CohBar

Mitochondria Based Therapeutics

Cantor Healthcare Conference

October 2019

WWW.COHBAR.COM

NASDAQ: CWBR

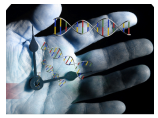
Forward Looking Statements

This presentation includes forward-looking statements which are not historical facts within the meaning of the Private Securities Litigation Reform Act of 1995. Forward-looking statements are based only on our current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, projections, anticipated events and other future conditions. In some cases you can identify these statements by forward-looking words such as “believe,” “may,” “will,” “estimate,” “continue,” “anticipate,” “intend,” “could,” “should,” “would,” “project,” “plan,” “expect,” “goal,” “seek,” “future,” “likely” or the negative or plural of these words or similar expressions. Examples of such forward-looking statements including but not limited to statements regarding anticipated outcomes of research and clinical trials for our lead candidate, CB4211, or other mitochondria based therapeutic (MBT) candidates; expectations regarding the future market for any drug we may develop; expectations regarding the growth of MBTs as a significant future class of drug products; statements regarding future partnership and collaboration opportunities; statements regarding anticipated therapeutic properties and potential of our MBTs or the properties, potential and effects of newly-discovered mitochondrial-derived peptides; statements regarding our capital resources and future financing plans, including our ability to successfully undertake financing activities; and expectations regarding our ability to effectively protect and expand our intellectual property. 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: our ability to successfully advance drug discovery and development programs, including the delay or termination of ongoing clinical trials; our possible inability to mitigate the prevalence and/or persistence of the injection site reactions, receipt of unfavorable feedback from regulators regarding the safety or tolerability of CB4211 or the possibility of other developments affecting the viability of CB4211 as a clinical candidate or its commercial potential; results that are different from earlier data results including less favorable than and that may not support further clinical development; our ability to raise additional capital when necessary to continue our operations; our ability to recruit and retain key management and scientific personnel; and our ability to establish and maintain partnerships with corporate 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: Mitochondria Based Therapeutics designed to treat chronic diseases and to increase healthy lifespan

- **Mitochondria:** Central role in energy production, signaling and communication. Mitochondrial dysfunction plays a underlying role in a number of chronic and age-related diseases.
- **Platform Technology:** Evaluation of over 100 peptides encoded in the mitochondrial DNA and their analogs for potential development into novel therapeutics
- **Clinical stage:** CB4211 in Phase 1a/1b trial for NASH and obesity. Improvement in NAS score, liver fat and triglyceride levels and body weight reduction shown in preclinical models
- **New peptides have wide range of effects in preclinical models:** Antifibrotic effects in IPF, enhanced killing of cancer cells by human blood cells in vitro**,** improved glucose tolerance in Type 2 diabetes
- **Potential Indications:** NASH, obesity, T2D, fibrotic diseases, cancer, cardiovascular and neurodegenerative diseases
- **IP:** 65+ CohBar patent filings, 8 issued patents licensed from UCLA/Albert Einstein/Mayo Clinic
- **Experienced team:** Successful track record of drug discovery, development and partnerships
- **Financial:** $16.8M 2Q 2019, runway expected into 3Q 2020, NASDAQ: CWBR

CohBar in Israel: Geroscience Summit September 4-5, 2019



- At the Weizmann Institute of Science in association with The National Institute for Aging - sponsored by The Nathan Shock Centers, in partnership with the Sagol Institute for Longevity Research
- Presentations by leading international and Israeli researchers on aging
- International Perspectives on Geroscience, an NIH wide initiative including 21 of 27 NIH institutes
- Presentations by 3 CohBar Founders:
  - **Dr. Nir Barzilai** – Improving health span of elderly: Not a science fiction anymore
    We have made progress in launching a large clinical study that aims to prove aging can be targeted but also to get FDA approval, so biotech and pharma will develop better drugs and their combinations to realize our potential health span.
  - **Dr. Pinchas Cohen** - Systems Biology of the Mitochondria — A Discovery Pathway for Aging-Related Pathologies
    We identified multiple open-reading-frames (ORFs) within the mitochondrial genome that encode putative peptides we call Mitochondrial-Derived-Peptides (MDPs) which represent a sub-class of a growing group of novel micro-peptides (from both mtDNA and nuclear chromosomes) which serve as signals related to cell and organismal protection and energy expenditure.
  - **David Sinclair, PhD.** - Promises and challenges of translating aging research
    We are at turning point in medicine, one that will allow us to dramatically improve human healthspans. The past 20 years of research into aging has seen great strides in our understanding of why we age and how to delay it, preventing most age-associated diseases.

מכון ויצמן למדע
WEIZMANN INSTITUTE OF SCIENCE

International Perspectives on GEROSCIENCE Meetings 2019 — sponsored by the NATHAN SHOCK CENTERS

THE SAGOL NETWORK

Year in Review 2018/2019

Research and Development:

- Initiated first clinical trial of CB4211
- Advanced new peptides with expanded disease targets
- Discovered new peptides target Apelin receptor, presented at ADA

Funding

- Completed $20M controlled equity offering

Leadership and Board

- Appointed new CEO
- Expanded Board of Directors

Partnering

- BIO 2019 - Increasing interest in “mitochondrial medicine” from pharma
- Focusing on CB4211 in the clinic and preclinical peptides

Intellectual Property

- Expanding portfolio of licensed patents and CohBar patent applications

Research in aging and age-related diseases increasingly focused on mitochondrial biology ("Mitochondrial Medicine")

A Mitochondrial Paradigm of Metabolic and Degenerative Diseases, Aging, and Cancer: A Dawn for Evolutionary Medicine

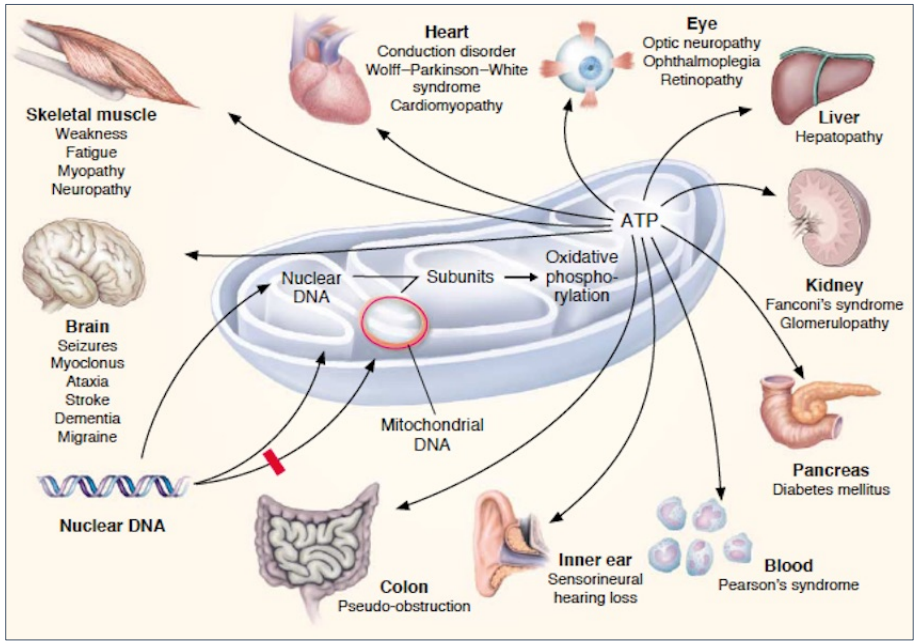
"….. Mitochondria are the only human genetic system that embodies the features necessary to explain the observed characteristics of the common age-relate diseases

…....mitochondrial decline and mtDNA damage are central to the etiology of the age-related metabolic and degenerative diseases, aging, and cancer."

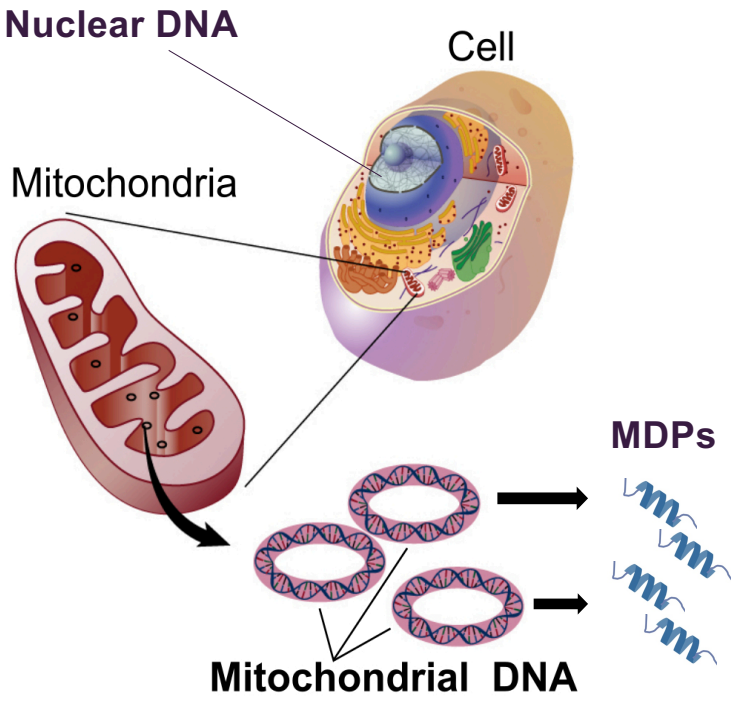
Douglas C. Wallace, Annual Review Genetics. 2005; 39; 359

Mitochondria: Central role in energy production, sensing, signaling, regulation, health, aging and disease

- Produce energy for the cell - ATP
- Regulate cellular metabolism
- Apoptosis – eliminate old cells
- Calcium storage and signaling
- Heat generation
- Intracellular lipid trafficking
- Regulate signaling transduction
- Cellular differentiation
- Control cell cycle and cell growth
- Hormonal signaling
- Anti-viral signaling protein release
- Stem cell regulation

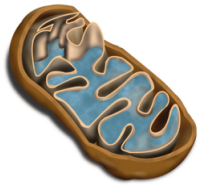
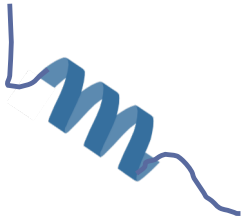
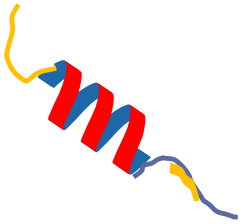


CohBar harnessing the potential of mitochondrial biology



- Mitochondria have their own genome
- CohBar's founders and scientists discovered sequences for peptides in the mitochondrial DNA – Mitochondrial derived peptides (MDPs)
- Certain MDPs have been found in circulation and within cells, and shown to be regulatory and signaling agents for metabolic and other processes
- CohBar has discovered, filed IP, and is evaluating over 100 MDPs and related analogs
- MDPs: A new source of therapeutic candidates
- Evolutionary biology: Certain MDPs conserved across species

Platform Technology: Evaluation of over 100 peptides encoded in the mitochondrial DNA and their analogs for development into novel therapeutics

Mitochondria (Powerhouse of the Cell)	Mitochondria Derived Peptide (“MDP”)	Optimized Peptide (“Analog”)	Mitochondria Based Therapeutic (“MBT”)
			

1 → 2 → 3

Identify	Optimize	Develop and Partner
Identify/characterize peptides with biological activity encoded within mitochondria	Optimize drug like properties	Prioritize for internal clinical development and partnership opportunities
File Intellectual Property (“Own the Space”)	Proprietary assays, Disease models	Advance lead therapeutic candidates to the clinic
Explore and quantify therapeutic potential across diseases	Match analogs with greatest therapeutic potential to medical needs and market opportunities	

Mitochondria Based Therapeutics: Potential disease targets

- Non-alcoholic steatohepatitis (NASH)
- Obesity
- Cancer
- Fibrotic Diseases
  - Pulmonary Disease: Idiopathic Pulmonary Fibrosis
  - Kidney Disease: Chronic Kidney Disease, Diabetic Nephropathy
  - Cardiovascular Disease: Heart Failure
- Type 2 Diabetes
- Neurodegenerative Diseases: Alzheimer's Disease

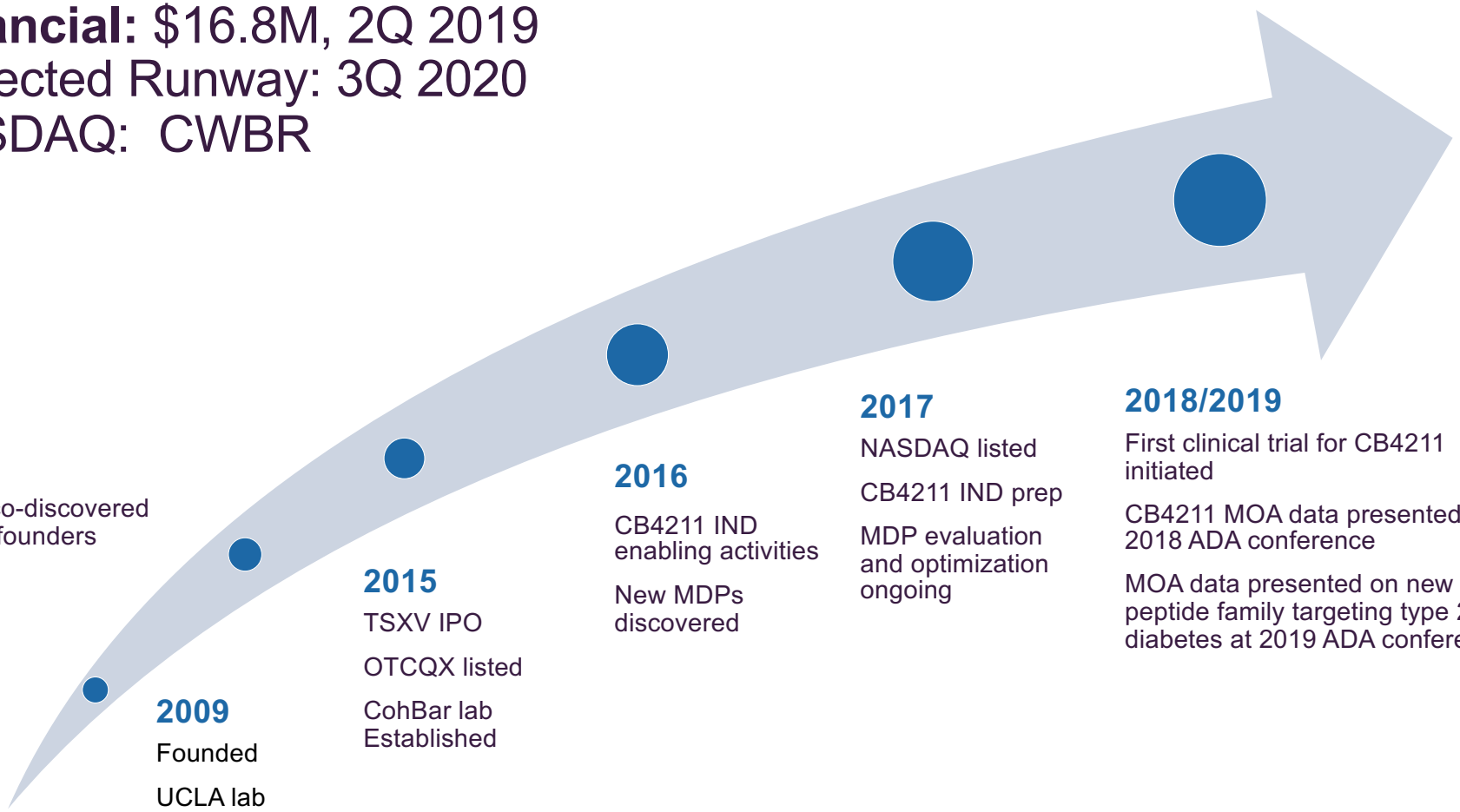
CohBar Pipeline

Target Indication	Preclinical Discovery and Optimization	IND Enabling Activities	Phase 1
CB4211			
NASH			
Obesity			
New Peptides			
NASH and Obesity			
Type 2 Diabetes			
Fibrotic Diseases			
Cancer			
Other Age-related Diseases			

Experienced Team: Successful track record of drug discovery, development and partnerships

Management	Prior Experience
Steven Engle Chief Executive Officer	20+ years of leadership experience with public biotech companies Development of breakthrough products in metabolic, autoimmune, oncologic, and infectious disease areas
Kenneth Cundy, PhD. Chief Scientific Officer	30+ years of drug discovery and development experience Development of 15 drugs with $100B+ in sales (including Hepsera®, Tamiflu®, Viread® and Horizant®)
Jeffrey Biunno, CPA, MBA Chief Financial Officer, Secretary and Treasurer	30+ years of financial experience Public, small, medium and large-cap companies Participated in three sales transactions
Jon Stern, MBA Chief Operating Officer, Director	Chief Executive Officer of CohBar from October 2013 to March 2016 Appointed to CohBar's Board of Directors in May 2014 Experience building companies in diverse industries

Financial: $16.8M, 2Q 2019
Expected Runway: 3Q 2020
NASDAQ: CWBR



2001

First MDP co-discovered by CohBar founders

2009

Founded

UCLA lab established

2015

TSXV IPO

OTCQX listed

CohBar lab Established

2016

CB4211 IND enabling activities

New MDPs discovered

2017

NASDAQ listed

CB4211 IND prep

MDP evaluation and optimization ongoing

2018/2019

First clinical trial for CB4211 initiated

CB4211 MOA data presented at 2018 ADA conference

MOA data presented on new peptide family targeting type 2 diabetes at 2019 ADA conference


CohBar
INC.
Research and Development Programs

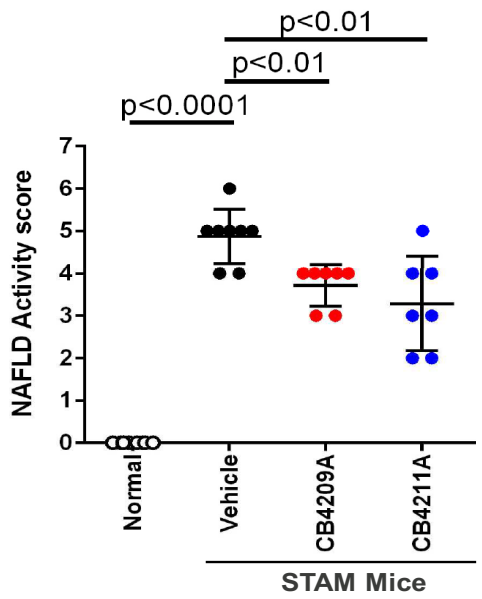
CB4211: Lead candidate in Phase 1a/1b trial for NASH and Obesity

- **CB4211:** a novel improved analog of the natural mitochondrial peptide MOTS-c
- **Preclinical data demonstrated**:
  - Reduction in the NAFLD Activity Score (NAS) in STAM® mice
  - Improvement in liver triglycerides and plasma ALT
  - Selective normalization of body weight in obese animals
- **Novel Mechanism of Action:** Enhances insulin effects on fat cells (adipocytes) leading to reduction of liver fat
- **Phase 1a/1b clinical trial ongoing:**
  - Phase 1a design: SAD/MAD safety, tolerability and PK in healthy subjects
  - Phase 1b design: Changes in liver fat, body weight, and biomarkers in obese NAFLD subjects
- **Activity readout:** relevant to NASH and obesity currently anticipated Q2 or Q3 2020
- **Synergistic effect:** with GLP-1 and PPARγ agonists in animal models of NASH and obesity

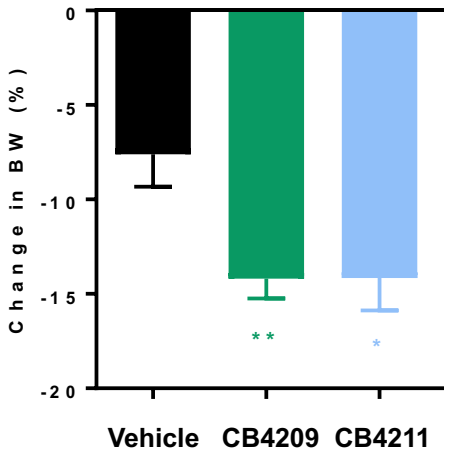
CB4211: Efficacy in Animal Models of NASH and Obesity

Reduced NAS Score in STAM Mice	Reduced Body Weight in DIO Mice	Reduced Liver Fat in DIO MIce

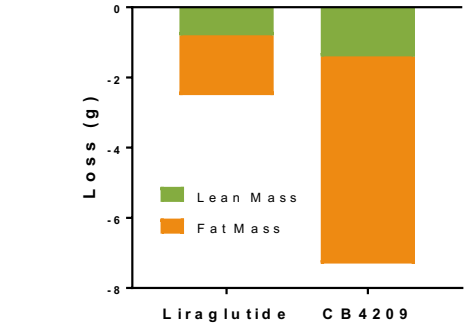
NAS Score at Day 21

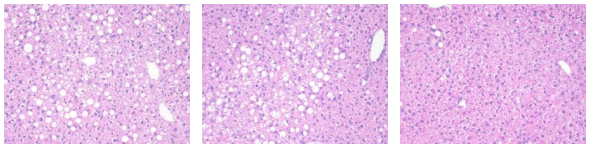


Body Weight Change at Day 21



Fat Mass and Liver Histology at Day 21





Vehicle Control | Liraglutide | CB4209

Fewer Fat Deposits

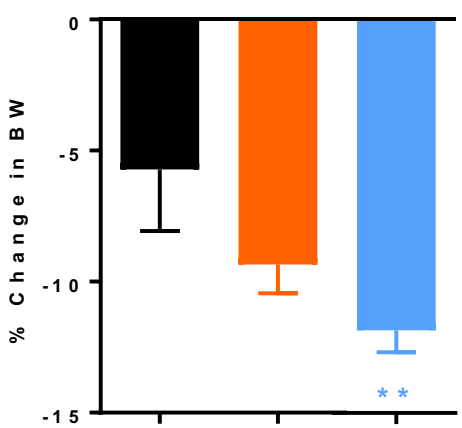
NAS = steatosis + inflammation + ballooning

[1]Source: Cundy KC, et al. AASLD Poster, 2017

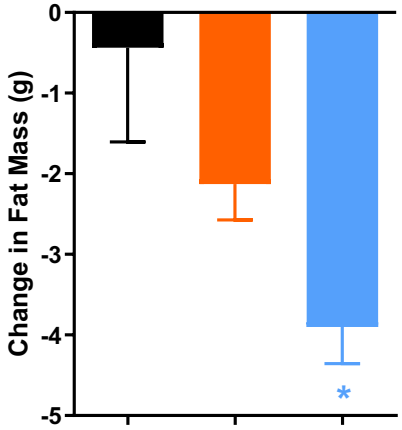
CB4211: Synergy with GLP-1 Agonist in NASH model enhances reduction in body weight and liver fat

More than 40% of NASH patients have diabetes and greater than 80% are obese[1]

Change in Body Weight at Day 21

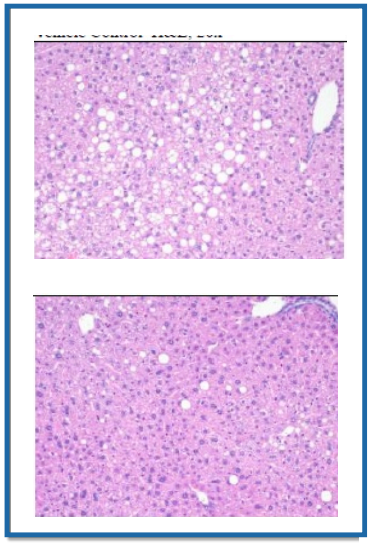


Change in Fat Mass at Day 21



Vehicle BID SC
Liraglutide QD IP
CB4211A 5 QD SC + Lirag

Liver Fat Deposits at Day 21

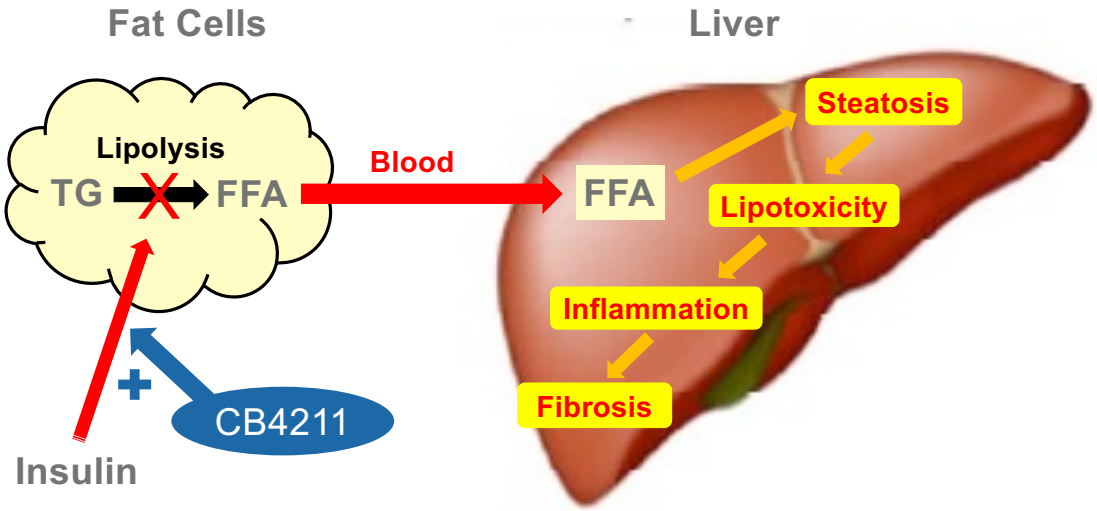


Liraglutide

Liraglutide + CB4211 5 mg/kg QD SC

[1] Diehl NEJM 2017
[2] Cundy KC, et al. AASLD 2017, Washington, DC.

CB4211: Acts on the Foundational Event in NASH



CB4211 reduces fatty acid (FFA) accumulation in liver - the foundational event in NAFLD-NASH progression

- Insulin normally regulates metabolism in liver, muscle, and fat cells
- NAFLD: loss of insulin sensitivity in fat cells, excess lipolysis, release of FFA into blood
- Excess fatty acid taken up by liver leads to fat deposits (steatosis), inflammation, and fibrosis
- CB4211 enhances signaling via the insulin receptor and decreases lipolysis in fat cells
- Results in decreased liver fat and NAS score in mouse models of NASH and obesity

Source: Grindstaff KG et al. ADA Poster Presentation, June , 2018

Preclinical: Multiple new peptides with wide range of effects in models of fibrotic disease, cancer, and type 2 diabetes

Fibrotic Diseases, new peptide analogs

- Decreased biomarkers of fibrosis in cultured human cells
- Decreased fibrosis and inflammation, and improved lung histopathology in mouse pulmonary fibrosis model
- Potential for efficacy in other fibrosis models including chronic kidney disease and heart failure

Cancer, new peptide analogs

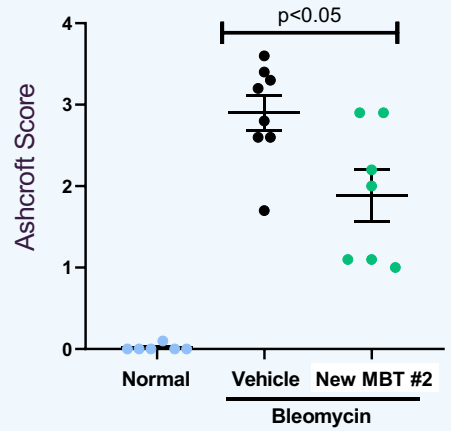
- Enhanced killing of cancer cells by human blood cells (PBMCs) in vitro
- Potential utility for immune system oncology indications alone or in combination

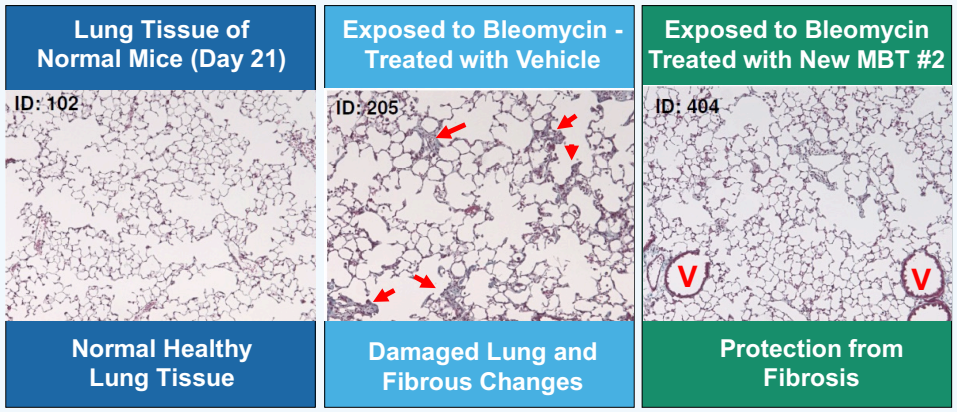
Type 2 Diabetes, CB5064 (MDP064) peptide analogs

- Improved weight loss and glucose tolerance in DIO mouse model
- MOA involves interaction with the apelin receptor, presented at ADA 2019

Fibrotic Diseases: Efficacy in Mouse Model of Lung Fibrosis (IPF)

Decreased Fibrosis in Lung Tissue





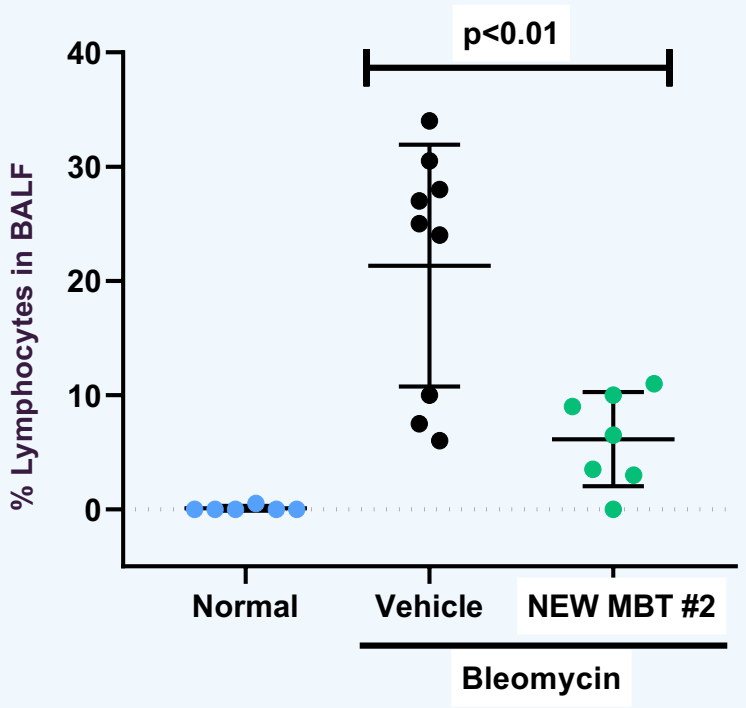
Red arrows – fibrous knots

V – blood vessel

Anti-fibrotic Effect of MBT #2 in Mouse IPF Model

- MBT #2 protects mouse lung from bleomycin-induced fibrosis
- Decreased fibrosis measured by histopathology (Ashcroft Score) after 21 days
- Comparable effect size to late stage anti-fibrotic in the same model (Medicinova)
- Confirmatory studies and further optimization ongoing/planned
- Potential for efficacy in other fibrosis models e.g., chronic kidney disease and heart failure

Fibrotic Diseases: Anti-inflammatory Effect in Mouse IPF Model



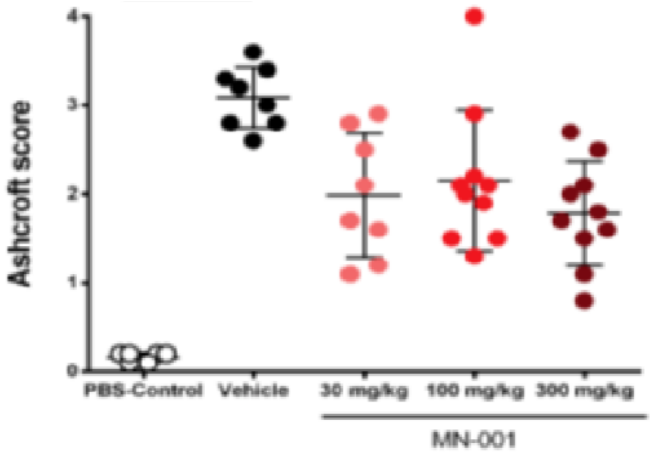
Anti-inflammatory Effect of MBT #2 in Mouse IPF Model

- Significant reduction in inflammatory cells in lung based on bronchoalveolar lavage
- Anti-fibrotic effect could result from early anti-inflammatory effects in this prophylaxis model
- Next step - examine direct anti-fibrotic effect in a therapeutic model in BLM induced fibrosis
- Peptide will be dosed 7 days after BLM injury to allow inflammatory stage to subside
- Potential utility for other inflammatory conditions

[1]CohBar Preliminary Data on File

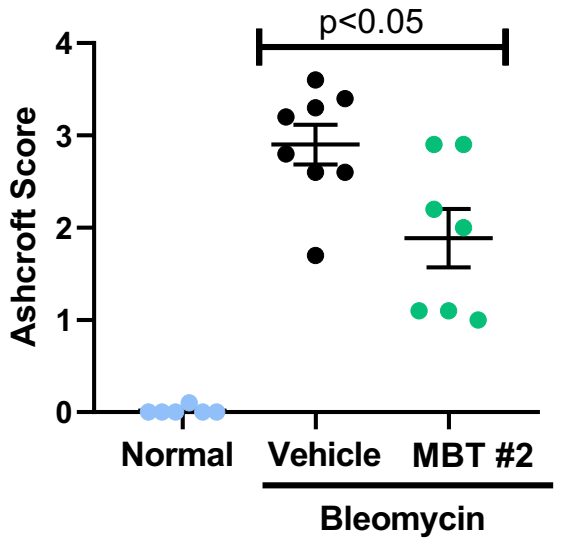
Fibrotic Diseases: Antifibrotic activity vs published data

Published Data for MN-001 (tipelukast) in the Same Bleomycin-Induced Fibrosis Model at 3 weeks[1]



- MN-001 (Medicinova):
- In Phase 2 clinical trial for IPF and entering P2 for NASH
- Down-regulates fibrotic genes: LOXL2, Collagen Type 1 and TIMP-1

Preliminary Data for MBT #2

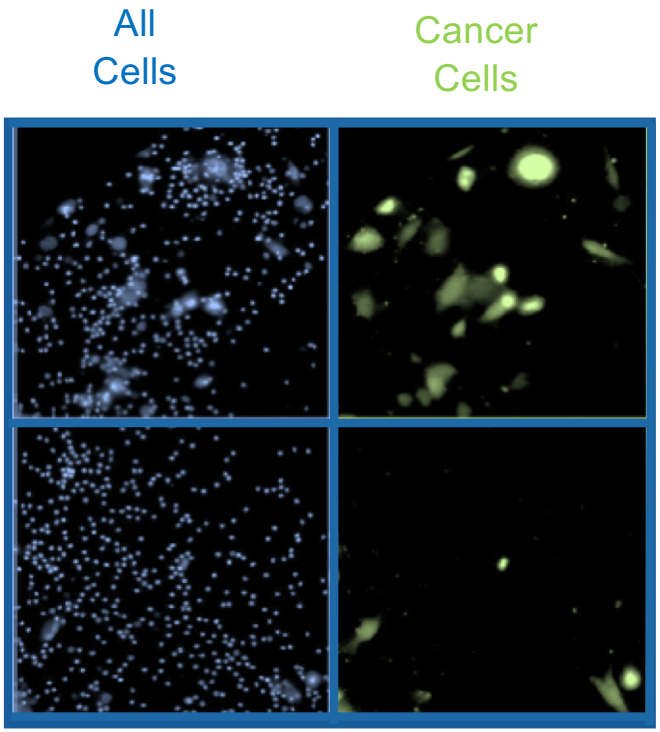


- MBT #2: Down-regulates expression of fibrotic proteins: Collagen Type 1, Collagen Type III, and αSMA

[1]Source: Matsuda K. ICLAF Meeting, 2017.

[2]CohBar Preliminary Data on File

Cancer: Peptide analogs enhance the killing of cancer cells by human blood cells, potential for use in immune system oncology

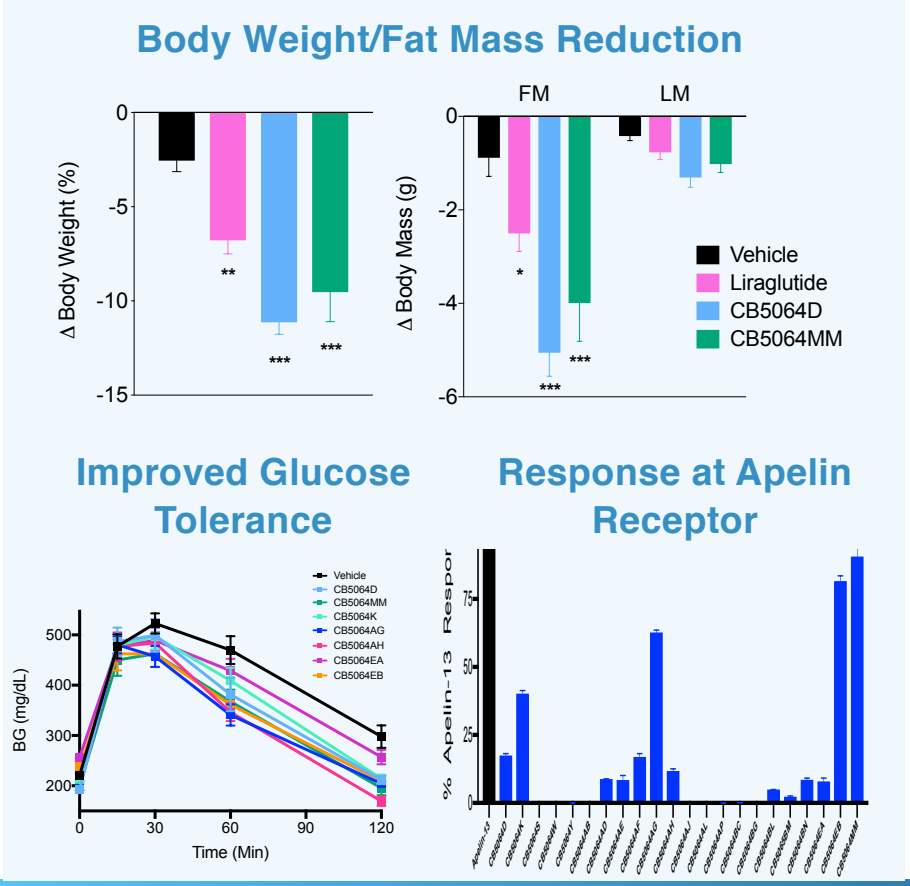


- MBT #3 produced a highly significant, $p<0.001$, reduction in number of cancer cells in the presence of PBMCs. No effect without PBMCs.
- Co-culture of peripheral blood mononuclear cells (PBMCs) -T-cells, B-cells and NK-cells - with human cancer cells -SKMEL28 melanoma cells
- Cells stimulated with LPS to induce immune response. Treated with peptide or vehicle control for 48 hours and cells quantified by selective staining and image analysis
- Additional MBTs show efficacy in this model

Representative images – Phenovista Biosciences

[1]CohBar Preliminary Data on File

Type 2 Diabetes: CB5064 analogs improve body weight and glucose tolerance in DIO mice and demonstrate selective agonism at the apelin receptor



- CB5064 is a new mitochondrial derived peptide discovered by CohBar
- CB5064 analogs produced body weight and fat mass reduction in DIO mice (dosed once daily for 10 days)
- CB5064 analogs improved glucose tolerance in DIO mice after 10 days of dosing
- MOA - selective interaction with the apelin receptor
- Apelin is a natural hormone widely expressed in adipose tissue, heart, lung, kidney, liver, brain, etc.
- Apelin plays a key role in energy metabolism, cardiovascular function, fluid homeostasis, angiogenesis, and in diabetic complications

Source: Grindstaff KG et al. ADA Poster Presentation LB-296, June 9, 2019.

Plan 2019/2020

- **Clinical:** Advancing CB4211 through phase 1a/1b with a data readout anticipated in Q2 or Q3 2020
- **Preclinical Peptides:** Utilizing our platform technology to further advance our preclinical assets toward candidate selection
- **Partnering:** Expanding partnering activities around increasing pharma interest in mitochondrial medicine, and CohBar's technology and approach to novel therapeutics
- **Financing:** Evaluating funding alternatives to support our clinical program and accelerate our preclinical projects
- **Investor Relations:** Broadening our institutional investor base, presenting at conferences and securing quality research coverage
- **Intellectual Property:** Continue growing our IP portfolio to maintain our leadership in mitochondrial based therapeutics and mitochondrial medicine

CohBar: Mitochondria Based Therapeutics designed to treat chronic diseases and to increase healthy lifespan

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