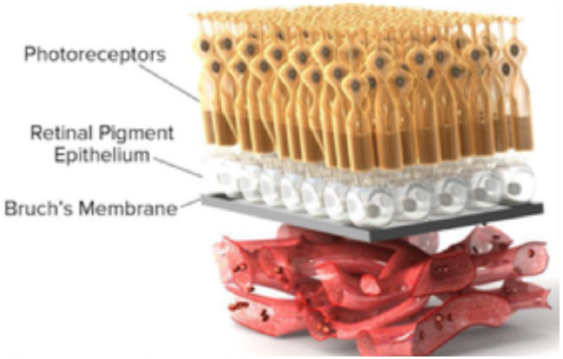


- Potent inhibitor of Bcl-2 anti-apoptotic family members
- Entering IND-enabling studies
- Licensed from Ascentage Pharma

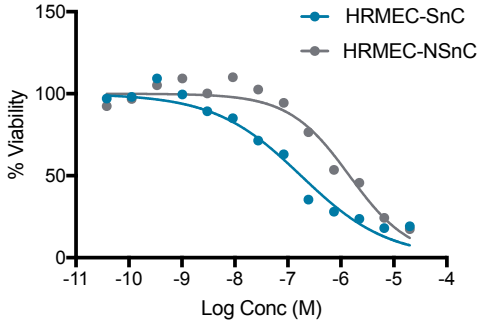
	Biochemical (pK_i)			Cellular TE (pIC_{50})	
	Bcl-xL	**Bcl-2**	**Bcl-W**	**Bcl-xL**	**Bcl-2**
UBX1967	9.6 (± 0.2, n=4)	9.3 (± 0.2, n=4)	9.4 (± 0.2, n=5)	9.6 (± 0.3, n=5)	8.6 (n=2)

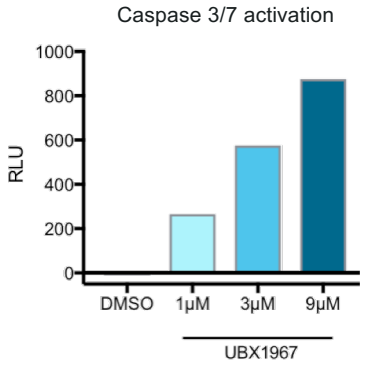
Unpublished UNITY Data

HIGHLY POTENT PAN-BCL2 SELECTIVE SENOLYTIC



Human retinal microvascular endothelial cells (HRMEC)

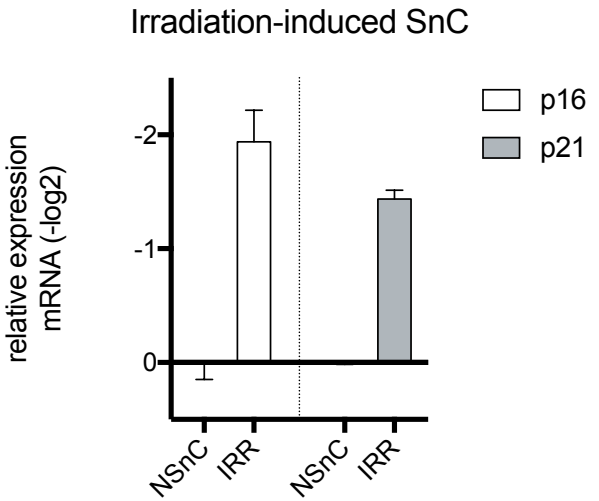


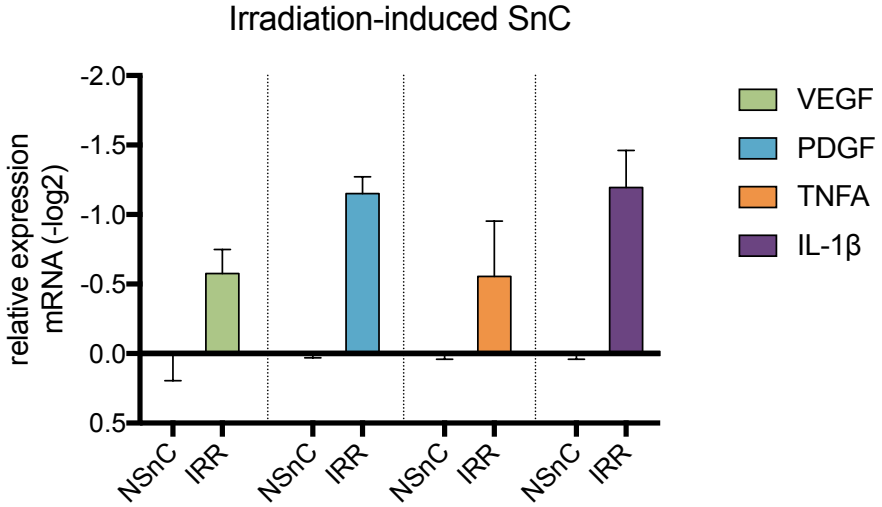


UBX1967 selectively eliminates HRMEC-SnCs over non-SnCs

Unpublished UNITY Data

INDUCTION OF SASP FACTORS IN SENESCENT HRMECS

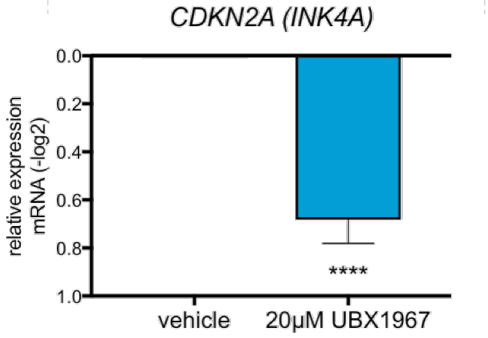




UBX1967 EFFICACY IN MOUSE OIR

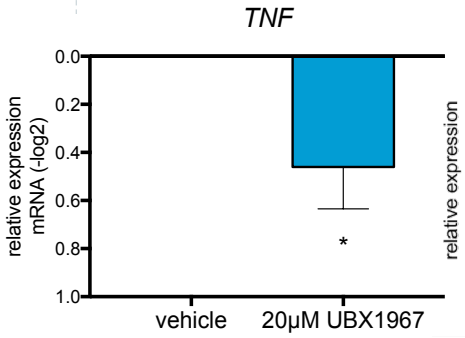
Intravitreal dosing eliminates senescent cells and modulates SASP in mice

Eliminates p16+ SnCs

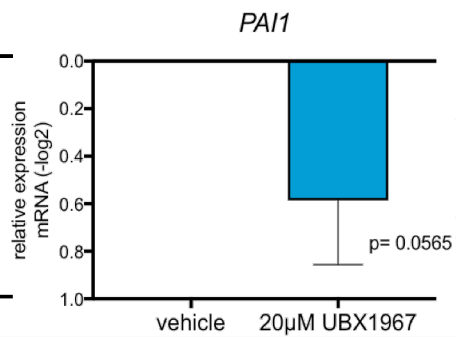


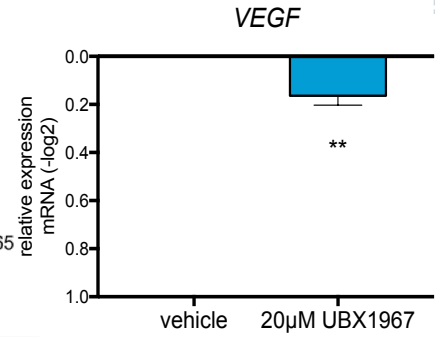
**** p<0.0001 v. vehicle by two-tailed t-test

Reduces SASP Expression (mRNA)



* p<0.05 v. vehicle by two-tailed t-test





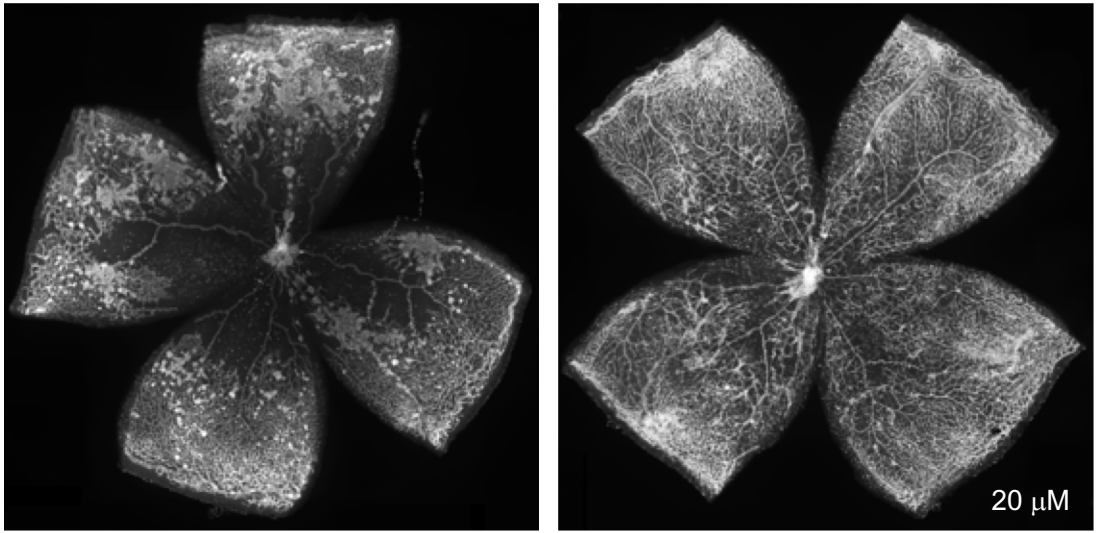
** p<0.01 v. vehicle by two-tailed t-test

Oxygen-induced retinopathy (OIR) mouse model

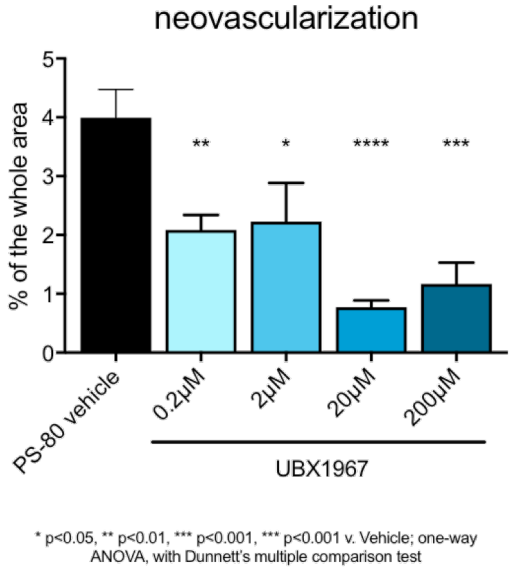
UBX1967 EFFICACY IN MOUSE OIR

Intravitreal dosing improves retinal vasculature

Vehicle	UBX1967



Improves Retinal Vasculature

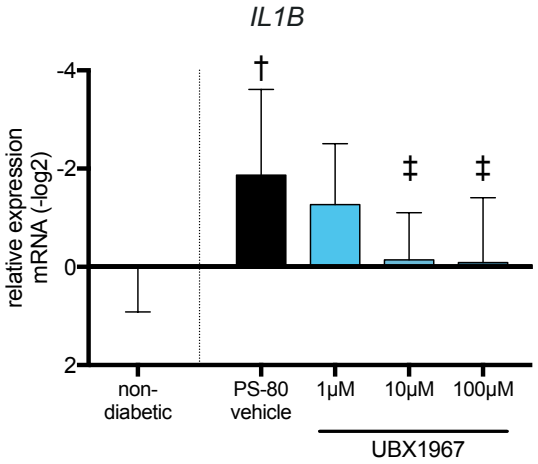


* p<0.05, ** p<0.01, *** p<0.001, *** p<0.001 v. Vehicle; one-way ANOVA, with Dunnett's multiple comparison test

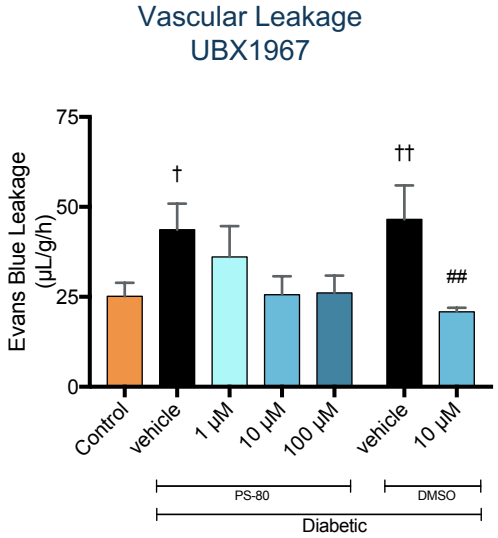
OIR mouse model

UBX1967 EFFICACY *IN VIVO*

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

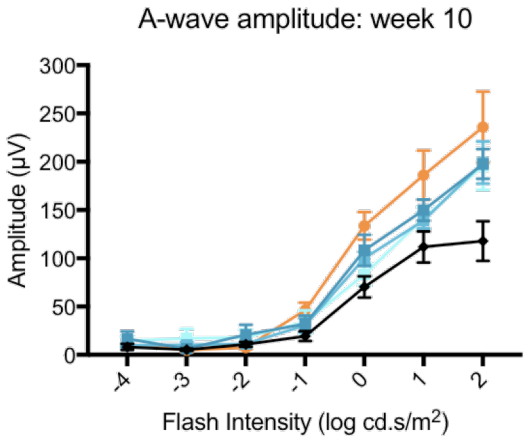


† p<0.05 v. non-diabetic control by two-tailed t-test;
‡ p<0.05 v. Vehicle; one-way ANOVA, with Dunnett's multiple comparison test



† 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

Streptozotocin (STZ) diabetic retinopathy mouse model

1 μM = ~1.7 ng of UBX1967

UBX1967 VALUE PROPOSITION IN MULTIPLE AGE-RELATED EYE DISEASES

DIFFERENTIATING PRECLINICAL FEATURES

1. **Pan-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

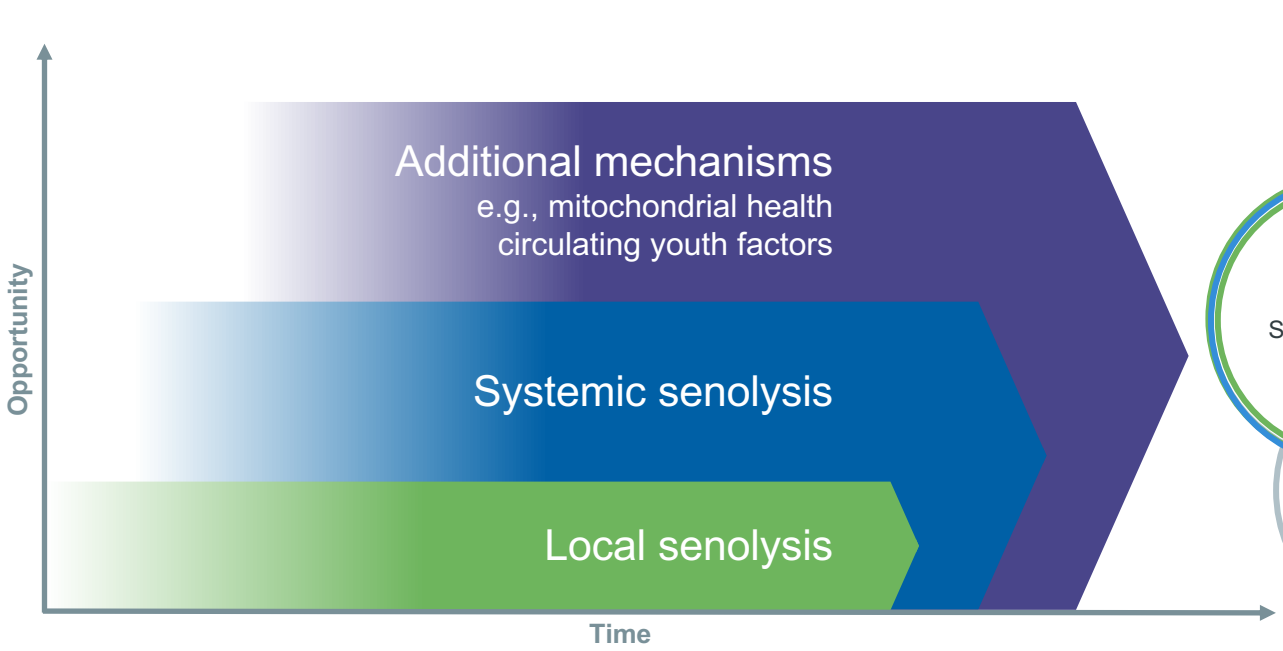
- $171.1 million cash and investments at December 31, 2018

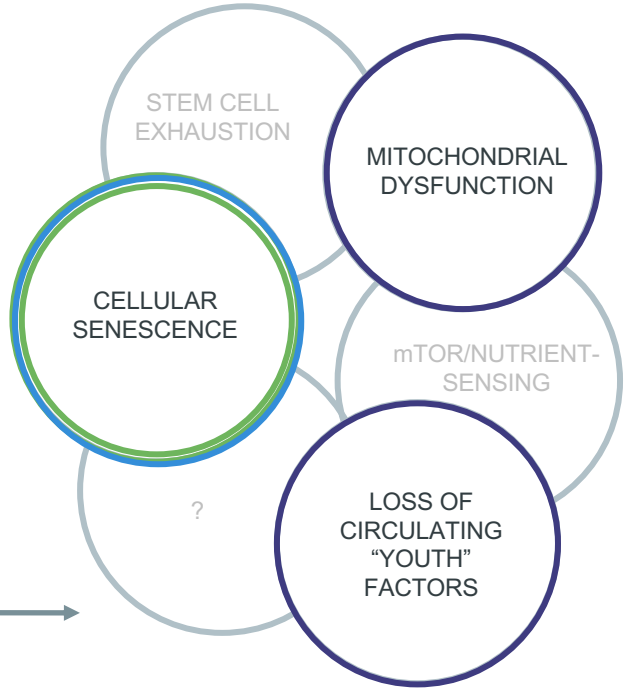
MILESTONES

- ✓ Q2 2018 – Initiation of UBX0101 Phase 1 in patients with OA
- Q2 2019 – Data from UBX0101 Ph1
- Early 2020 – ophthalmology IND filing
  - enables multiple indications, including AMD, DR and DME

BROAD STRATEGY TO EXTEND HEALTHSPAN

Early effort in local senolytic therapy will expand to systemic senolytics and other mechanisms





UNITY plans to address multiple modalities to fully enable potential of age-related therapies

Health·span |helth' span| *noun*

The period of one's life unburdened by the diseases of aging

See also: anti-aging, healthy longevity

HEALTHSPAN

UNITY BIOTECHNOLOGY