Breakthrough Science to Increase Healthy Lifespan

Q3 Investor Presentation
November 13, 2017

OTCQX: CWBR
TSXV: COB.U

www.COHBAR.com

Q3 Financials
Jeff Biunno, CFO

Forward Looking Statements

This presentation includes forward-looking statements (statements which are not historical facts) within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding future plans, intentions, expectations, business prospects and opportunities. Examples of such forward-looking statements include: statements regarding anticipated outcomes and timing of our research and development programs, IND-enabling activities and pre-clinical and clinical trials for our MBTs; expectations regarding the future market for any drug we may develop; statements regarding the anticipated therapeutic properties of our MBTs or the properties and effects of newly-discovered mitochondrial-derived peptides; expectations regarding our ability to effectively protect and expand our intellectual property; statements regarding potential partnership programs and financing activities; and statements regarding the sufficiency and availability of capital to fund our operations. You are cautioned that such statements are not guarantees of future performance and that actual results or developments may differ materially from those set forth in these forward looking statements. Factors that could cause actual results to differ materially from these forward-looking statements include: unanticipated difficulties or unfavorable results encountered in our research and development programs; our ability to raise additional capital when necessary to continue our operations and complete our research and development programs; regulatory risks, including the risk of adverse decisions by regulatory authorities that could affect the viability or commercial potential of our drug candidates; our ability to recruit and retain key scientific personnel; competitive risks, and our ability to establish and maintain partnerships with research and industry partners. Additional assumptions, risks and uncertainties are described in detail in our registration statements, reports and other filings with the Securities and Exchange Commission and applicable Canadian securities regulators, including the “Risk Factors” set forth in our Annual Report on Form 10-K, as supplemented by our quarterly reports on Form 10-Q. The forward-looking statements and other information contained in this presentation are made as of the date hereof and CohBar, Inc. does not undertake any obligation to update publicly or revise any forward-looking statements or information, whether as a result of new information, future events or otherwise, unless so required by applicable securities laws.

CohBar INC.

Science and Business
Simon Allen, CEO

Speakers

Highly experienced and successful scientists and biotech executives

Simon Allen B.Sc., MBA
Chief Executive Officer

- **20 years of biotech business development experience**
  - Senior positions at Ambrx, Kalypsys, CovX, Nuvelo, SkyePharma
  - More than 20 partnerships, licenses, divestments and M&A transactions
  - Generated significant non-dilutive funding and investor returns
- **Capital Markets**
  - Burdett Buckeridge & Young
  - Robertson Stephens & Co.
- **Lab**
  - Gilead, Burns Philp
  - Antivirals and genetics

Kenneth Cundy, PhD.
Chief Scientific Officer

- **29 years of drug discovery and development experience**
  - Senior scientific positions at Xenoport, Gilead Sciences, Sterling
  - Development of 15 drugs (including Hepsera®, Tamiflu®, Viread® and Horizant®) with $100B+ in sales
  - Co-invented the antiviral tenofovir (marketed in Viread®, Atripla®, Complera® Stribild® and Truvada®) and nanocrystal technology
  - Filed **15 INDs** and **6 NDAs** with the FDA
- **IP and Scientific Publications**
  - 150+ US and International Patents
  - 70+ papers

Our Vision addresses enormous medical needs

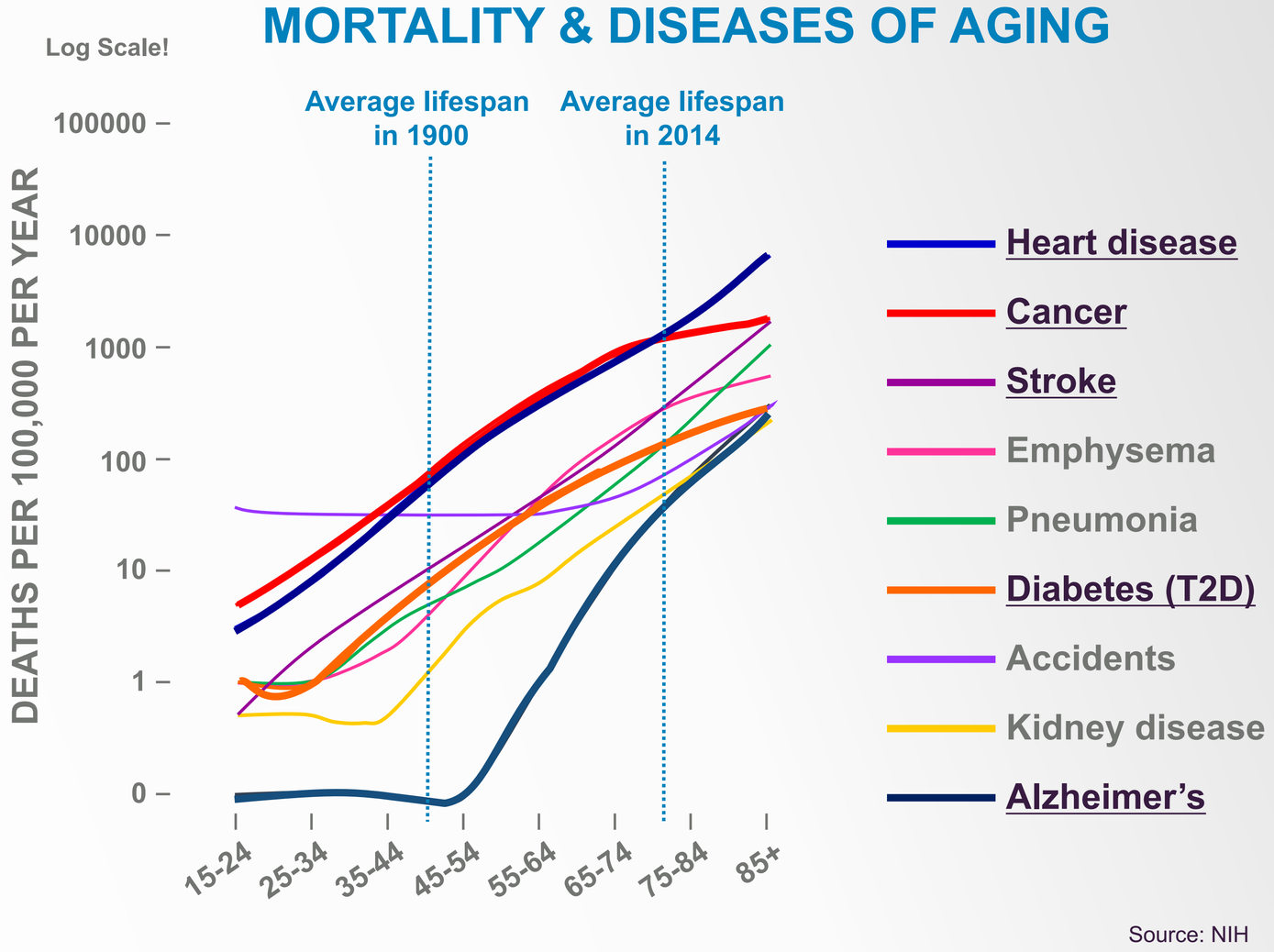
CohBar's vision is to increase healthy lifespan by developing effective treatments for the underlying metabolic dysfunction driving the diseases of aging and age-related mortality, such as obesity, fatty liver disease, NASH, cancer, Type 2 Diabetes, and cardiovascular and neurodegenerative diseases.

Collectively, these diseases are the primary cause of the global epidemic of non-communicable diseases of aging, which is already responsible for 70% of the global death rate and 80% of US Medicare costs, and which is projected to cost the global economy over $30T by 2030.

Sources: World Health Organization, CMS

Diseases associated with metabolic dysfunction are leading causes of death

- Onset and incidence of these diseases significantly precedes mortality from them
- Metabolic dysfunction and associated diseases may exist for decades, i.e. growing obesity and T2D onset among youth
- Nearly 92% of older adults have at least one chronic condition, and 77% have at least two comorbidities (Focus of TAME FDA study)
- Other epidemic metabolic diseases such as obesity, fatty liver disease (as well as Type 2 diabetes) are significantly associated with the leading causes and with each other
- On average, ages 65 to 69 years old take nearly 14 prescriptions, ages 80 to 84 take 18 prescriptions, per year
- Centenarians have minimal illness (or medical expense) until their final few years of life (chronological vs. biological age)

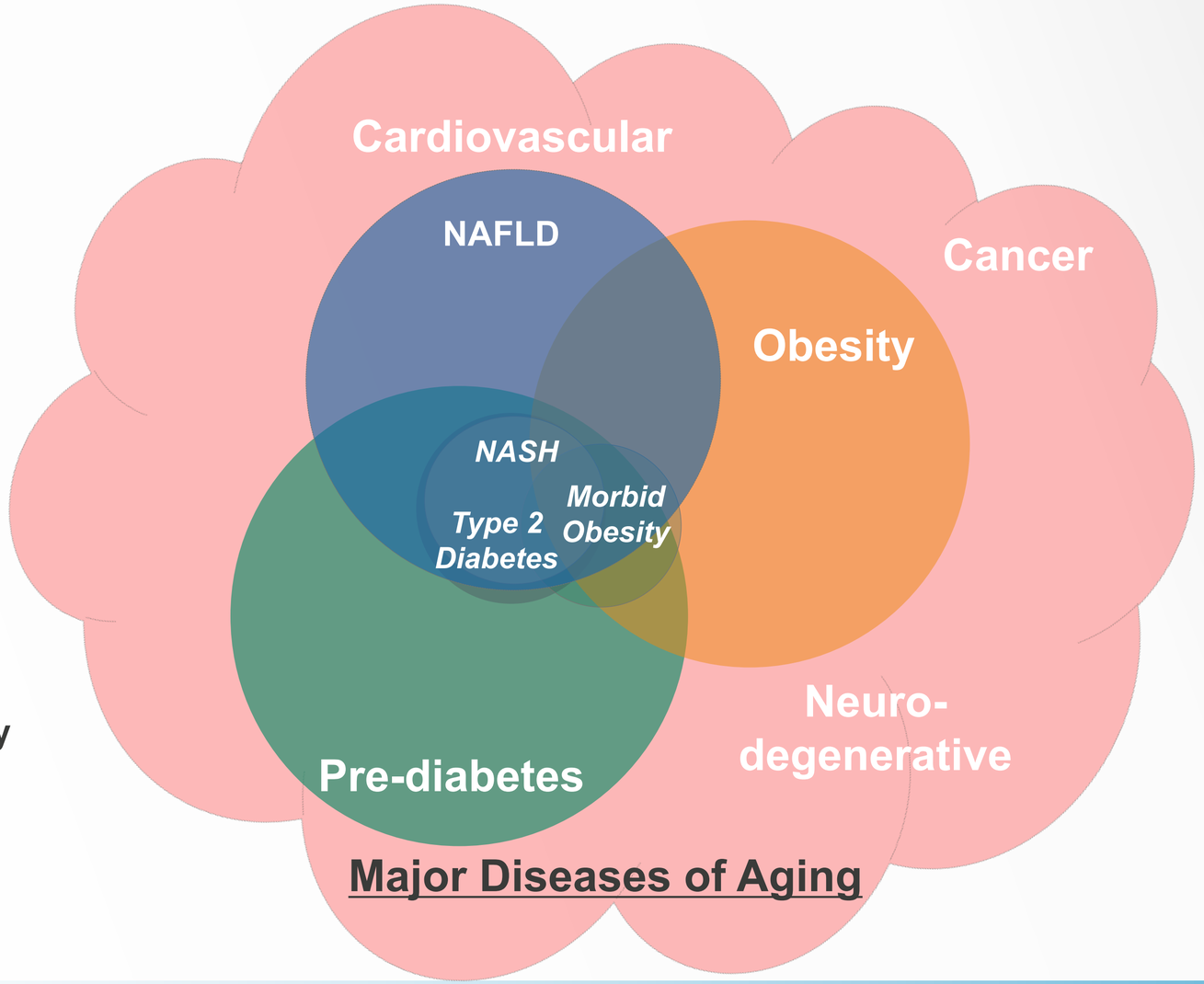


Source: NIH

Comorbid diseases of aging with underlying metabolic dysfunction

- **At least 68 percent of people age 65 or older with diabetes die from some form of heart disease; and 16% die of stroke.**
- **Adults with diabetes are two to four times more likely to die from heart disease than adults without diabetes.** ***(AHA)***
- **People with Type 2 diabetes and NAFLD have 2x the "all-cause" mortality rate.**

  (Diabetes UK Conf.)



- **About 20% of all cancers are caused by excess weight.**

  (J. Obesity)
- **"strong correlation between obesity and the development of Alzheimer's disease and Parkinson's disease"**

  (Life Sciences, 2017)
- **"obesity is one of the most important independent risk factors for cardiovascular disease"**

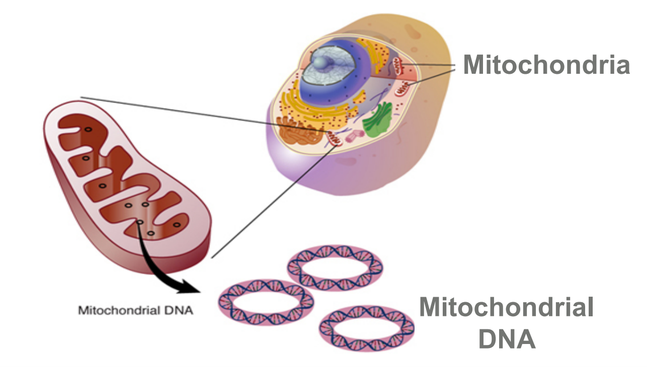
  (NIH)

Our Founders' Breakthrough Research into Mitochondria, a Core Component of Metabolism

Mitochondria bioenergetics produce 90% of cellular energy from nutrients.[1]

- Have their own maternally inherited DNA, 200,000x smaller than the nuclear genome
- Historically believed to have 37 genes
- 1000's of mitochondria per cell, 20% of cell volume

Mitochondrial function **(critical to metabolism) declines with age and metabolic dysfunction**



1: Douglas C. Wallace, A mitochondrial bioenergetic etiology of disease

Our Founders' discoveries, the result of 15+ years of research by them and their collaborators funded by $30M+ in research grants, are the foundation of our technology platform:

- Discovered the existence of hundreds of new potential genes in the mitochondrial genome that encode peptides (small proteins)
- Identified the first mitochondrial derived peptide ("**MDP**") linked to age related diseases (1998) and human centenarians and offspring
- Demonstrated the metabolic regulation and protection potential of additional MDPs with cell and animal models across multiple age-related disease

Our Founders' research and discoveries identified MDPs as a set of previously unrecognized metabolic mechanisms for regulation and protection, and a potential source of therapeutics for metabolic dysfunction and associated diseases of aging.

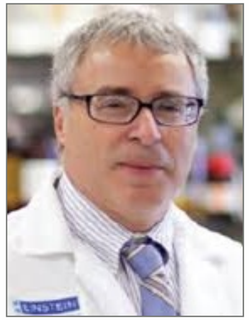
Our Founders

World-recognized scientific and medical experts in genetics, metabolism and aging, and veterans in drug research and development

Pinchas Cohen, M.D.

- Dean of the USC Davis School of Gerontology
- EUREKA-Award and NIH-Director-Transformative RO1-Grant recipient for work in the field of mitochondrial peptides

Nir Barzilai, M.D.



- Professor of Genetics and Director of the Institute for Aging at the Albert Einstein College of Medicine (25 years)
- Leading the TAME Study with FDA
- Holds NIH Merit award focusing on the metabolic decline of aging

John Amatruda, M.D.

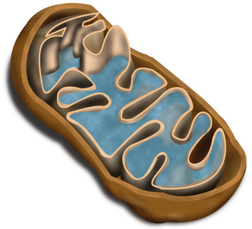
- More than 35 years experience at pharma and academia including Merck, Bayer and the NIH
- Former SVP and Franchise Head for Diabetes and Obesity at Merck Research Labs
  - Led development of Merck's Januvia, a $6B/year diabetes drug

David Sinclair, PhD.

- Professor of Genetics at Harvard Medical School
- Founder of Sirtris Pharmaceuticals and OvaScience
- On TIME's 2014 list of 100 most influential people in the world

CohBar's Execution Strategy: Identify and Optimize MDPs, Develop into MBTs

Mitochondria (Powerhouse of the Cell)

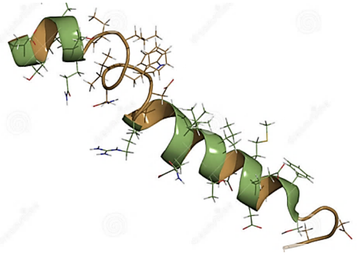


1

Mitochondria Derived Peptide ("MDP")

2

Optimized Peptide ("Analog")



3

Mitochondria Based Therapeutic ("MBT")

Identify

Identify/characterize peptides with biological activity encoded within mitochondria

File Intellectual Property ("Own the Space")

Quantify therapeutic potential across diseases

Optimize

Optimize drug like properties

- Proprietary assays
- Disease models

Match analogs with greatest therapeutic potential to medical needs and market opportunities

Develop and Partner

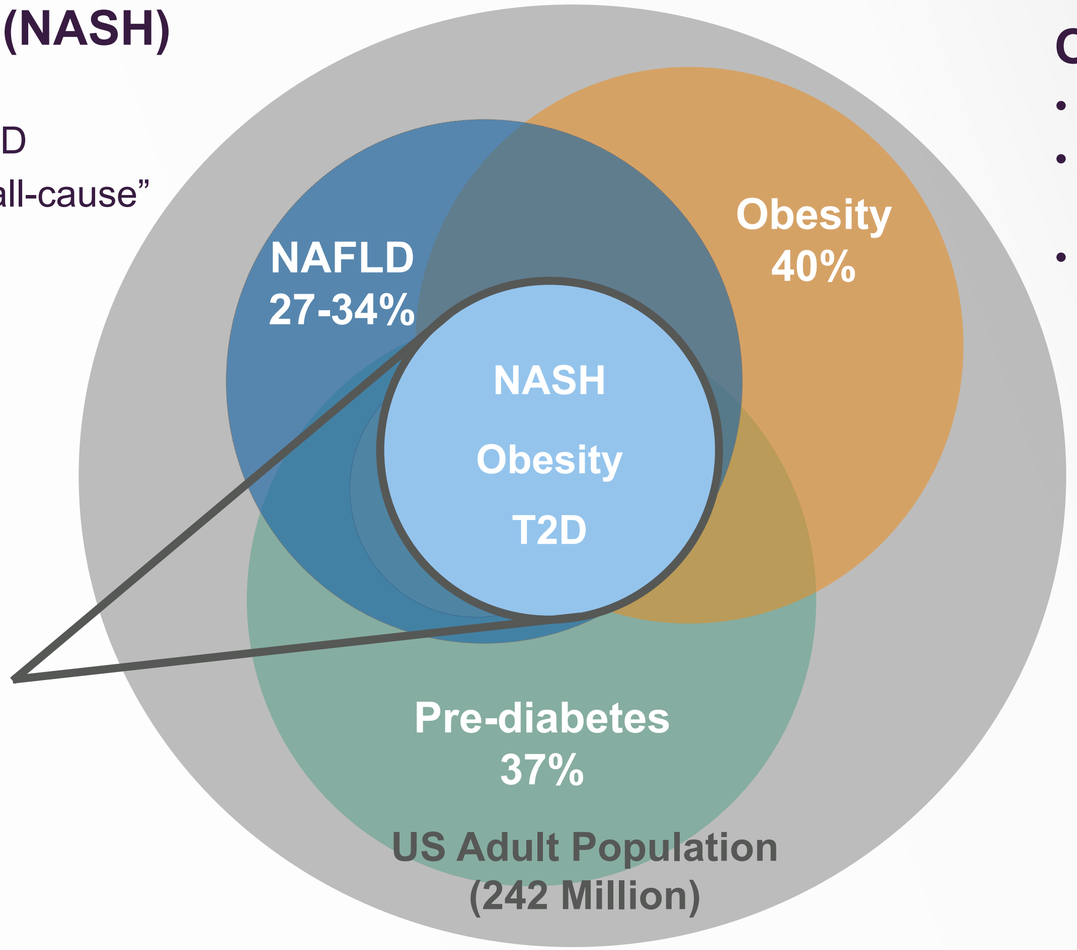
Prioritize for internal clinical development and partnership opportunities

Advance lead therapeutic candidates to the clinic

CohBar's Core Disease Focus – Epidemic Metabolic Diseases

Nonalcoholic Steatohepatitis (NASH)

- Up to 12% of US adults have NASH
- 70-90% are morbidly obese or have T2D
- People with T2D/NAFLD have 2x the "all-cause" mortality rate



Obesity

- 40% of US Adults are obese
- Associated with T2D, coronary heart disease, stroke, hypertension and cancer
- 91% of obese may have NAFLD

Core Disease Focus

Addressable patient populations

NASH: 12M-29M

Obesity: 80M

T2D: 21M

Type 2 Diabetes

- 9% of US adults have Type 2 Diabetes (T2D)
- Obesity and age are major risk factors

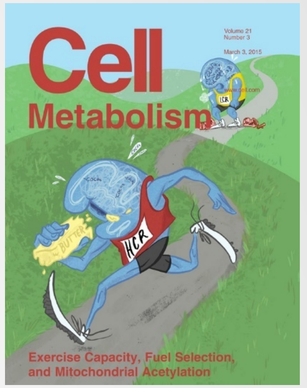
Source: Company data and other sources including CDC, NIH, WHO, World Journal of Gastroenterology, American Diabetes Association, Diabetes Management, Journal of Clinical Endocrinology and Metabolism, American Society for Metabolic and Bariatric Surgery

Lead Program

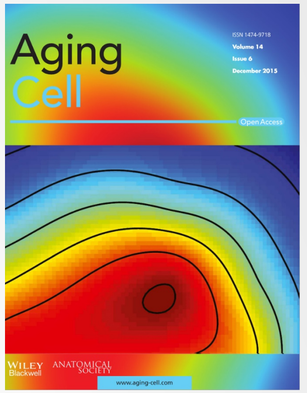
Lead MBT Program (CB4209/CB4211)

Novel Peptide Analogs of the Natural Peptide MOTS-c

- Based on MOTS-c, an MDP discovered by our founders
- MOTS-c is encoded in mitochondrial genome and secreted
- MOTS-c showed potential for regulation of metabolic processes
- Structure optimized by CohBar to achieve novel, improved analogs
- Identified CB4209 and CB4211 as clinical candidates
- Demonstrated activity in preclinical models of NASH and Obesity
- Comprehensive intellectual property coverage filed
- **Would be the first mitochondria based therapeutic to be advanced to clinical testing**



Cell Metabolism, March 2015

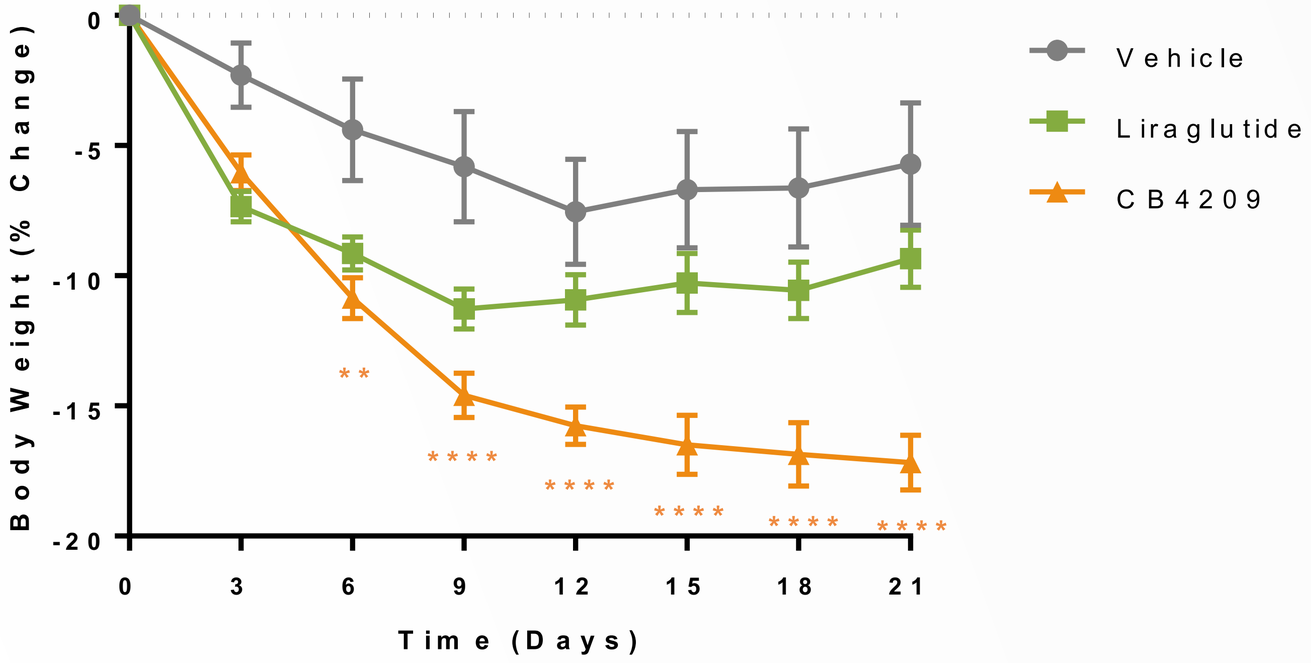


Aging Cell, Aug 2015

CB4209/11 Reduce Body Weight in Obese Mice

Sustained Weight Reduction
(Diet Induced Obesity Model)

3x Greater Overall Weight Loss*

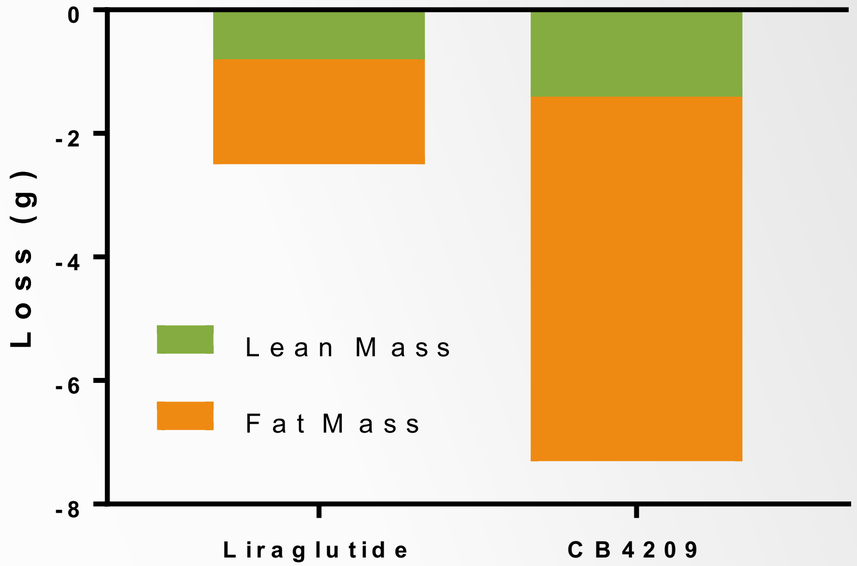


Liraglutide = -3.6% CB4209 = -11.5%

* Corrected for Vehicle

Selective Reduction of Fat Mass vs Lean Mass

2x Greater Selectivity



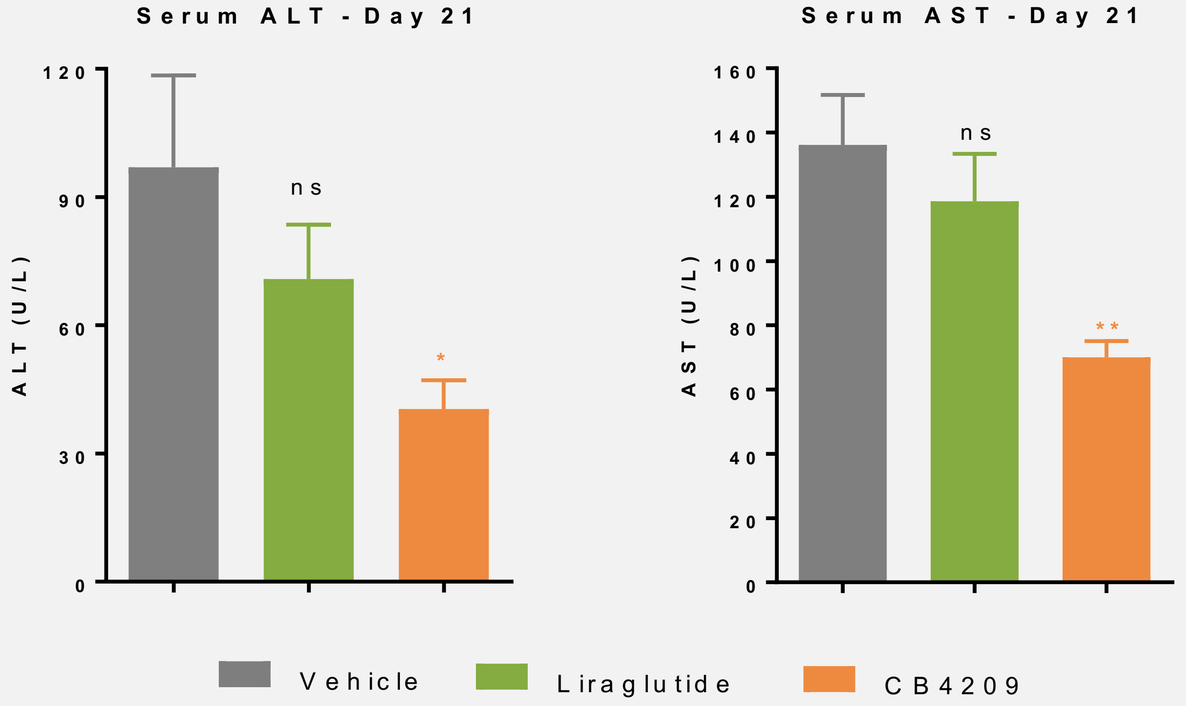
Fat:Lean Liraglutide = 2:1 CB4209 = 4:1

(Similar Data Observed with CB4211)

CB4209/11 Reduce Liver Enzymes and Triglycerides

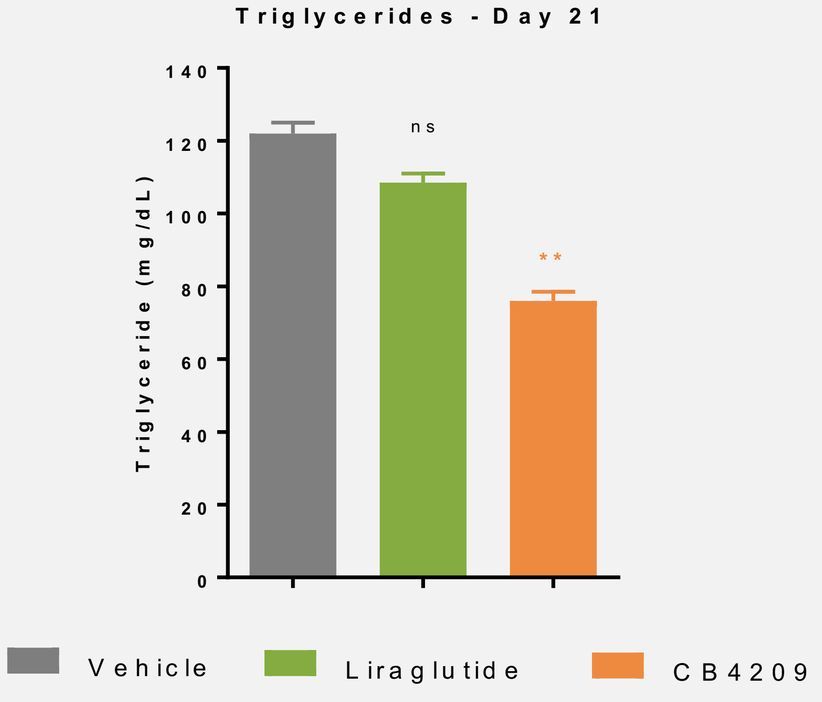
Liver Enzymes
(Serum Markers of Liver Damage)

~ 50% Reduction



Triglycerides
(Serum Marker of Fatty Liver)

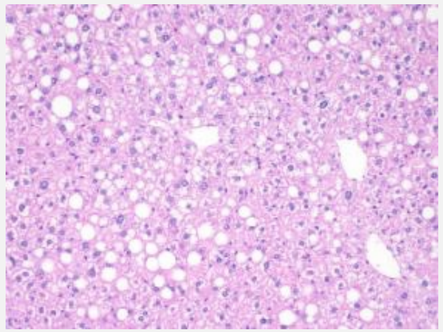
~ 30% Reduction



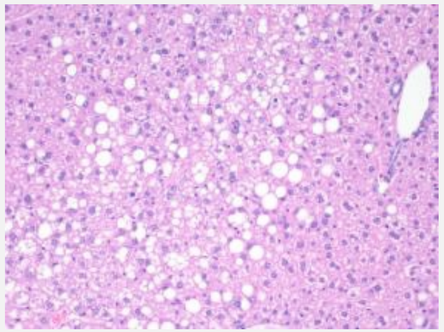
(Similar Data Observed with CB4211)

CB4209/11 Reduce Liver Fat (Steatosis)

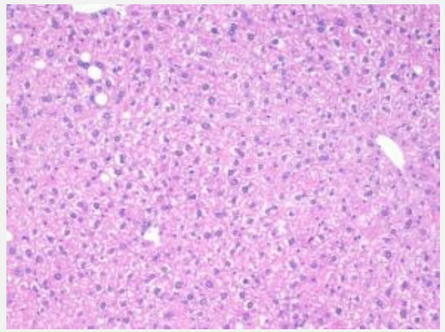
Decreases Liver Fat
(40x Magnification from DIO mice)



Vehicle Control



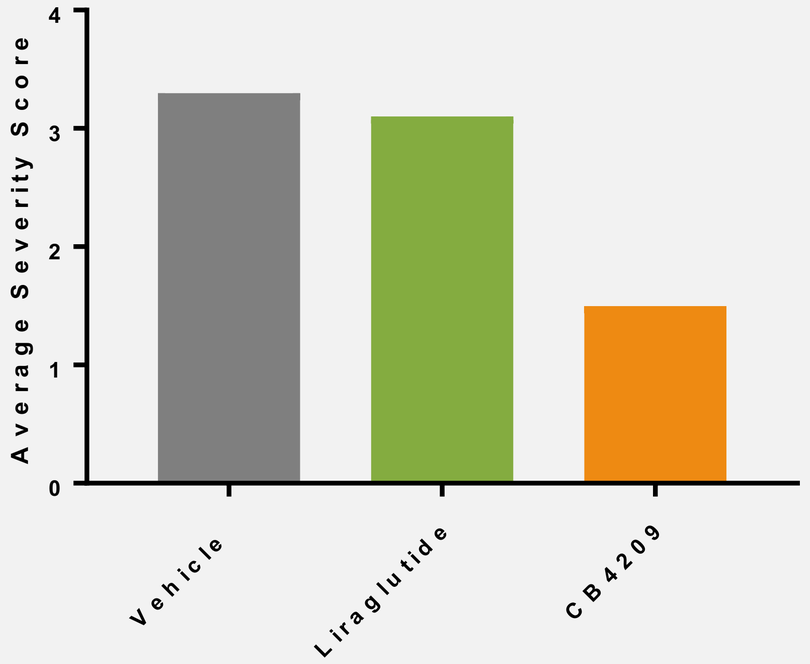
Liraglutide



CB4209

Fewer Fat Deposits

Reduces Severity of Fat Deposits
(Scale of 1-4)



1 = minimal, 2 = mild, 3 = marked, 4 = severe

(Similar Data Observed with CB4211)

CB4209/11 in Combination with GLP-1 (DIO Model)

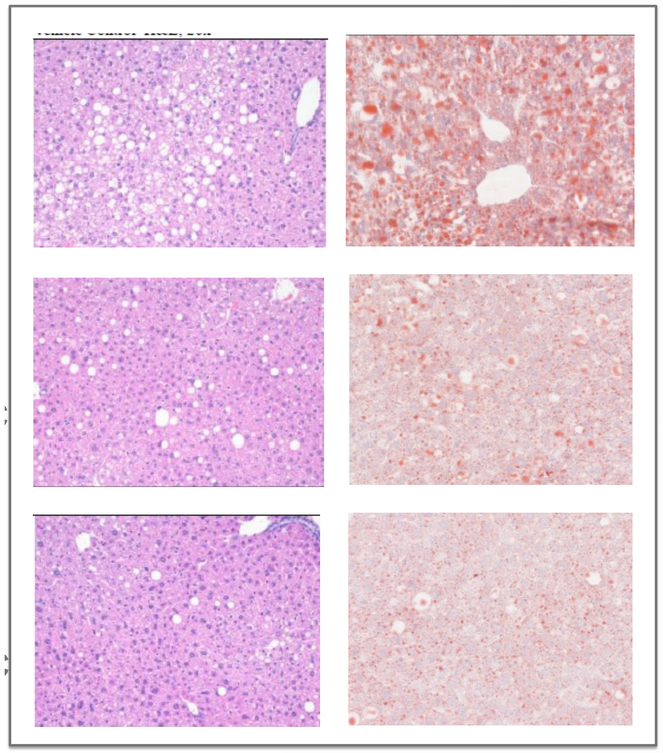
Decreased Fat in Liver at Day 21
(DIO Mouse Model)

Fat Staining Method:
H&E (white) Oil Red O (red)

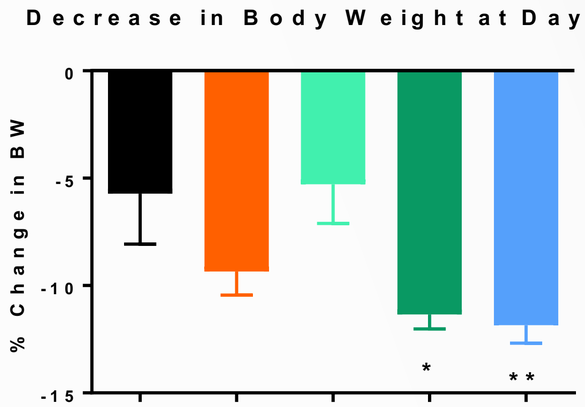
Liraglutide

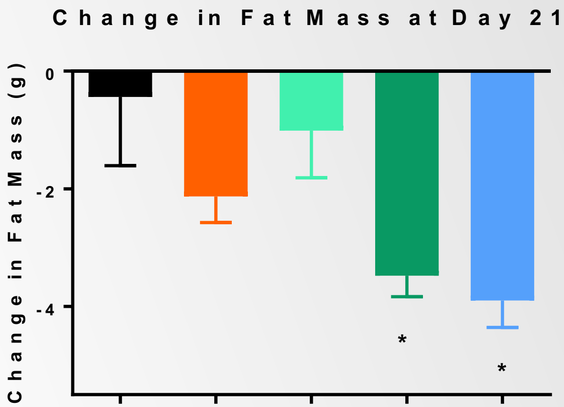
Liraglutide + CB4209 5 mg/kg QD SC

Liraglutide + CB4211 5 mg/kg QD SC



Improved Body Weight/Fat Mass Reduction
(DIO Mouse Model)

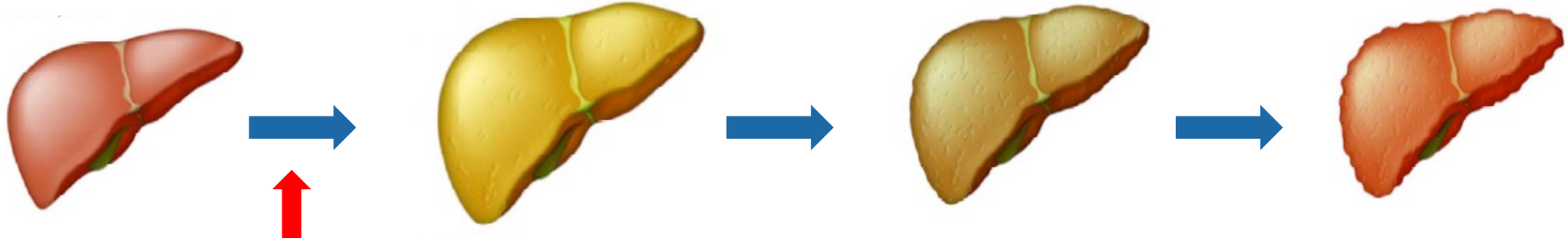




Vehicle BID SC
Liraglutide QD IP
CB4209 5 QD SC
CB4209 5 QD SC + Lirag
CB4211 5 QD SC + Lirag

QD: Once daily; SC - subcutaneous

Non-alcoholic Steatohepatitis (NASH)



Healthy Liver

NAFLD
Fat Accumulation
(Steatosis)

NASH
Steatosis, Cellular
Injury and
Inflammation

Fibrosis/Cirrhosis

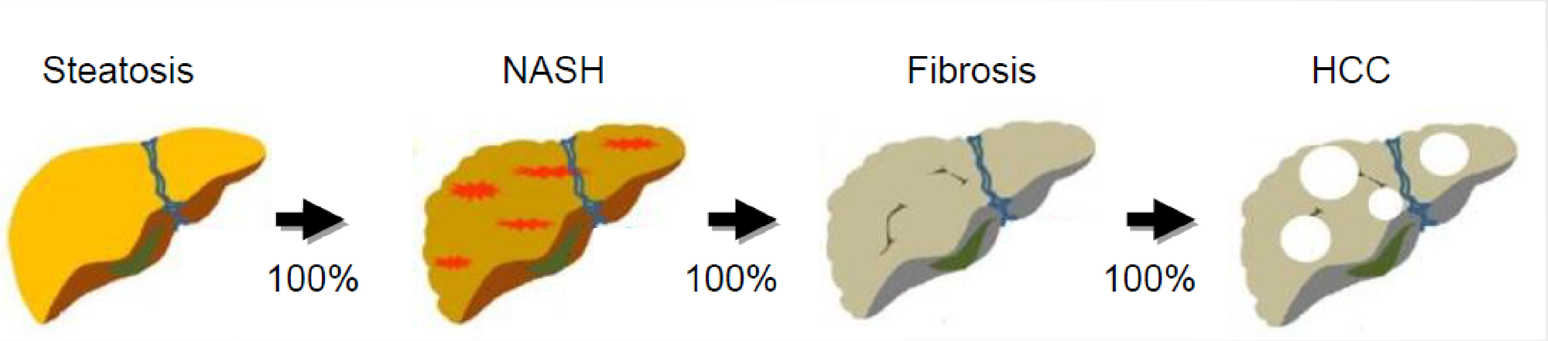
Obesity/Type 2 Diabetes
Metabolic Dysfunction
Overnutrition
Sedentary Lifestyle

Liver Failure
Cancer (HCC)
Transplant
Death

The NAFLD Activity Score (NAS)

- Scale used for clinical diagnosis of NASH in patients following liver biopsy
- Quantitative measure of the progression of NASH used in clinical studies
- Histological scoring system developed at NIH
- Provides a single numerical scale that correlates well with severity of NAFLD/NASH
- Composite of three different components:
  - Fat deposits in the liver (steatosis)
  - Cellular damage to the liver (hepatocyte ballooning)
  - Inflammation of the liver
- Same scale is used in preclinical models of NASH (e.g., STAM® mice)

The STAM® NASH Model Developed by SMC (Stelic)



- STAM mice are initially exposed to a chemical insult followed by high fat diet beginning at 4 weeks of age
- All animals develop symptoms of NASH by 6 weeks of age, chronic fibrosis by about 12 weeks of age, and hepatocellular carcinoma (HCC) by 16 weeks of age
- Effects on NASH can be evaluated rapidly with 3 weeks of treatment (NAS Score)
- Longer studies required to assess other effects (fibrosis and cancer)

The STAM Mouse Model of NASH is Widely Used

The STAM Mouse Model is a Well-established Preclinical Model of NASH

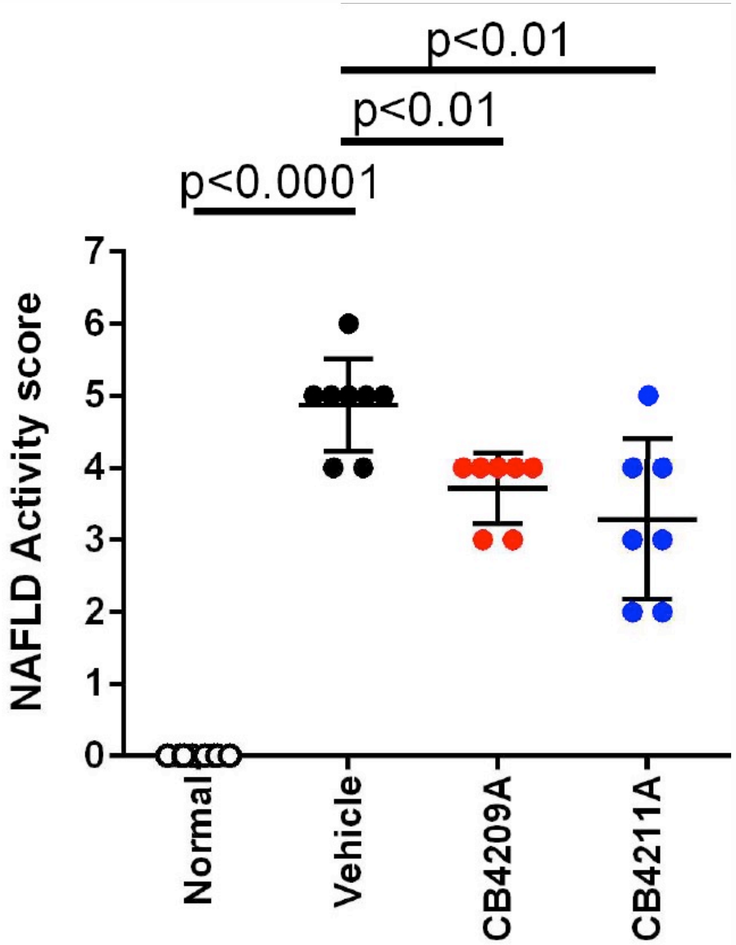
- Mice at 6 weeks of age meet same baseline diagnostic criteria for NASH used in humans
- Model has been used for preclinical assessment of wide range of potential NASH drugs*
- Effects on NAS score can be assessed after just 3 weeks of treatment (9 weeks of age):

  Intercept's Obeticholic acid (FXR agonist) reduced NAS by 23%*

  Tobira/Allergan's Cenicriviroc® (CCR2/CCR5 inhibitor) reduced NAS by 25 to 30%*

*Sources: SMClab.co.jp; Kremoser et al. NASH 2010 Conference; Lefebre et al., PLoS ONE11(6): e0158156.

CB4209/11 Reduce NAS Score in STAM Model of NASH



CB4209 and CB4211 Significantly Reduced NAFLD Activity Score (NAS)

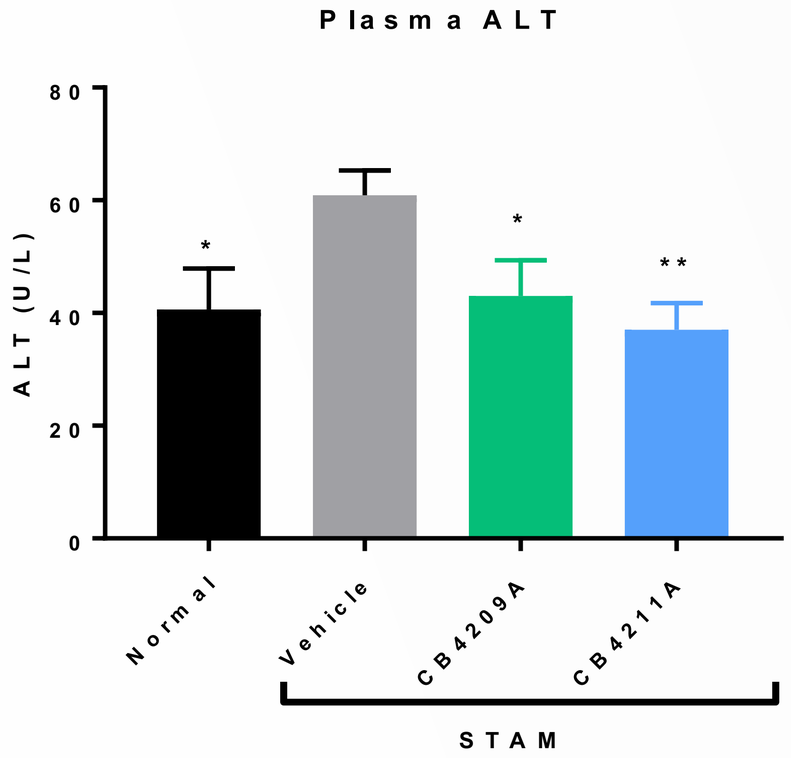
- NAS is a composite score of liver fat (steatosis), liver cell damage (hepatocyte ballooning) and inflammation.
- Treatment with CB4209 or CB4211 led to significant reduction in NAS at 9 weeks of age:

  CB4209: 24% reduction in NAS

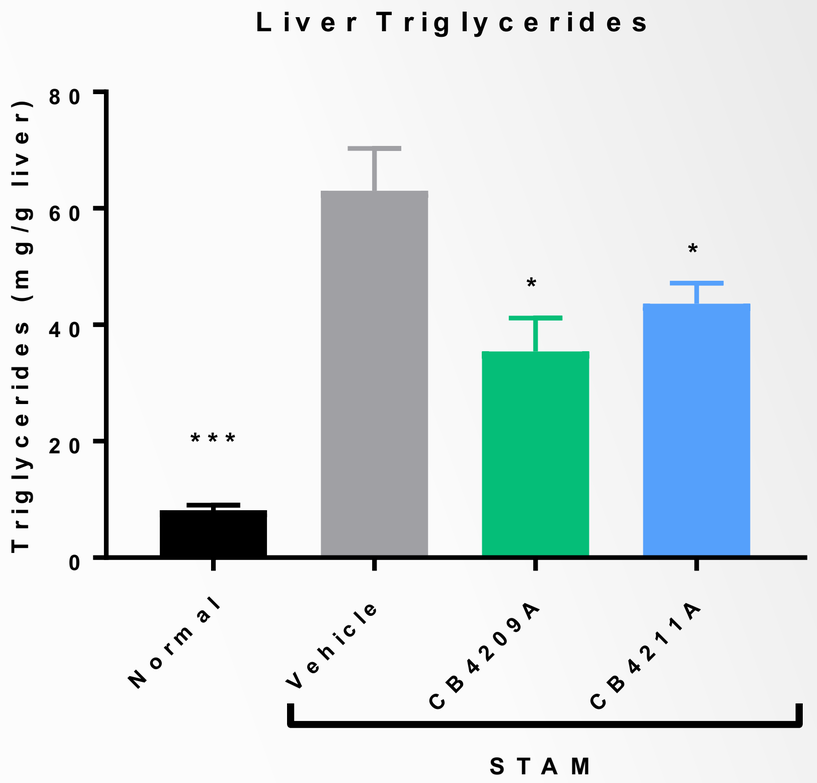
  CB4211: 33% reduction in NAS
- Both peptides significantly reduced plasma ALT (marker of liver damage) and liver triglyceride (fat) levels

CB4209/11 Improve Biomarkers in STAM NASH Model

Significant Reduction in Liver Damage
(STAM NASH Model)



Significant Reduction in Liver Fat
(STAM NASH Model)



P values: * <0.05; ** <0.01, ***<0.001

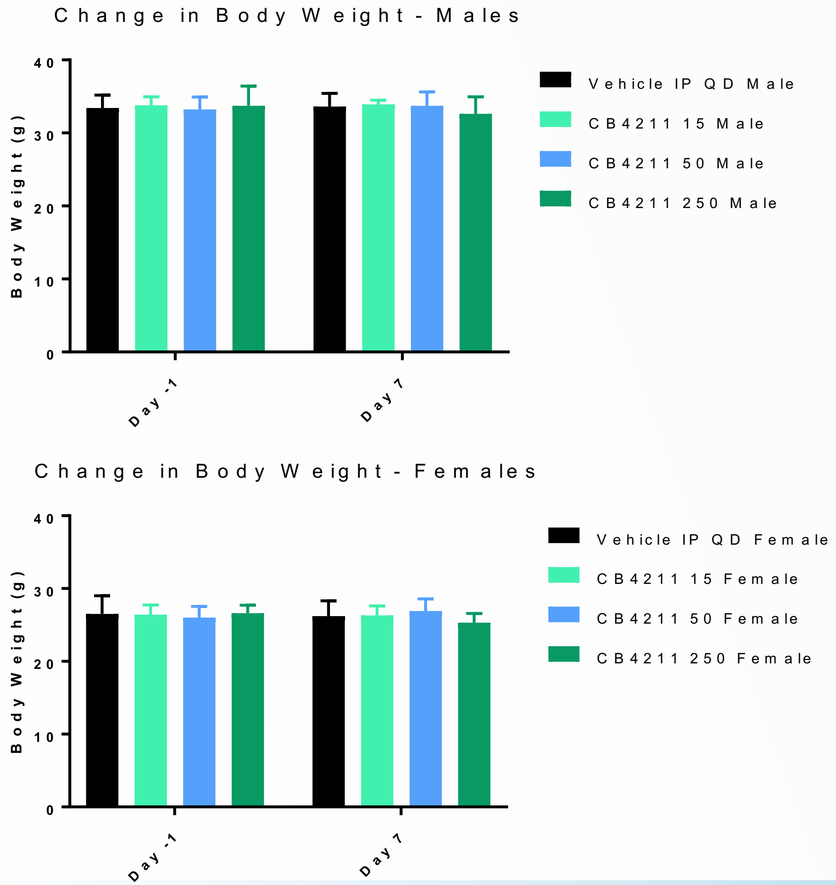
CB4209/11: Potential for Treating NASH

Significant Efficacy in Widely Used Animal Model of NASH

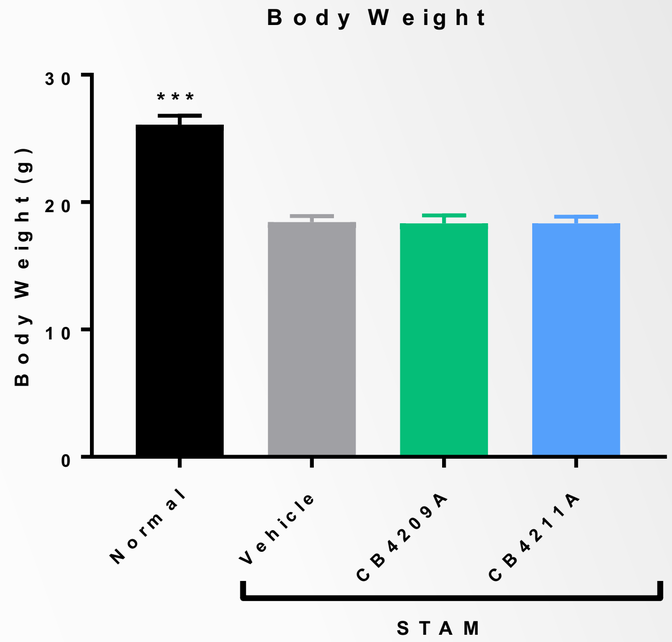
- Treatment of STAM NASH mice with either CB4209 or CB4211 resulted in:
  - Significant reduction of the non-alcoholic fatty liver disease (NAFLD) activity score
  - Significant improvement in liver triglycerides and plasma ALT (marker of liver damage)
- Confirms anti-steatotic effects seen in prior DIO mouse studies
- CB4211 regulates free fatty acid release from fat cells reducing lipotoxicity.
- Company presented details of its preclinical NASH/DIO results at the AASLD Liver Meeting® 2017, in Washington DC
- “Lipotoxicity is a key mechanism in a number of metabolic diseases and is thought to be responsible for the progression of NAFLD to NASH”. - NASH expert and CohBar advisor Dr. Rohit Loomba MD, Professor of Medicine in the Division of Gastroenterology at UC San Diego and Director, NAFLD Research Center

CB4209/11: No Weight Loss in Non-Obese Animals

No Effect on Weight in Healthy Mice
(7 Day Mouse DRF Study)



No Effect on Weight in Underweight STAM NASH Mice
(21 Day STAM NASH Study)



CB4209/11 Preclinical Differentiators

- Novel mechanism of action – regulation of fat cells (adipocytes)
- Significant reduction in NAS score (STAM)
- Significant reduction of liver steatosis, liver enzymes and triglycerides (DIO /STAM)
- Normalization of body weight in obese animals (DIO)
- No effect on body weight in non-obese animals
- Synergy with GLP-1 mechanism supports potential use with other drugs (DIO)
- 3x greater body weight loss than liraglutide (DIO)
- 2x improvement in fat mass ratio than liraglutide (DIO)

Path to the Clinic

CB4209 and CB4211 entered IND enabling activities using leading contract research organizations:

- Initial assessment of safety in dose range finding studies - completed
- Scale up of analog synthesis and process optimization - completed
- Selection of final candidate for remaining IND activities (CB4211) - completed
- GMP manufacturing and stability studies of drug substance - completed
- Safety evaluation in standard preclinical studies – nearing completion
- Development, GMP manufacturing and stability studies of clinical formulation – on track
- Development of initial clinical study protocol and supporting materials – on track
- Planning for pre-IND meeting with FDA and initiation of clinical testing – on track

Clinical Strategy Considerations

Typical NASH Clinical Development Path

- Phase 1 SAD/MAD in healthy normal subjects followed by Phase 2a in NASH patients
- Disadvantages:
  - NASH Phase 2a efficacy studies are costly and time consuming
  - Multiple studies are competing for the same patient population - slow to enroll
  - Treatment periods of >12 weeks are needed for biopsy-driven outcomes
  - Speculative dose selection, not based on activity data
  - Would not provide activity readout until mid 2020

Opportunity for an Alternative Clinical Path for CB4211

- Body weight effects of CB4211 are seen early in obese animals – weight normalization
- Similar doses of CB4211 also reduce liver fat and NAS score in NASH animals
- CB4211 has unique properties enabling changes in body weight and liver fat to be used as indicators of activity related to NASH and obesity in a short term clinical study

CB4211 Phase 1a/b Study Design

- Conventional Phase 1a SAD/MAD in healthy normal subjects
  - Safety, tolerability, PK, selection of maximum dose, cardiovascular safety assessment
- Placebo controlled Phase 1b arm in obese subjects with NAFLD– exploratory activity study
  - 4 week treatment period with once daily subcutaneous dosing at maximum dose
- Activity assessed by change in liver fat (MRI-PDFF) and body weight
  - Precedent for significant effect on liver fat after 4 week treatment (Merck Phase 1b study NCT01431521)
  - Provides earlier data on changes in liver fat (foundational event of NASH) and bodyweight in obese humans (primary endpoint for obesity)

Phase 1a/b Design:

CB4211 Clinical Plan

Activities:

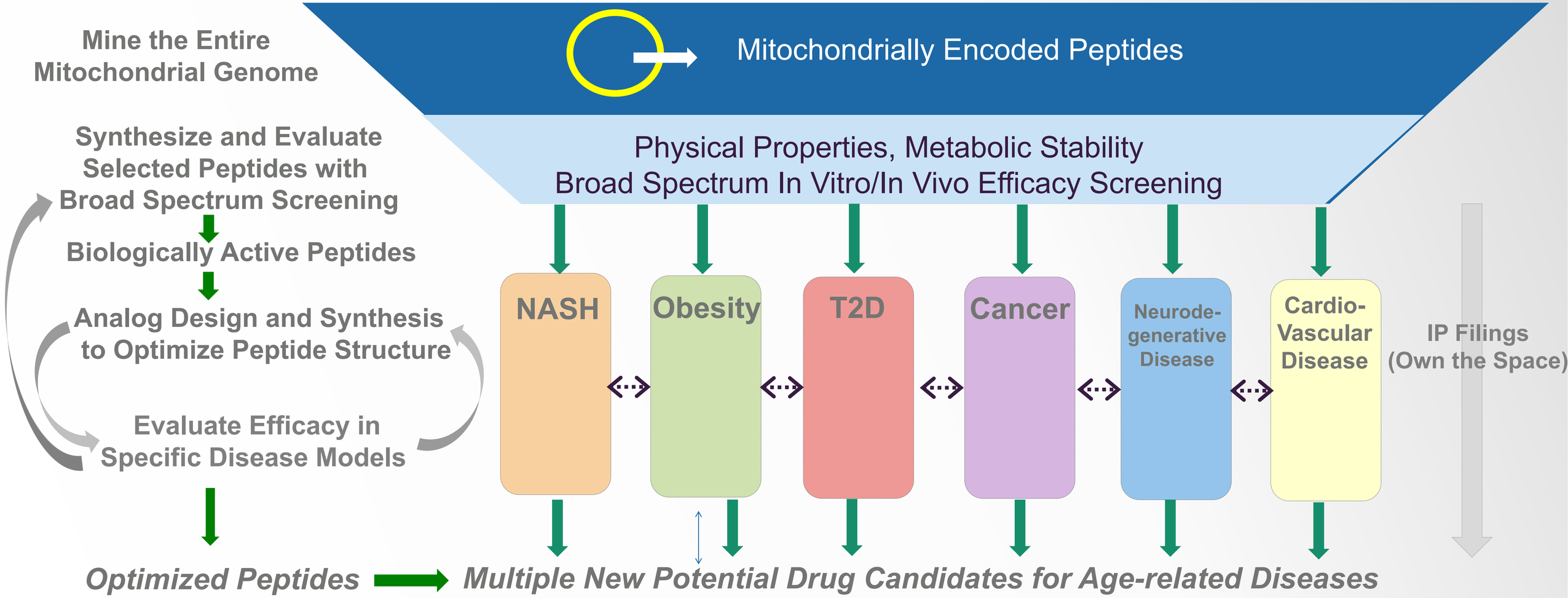
- Generating safety data and drug supply to support the expanded scope – on track
- Working with clinical CRO partner Covance on clinical logistics
- Pre-IND meeting with FDA – planned early 2018
- First human dose - planned mid 2018
- Top-line data from Phase 1a/b – planned early 2019

Advantages compared to a typical Phase 2a NASH study:

- Potentially provides a readout of activity relevant to NASH and obesity more than a year earlier
- Saves approximately $3-5M in cost of achieving an initial activity readout
- Validates dose selection before starting larger, more expensive studies
- Reduces risk for Phase 2 and subsequent studies

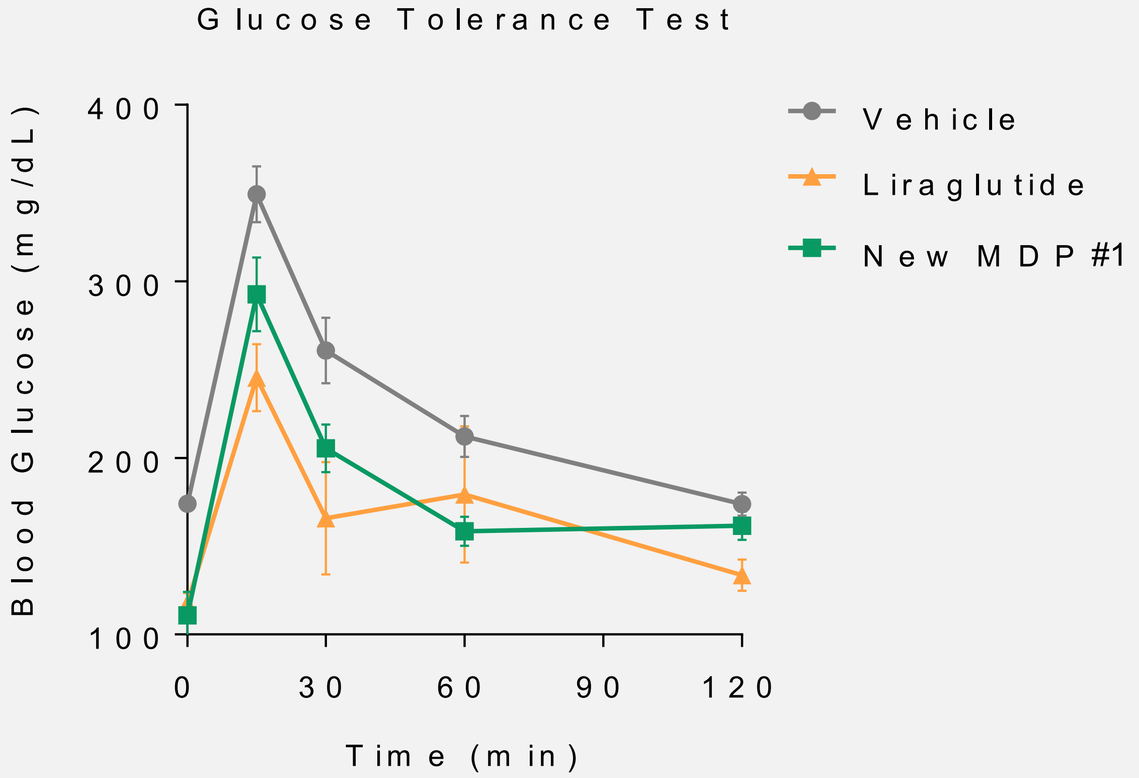
Platform and New Peptides

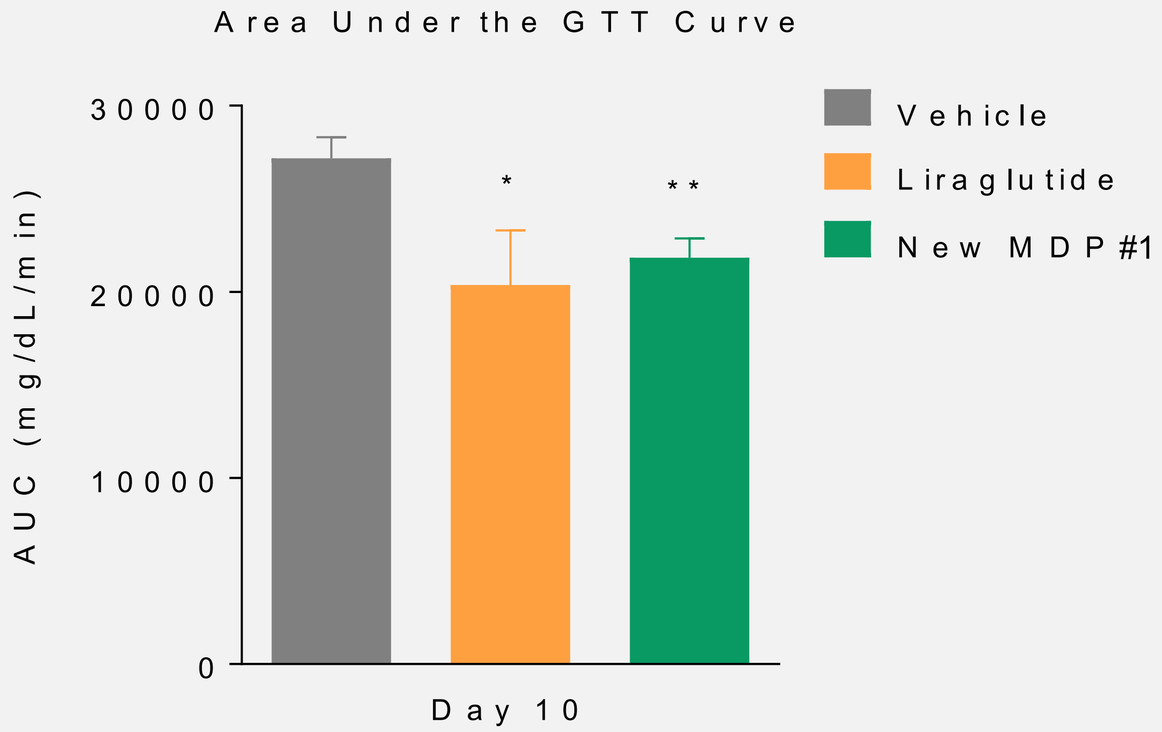
CohBar's MBT Drug Discovery Platform/Process



New MDPs – Improved Glucose Tolerance in Mice

Glucose Tolerance Test – Bolus Injection of Glucose
(10 Day DIO mouse study)

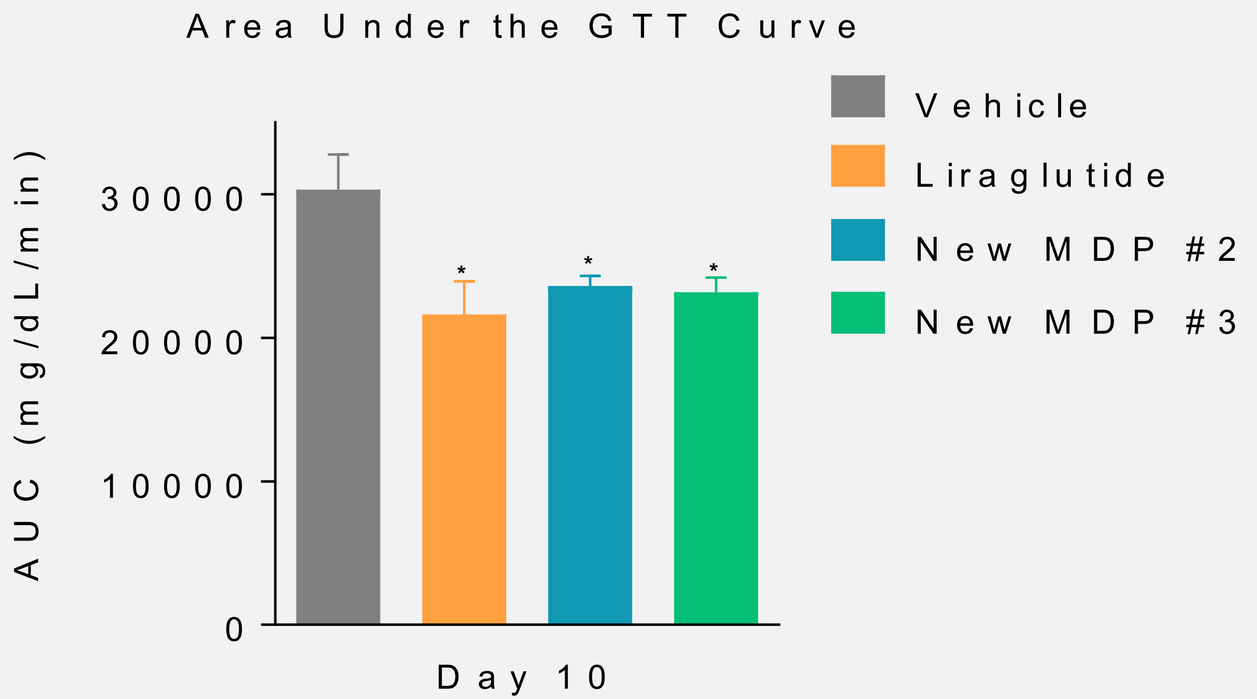




(New MDPs not yet optimized)

New MDPs – Improved Glucose Tolerance in Mice

Numerous Novel CohBar Peptides Decrease Glucose Exposure in Glucose Tolerance Tests
(10 Day DIO mouse study)



(New MDPs not yet optimized)

New MDPs - In Vitro Inhibition of Tumor Cell Proliferation

20 Different Human Tumor Cell Types

20 Novel Peptide Analogs

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T
1	8	4	5	8	8	4	6	5	5	7	6	10	6	5	7	7	8	8	9	7
2	9	5	8	8	8	8	7	7	8	8	7	8	8	6	7	8	8	5	8	8
3	8	6	8	3	6	8	8	4	8	5	7	6	7	5	8	8	7	8	8	6
4	8	8	9	6	8	8	9	8	8	8	8	9	8	6	8	8	8	8	8	9
5	8	8	12	8	6	8	9	8	8	8	8	10	8	8	9	8	8	8	8	9
6	8	8	8	3	9	8	8	8	8	8	6	8	8	8	7	8	8	9	8	8
7	8	8	9	8	8	9	8	8	8	7	8	8	8	6	8	8	8	9	8	8
8	8	6	8	7	8	7	7	7	7	8	7	6	8	6	8	8	8	8	8	10
9	9	8	8	8	8	6	7	8	5	8	6	8	8	6	8	8	9	8	8	8
10	8	7	8	8	8	8	7	7	8	7	6	8	8	8	7	8	8	8	8	8
11	8	9	10	3	8	8	9	7	9	7	8	11	8	7	8	9	8	8	8	9
12	8	8	10	6	9	8	9	8	9	8	8	9	8	7	9	9	8	8	8	9
13	8	8	10	6	7	7	8	8	8	8	7	8	7	8	9	8	8	8	8	9
14	9	8	12	9	6	8	8	9	8	6	8	10	8	9	8	8	8	8	8	9
15	7	2	4	3	6	8	5	5	4	2	1	3	2	6	4	7	4	2	8	1
16	5	8	4	5	4	7	5	4	4	4	2	1	6	3	4	5	6	6	7	2
17	7	7	5	4	6	8	8	5	7	6	6	3	5	4	5	6	8	4	6	6
18	7	7	6	7	6	7	6	5	6	5	6	3	6	6	6	7	8	8	8	6
19	9	7	7	7	11	8	8	6	8	5	8	7	6	5	8	8	8	8	8	8
20	10	8	7	5	6	6	8	6	6	8	7	6	8	4	8	9	9	8	8	8

Lower number/more red = less proliferation

Significant Reduction in Tumor Cell Growth in Vitro
(Human Tumor Cells in Vitro)

- Systematic evaluation of human tumor cell proliferation
- 20 different human tumor cell types screened against 20 CohBar peptides
- Evidence of effects across a broad range of tumor types

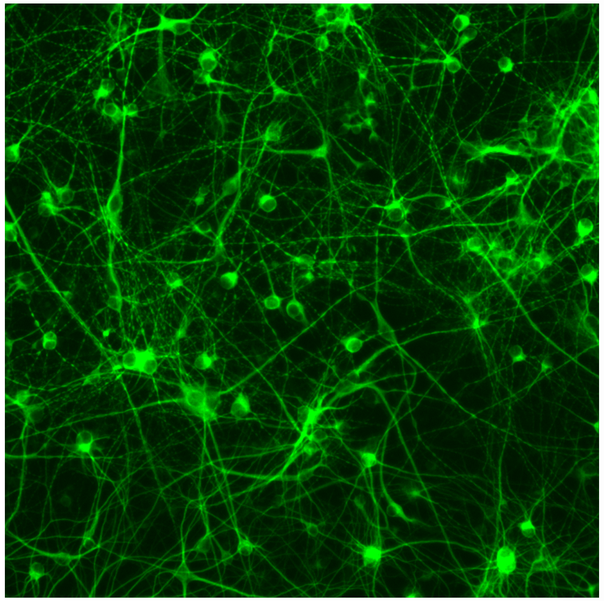
(New MDPs not yet optimized)

New MDPs – Neuroprotective Effects in Cultured Neurons

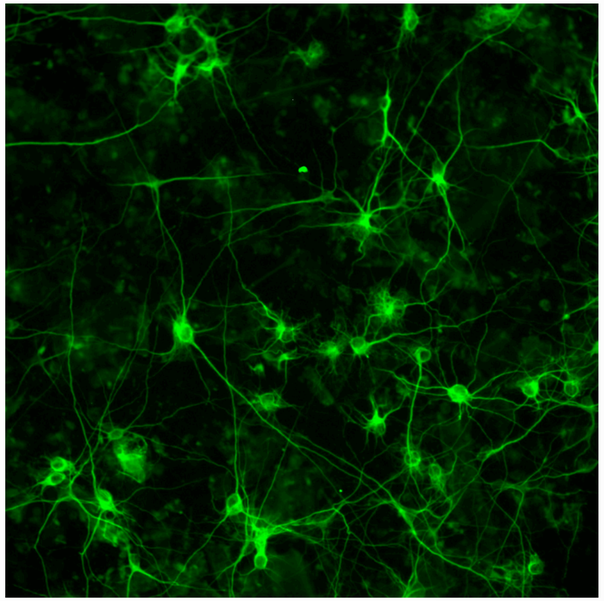
Primary Cortical Neurons (Rat)

Primary Cortical Neurons Exposed to Amyloid Beta

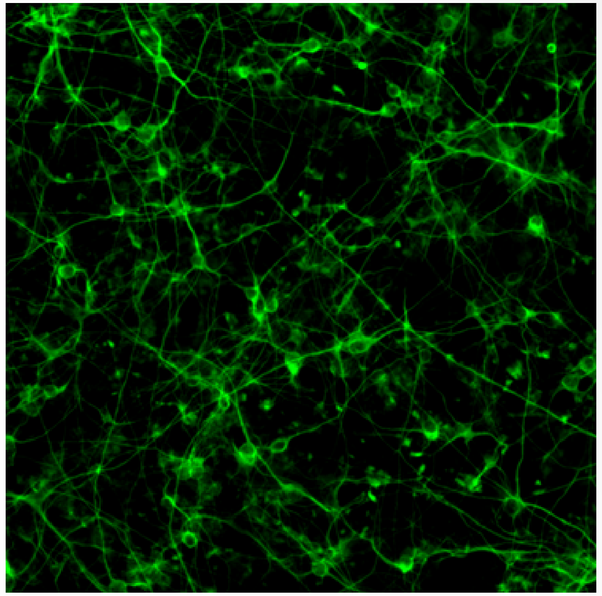
New MDP #4 Protects Neurons and Neurite Network from Amyloid Beta Toxicity



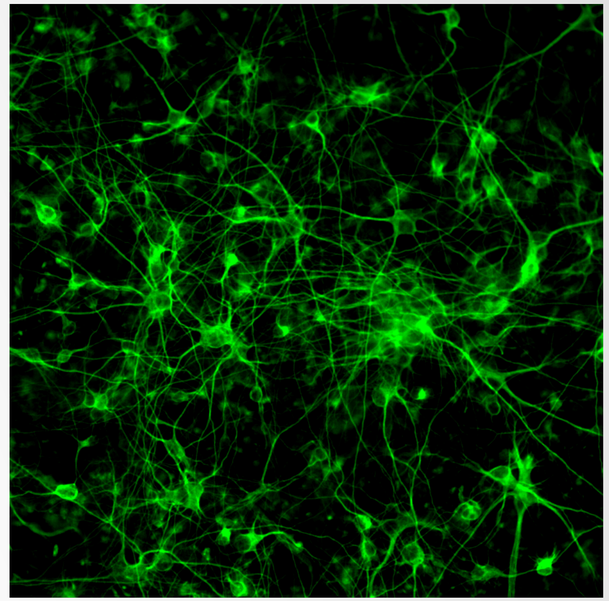
Untreated Control



$A\beta_{1-42}$ (20 µM) at 24 hr



$A\beta_{1-42}$ (20 µM)
+ New MDP #4 (100 nM)



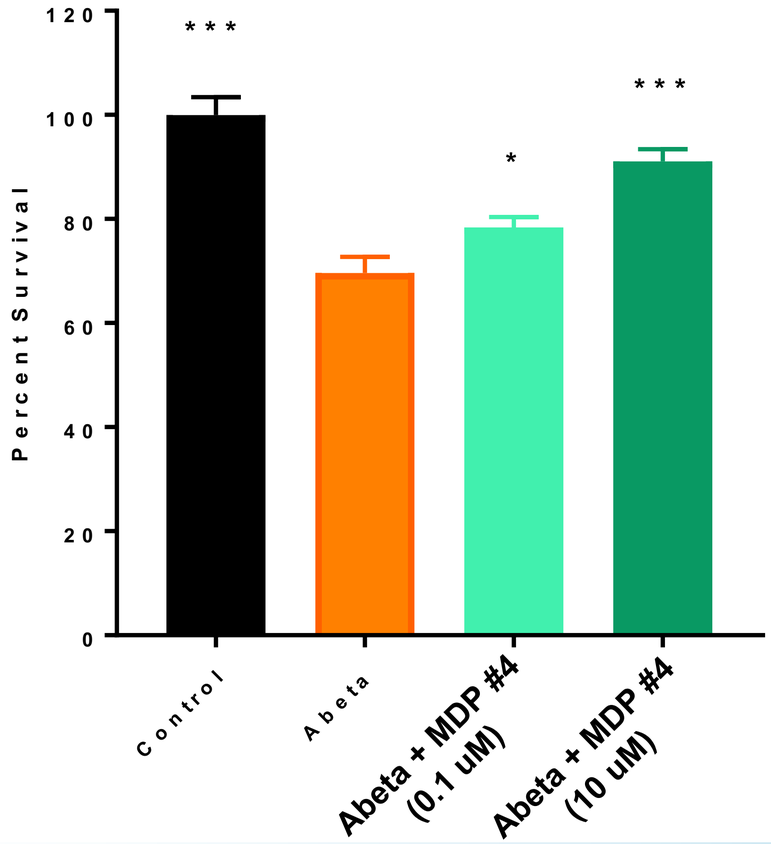
$A\beta_{1-42}$ (20 µM)
+ New MDP #4 (10 µM)

Example of a CohBar New MDP that is Neuroprotective in this Cell-Based Model of Alzheimer's Disease

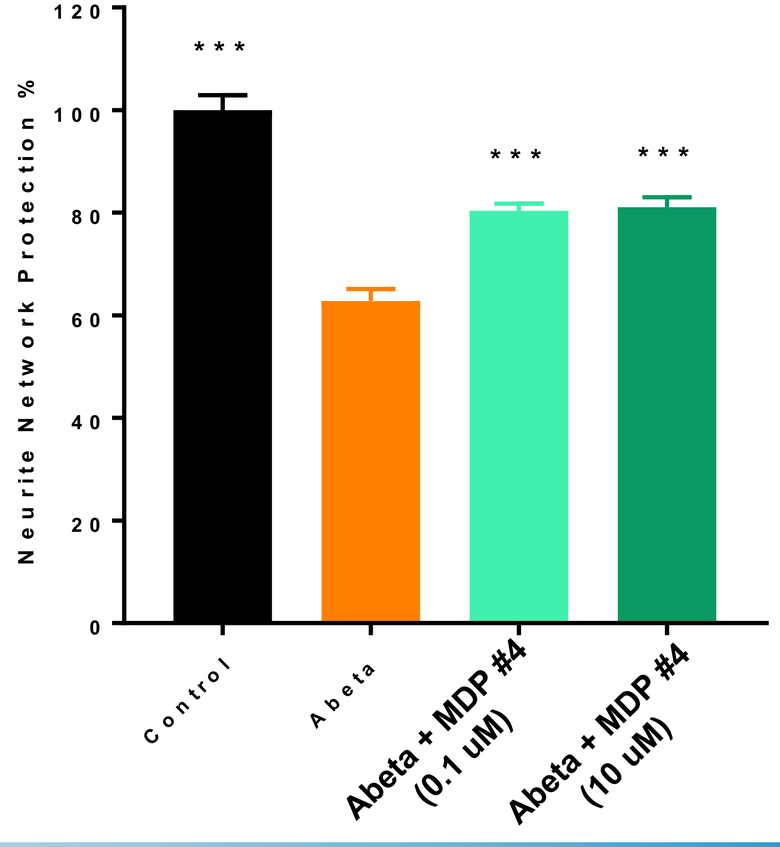
(New MDPs not yet optimized)

New MDPs – Neuroprotective Effects in Cultured Neurons

Improves Survival of Rat Primary Cortical Neurons Exposed to Amyloid Beta



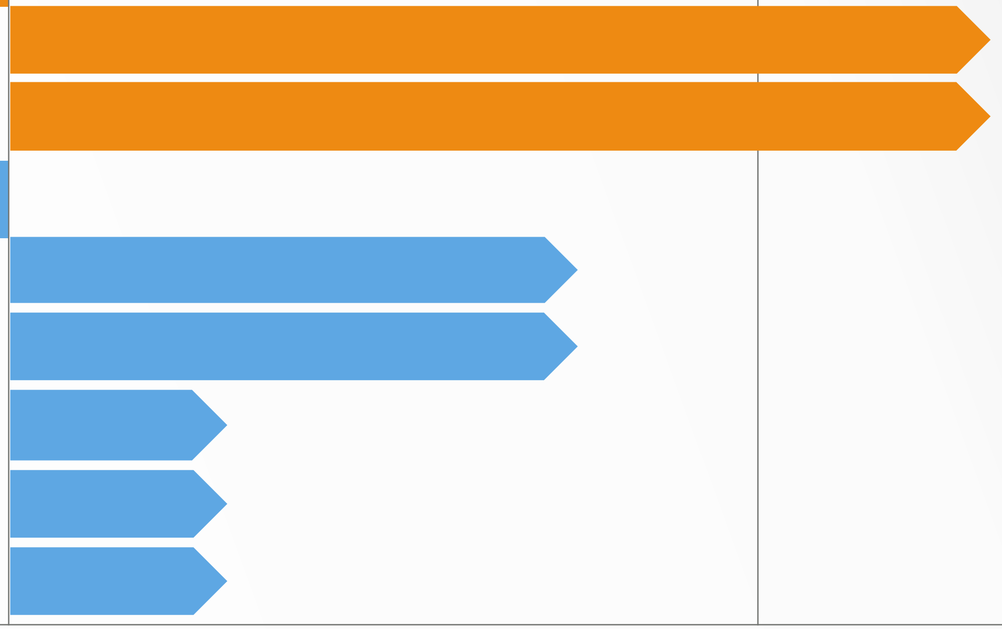
Protects Neurite Network from Exposure to Amyloid Beta



(New MDPs not yet optimized)

CohBar's Pipeline

Target Indication	Preclinical		IND Enabling Activities	Phase 1
	Discovery	Optimization		
CB4211 (MOTS-c Analog)				
NASH				
Obesity				
MDP Analogs				
Obesity/NASH				
Type 2 Diabetes				
Cancer				
Neurodegenerative Diseases				
Cardiovascular Disease				



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Market Perspective & Partnering

CB4211 Potential Value

Preclinical Differentiator	Potential Value
☑ Novel Mechanism of Action ☑ Significant reduction in NAS score ☑ Normalization of Body Weight ☑ Significant reduction of liver steatosis, liver enzymes and triglycerides ☑ 3x greater body weight loss ☑ 2x improvement in fat mass ratio ☑ Synergy with GLP-1	• A novel mechanism that addresses a foundational event in metabolic dysfunction • The ability to regulate metabolism and normalize body weight is unique within the industry • Potential to treat NASH and Obesity • Reduction of NAS score by 33% in the STAM model compares favorably to the leading NASH assets • DIO model demonstrated superiority over liraglutide, the market leader for obesity • Combination with other drugs, like GLP-1, could improve their efficacy and safety

Source: CohBar team analysis.

CB4211 Market and Pharma Perspective

	NASH	Obesity
US Patients:	Estimated 12-29 million	Estimated 80 million
Market Size:	Greenfield	$4 billion (all indications)
Market Perspective:	• No approved therapeutics - Numerous drugs in development • Addressable market could exceed $40 billion • Diagnosis and treatment rapidly evolving • Combination therapy could optimize patient outcomes • Increasingly linked to comorbidities: obesity, T2D, cancer and cardiovascular disease	• No safe and effective therapeutics - Substantial cardiovascular risks - Insufficient body weight loss • Inexpensive generics prescribed except for GLP-1 (revenue leader) • Evolving reimbursement • Increasingly linked to comorbidities: NASH, T2D, cancer and cardiovascular disease
Pharma Perspective:	• Justifies *substantial* investment • Supports multiple block-buster drugs • Looking to acquire and develop multiple assets that: - Act at different stages of the disease - Work via different mechanisms of action - Are at all stages of development	• Need safe and efficacious drugs • Existing mechanisms for obesity are considered “toxic”, novel mechanisms are attractive • Treating obesity is complementary to the treatment of NASH

NASH Partnerships

	Biotech	Pharma	Drug	Transaction	Year
☑ Torreya	MiNA	Boehringer Ingelheim	RNAi therapeutics	$358M commitment for up to three ***preclinical*** NASH *"*targets"	**2017**
☑ Torreya	Dicerna		RNAi therapeutics	$200 million (upfront payment, development and commercial milestone payments, royalties) for an undisclosed ***preclinical*** asset.	
	Bird Rock Bio	J&J	namacizumab	Undisclosed option to acquire company for a ***Phase 1a*** *asset*	
☑ Torreya	Conatus Pharma	Novartis	Emricasan (pan-caspase inhibitor)	$50M upfront with undisclosed licensing terms for a ***Phase 2a*** *asset*	**2016**
	Nitto Denko	BMS	ND-L02-s0201	$100M upfront with undisclosed licensing terms for a ***Phase 1b*** *asset*	
	Akarna Therapeutics	Allergan	AKN-083 (FXR agonist)	$50M acquisition for a ***preclinical*** *asset*	
	Tobira Therapeutics		Cenicriviroc (TAK-652)	$1.7B acquisition for a ***Phase 2*** *asset*	
	Nimbus	Gilead	NDI-010976 (ACC inhibitor)	$1.2B asset acquisition for a ***Phase 1*** *asset* • $400M upfront $800M earn-outs with $200M earned Nov 2016	
	Pharmaxis	Boehringer Ingelheim	PXS4728	$233M acquisition of a ***Phase 1*** *asset* • $29M upfront, $58M in clinical and $147M in regulatory/commercial milestones	**2015**
	NGM Biopharma	Merck	NP-201	$450M license agreement for ***preclinical*** *asset* • $94M upfront, $106M equity at a 20% premium, $250M in development funding	
☑ Torreya	Phenex	Gilead	PX102, PX103, PX104 (FXR agonists)	$470M acquisition of a ***Phase 1*** *asset*	

Source: Global Data and Torreya Partners

Partnering Program

Objective: Non-dilutive funding to support development of multiple CohBar pipeline assets

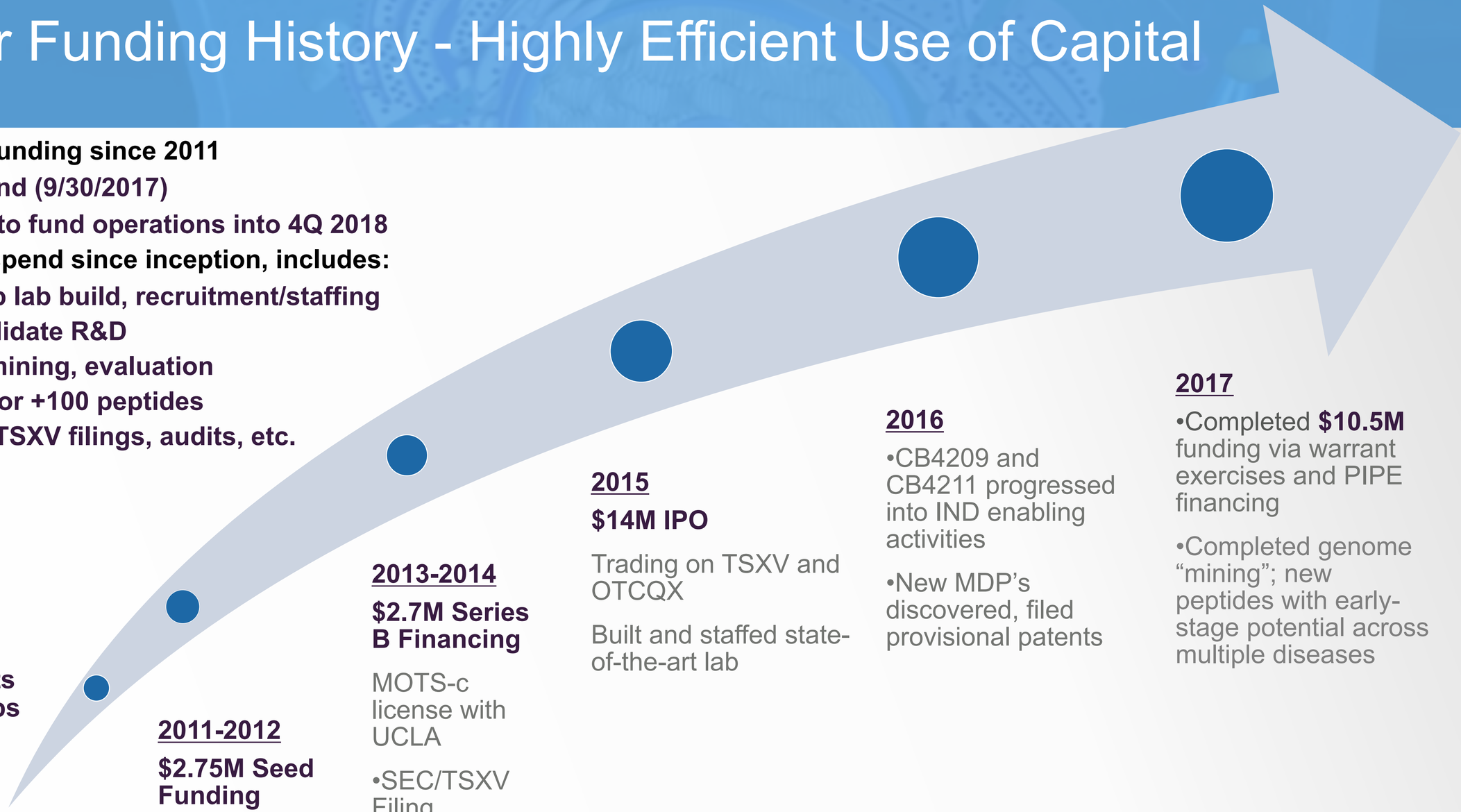
- Prioritizing new assets for internal vs. partnered development
  - Matching our assets with partner's interest, experience and portfolio
- Exploring/evaluating opportunities for partnering CB4211
- Retained Torreya Partners as our strategic partnering advisor
  - Proven track record (over 150 partnerships since 2007), strong global pharma network
  - Experienced and successful in partnering NASH assets:
    - ✓ Dicerna partnership with Boehringer Ingelheim, $200M total deal size
    - ✓ Conatus partnership with Novartis, $50M upfront + backend
    - ✓ Phenex sale to Gilead for $470M

CohBar INC.

Company Funding, Objectives and Plans

CohBar Funding History - Highly Efficient Use of Capital

- **~$31M Total funding since 2011**
- **$10.3M on hand (9/30/2017)**
  - **Expected to fund operations into 4Q 2018**
- **~$21M Total spend since inception, includes:**
  - **Ground up lab build, recruitment/staffing**
  - **Lead candidate R&D**
  - **Genome mining, evaluation**
  - **IP filings for +100 peptides**
  - **IPO, SEC/TSXV filings, audits, etc.**



1998

~$30M in grants to founders labs (ongoing)

First MDP discovered

2011-2012

$2.75M Seed Funding

Signed UCLA License

2013-2014

$2.7M Series B Financing

MOTS-c license with UCLA

•SEC/TSXV Filing

2015

$14M IPO

Trading on TSXV and OTCQX

Built and staffed state-of-the-art lab

2016

•CB4209 and CB4211 progressed into IND enabling activities

•New MDP's discovered, filed provisional patents

2017

•Completed **$10.5M** funding via warrant exercises and PIPE financing

•Completed genome "mining"; new peptides with early-stage potential across multiple diseases

CohBar's Efficient Semi-Virtual Operating Model

- Focus on proprietary internal capabilities while outsourcing commoditized activities to a competitive third party marketplace
- Working with leading CROs and academic institutions enables a powerful, scalable, flexible and efficient operating plan
- Substantially reduces our infrastructure and minimizes investments in capital equipment
- Facilitates partnering and technology transfer

COVANCE
SOLUTIONS MADE REAL®

BACHEM
PIONEERING PARTNER FOR PEPTIDES

integritybio

SMC Laboratories, Inc.

MPI RESEARCH

charles river

USC University of Southern California

EINSTEIN
Albert Einstein College of Medicine

MAYO CLINIC

University of CINCINNATI

2017 Progress to Date

Lead Clinical Candidate

- Significant progress through IND enabling activities
- Confirmed DIO activity in STAM NASH model, presented at AASLD Liver Meeting 2017
- Developed an expanded Phase 1a/b clinical plan for early activity readout

New MDPs

- Completed our genome mining process targeting all therapeutically relevant peptides encoded within the mitochondrial genome
- Identified a number of MDPs with early indication of significant therapeutic potential for multiple diseases
- Filed additional provisional, PCT, and international patent applications (100+ peptides to date)

Strategic Partnering

- Retained Torreya Partners, expanded our partnering program and prospective partners

Investment Community

- Increased CohBar awareness and shareholder base in US investment community

Funding

- Completed $5.3M warrant funding in January, $5.2M PIPE in July
  - Extended our cash runway, provided flexibility for optimal timing/value in partnering

2017-2018 Objectives and Plans

Lead Program CB4211 in the Clinic

- Complete pre-clinical activities and IND filing
- Initiate/execute expanded Phase 1a/b clinical plan

New MDPs

- Continue to evaluate therapeutic potential of new peptides (and further secure IP rights)
- Optimize and prioritize best analogs for therapeutic potential and market opportunities

Strategic Partnering

- Continue to develop potential partner interest with new preclinical and clinical data
- Explore partnership opportunities for earlier stage assets

2017-2018 Objectives and Plans (cont.)

Investment Community

Background

- Predicted inflection in late 2017
  - Liver Conference (AASLD)
  - Investor awareness programs
  - Continued progress toward the clinic
- Completed midyear raise to extend cash runway, provide flexibility for optimal partner timing/value

Going Forward

- Uplist to the NASDAQ in process
- Establish an institutional investor base

Funding

- Evaluate potential funding alternatives
  - Accelerate new peptides toward the clinic
  - Extend cash runway

Summary – We believe CohBar will increase healthy lifespan

- Strategically targeting the metabolic dysfunction underlying global epidemic of diseases of aging
- Breakthrough research and discoveries from decades of research by our world-renowned founders
- Unique opportunity to address enormous medical needs and related market opportunities
- First MBT, with unique characteristics and therapeutic potential for treatment of multiple diseases, on track to the clinic
- Growing inventory of new peptides showing significant early-stage therapeutic potential
- Building an expanding pipeline with potential for development as first-in-class therapies targeting multiple diseases of aging
- Significant strategic IP ownership/protection in place to preserve our lead in the MBT space
- Partnering strategy and expertise in place, process underway targeting non-dilutive funding
- Plans to expand our visibility in the investment community and access to funding
- Strong scientific and management team with demonstrated ability to execute quickly and efficiently
- Cash and investments in hand to fund operations into 4Q2018

Our Vision

CohBar's vision is to increase healthy lifespan by developing effective treatments for the underlying metabolic dysfunction driving the diseases of aging and age-related mortality, such as obesity, fatty liver disease, NASH, cancer, Type 2 Diabetes, and cardiovascular and neurodegenerative diseases.

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