KOL Call and Webcast

Mitochondria – A Source for Novel Therapeutics

May 2, 2019
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. 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 our capital resources and future financing plans; statements regarding anticipated therapeutic properties and potential of our MBTs or the properties, potential and effects of newly-discovered mitochondrial-derived peptides; 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: unanticipated difficulties or unfavorable results encountered in our research and development programs or in clinical studies, including but not limited to the possibility that the Phase 1 clinical trial will remain suspended for longer than anticipated or may not be resumed; CohBar’s 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; our ability to raise additional capital when necessary to continue our operations; our ability to recruit and retain key scientific personnel; and our ability to establish and maintain partnerships with 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.
Dr. David Sinclair – The Role of Mitochondria in Health and Aging
Mitochondria: Key Facts

- Mitochondria are found in most cells
- Have their own genome, maternally inherited
- Numbers vary from 100s to 1000s
- Thought to contain only 37 genes including those needed to make energy
- Point mutations or deletions in mtDNA have been associated with a large spectrum of diseases
- Symptoms such as muscle weakness, cardiomyopathy, optic nerve atrophy, retinal dystrophy, impaired hearing and type 2 diabetes
Mitochondria make energy essential for life

https://www.mda.org/disease/mitochondrial-myopathies/causes-inheritance
Where did they come from?

An ancient symbiont living within

- Produce cellular energy
- Control cell survival
- Signal to the rest of the body
- A cause of aging
Mitochondria have their own genome

https://en.wikipedia.org/wiki/Mitochondrial_DNA

https://www.researchgate.net/fig2_318420329
Inside Mitochondria

https://en.wikipedia.org/wiki/Mitochondrial_DNA
Mitochondria come in all shapes and sizes

http://www.nature.com/nature/journal/v491/n7424/full/nature11707.html?WT.ec_id=NATURE-20121115
How do mitochondria work?

https://www.nature.com/scitable/topicpage/mtdna-and-mitochondrial-diseases-903#
Mitochondrial Mutations and Disease

- Hypothalamus and pituitary gland
  - Growth hormone deficiency
  - Hypogonadotropic hypogonadism
  - Secondary adrenal insufficiency
  - Central hypothyroidism

- Parathyroid glands
  - Primary hypoparathyroidism

- Genetic defects
  - Large-scale mtDNA deletion, MT-7L1

- Thyroid gland
  - Primary hypothyroidism

- Genetic defects
  - MT-7L1, PTTH2

- Adrenal glands
  - Primary adrenal insufficiency

- Genetic defects
  - STAR, CYP11A1, HSD3B2, MT-1K, large-scale mtDNA deletion, MIP57, OGISL1, MOF5F7, CTER, NNI, TXNRD2

- Pancreas
  - Diabetes mellitus
  - Exocrine pancreatic insufficiency

- Genetic defects
  - MT-7L1, MT-1K, MT-TS2, MT-TE, large-scale mtDNA deletion, POLG, RN10B, CPA1, MPV17

- Ovaries
  - Premature ovarian failure
  - Perinatal syndrome

- Genetic defects
  - POLG, C12orf65, LRPPRC, CLPB, COX10, CLPB, AMRS2, C10orf72, HARS2, LARS2

- Testes
  - Hypogonadotropic hypogonadism

- Genetic defects
  - STAR, CYP11A1, HSD3B2, POLG, C10orf52, TXMP, LRPPRC

- Skeletal muscle
  - Weakness
  - Fatigue
  - Myopathy
  - Neuropathy

- Heart
  - Conduction disorder
  - Wolff-Parkinson-White syndrome
  - Cardiomyopathy

- Eye
  - Optic neuropathy
  - Ophthalmoplegia
  - Retinopathy

- Liver
  - Hepatopathy

- Kidney
  - Fanconi’s syndrome
  - Glomerulopathy

- Pancreas
  - Diabetes mellitus

- Blood
  - Pearson’s syndrome

- Inner ear
  - Sudden sensorineural hearing loss

- Nuclear DNA

- Seizures

- Mitochondrial DNA

- Brain
  - Seizures
  - Myoclonus
  - Ataxia
  - Stroke
  - Dementia
  - Migraine

- Colorectal cancer
  - Pseudo-obstruction

- Nature Reviews | Endocrinology

https://universe-review.ca/10-05-mito02.gif
Mitochondrial mutations can lead to diabetes
Mitochondria decline is a cause of aging

https://www.nature.com/articles/ncb0905-853
Mitochondria talk to other cells when stressed
Mitochondrial-derived peptides - A New Therapeutic Paradigm

https://www.nature.com/articles/ncb0905-853
Founder of the field – Professor Hassy Cohen

https://www.nature.com/articles/ncb0905-853
Dr. Pinchas Cohen – The Discovery of Mitochondrial Peptides
A Novel Platform for Drug Development for Diseases of Aging
Signals Encoded Within the Mitochondrial Genome

Mitochondrial Communication

MDPs
Humanin
SHLP 1-6
MOTS-c

Protection
Metabolism
Cell function

Mitochondrial signals to the nucleus and to distant cells
Mitochondrial Derived Peptides (MDPs)

First Discovered:
Humanin (Early 2000s)

PNAS Proceedings of the National Academy of Sciences of the United States of America
Humanin: The First Mitochondrial Peptide

Cloned by several groups using various methods

- **Conserved Sequence in mammals**
- Produced as a polyadenylated mRNA
- Smaller and distinct from the rRNA
- Translated in the cytoplasm and secreted
- Highly conserved
- Present in Brain, Testes, CSF, Plasma

- **Cytoprotective / Metabolo-protective**
Multiple Open Reading Frames within the Mitochondrial Genome

Newly developed roadmap of peptides
Biology of Humanin

Humanin levels are higher in familial exceptional longevity

Humanin levels are related to age

P<0.03
Naturally occurring mitochondrial-derived peptides are age-dependent regulators of apoptosis, insulin sensitivity, and inflammatory markers.

The Discovery of MOTS-c

No reduction in muscle

**The Mitochondrial-Derived Peptide MOTS-c Promotes Metabolic Homeostasis and Reduces Obesity and Insulin Resistance**

Changhan Lee, Jennifer Zeng, Brian G. Drew, Tamer Sallam, Alejandro Martin-Montalvo, Junxiang Wan, Su-Jeong Kim, Hemal Mehta, Andrea L. Hovenner, Rafael de Cabo, and Pinchas Cohen

*Leonard Davis School of Gerontology, University of Southern California, Los Angeles, California 90089, USA*
Characterization of MOTS-c

An age-dependent 12S-derived insulin-sensitizing mito-peptide peptide that activates AMPK and rejuvenates muscle metabolism.
MOTS-c is induced by exercise and is a marker of training.

**Effects of Exercise on plasma MOTS-c**

- **Effect of 3-months training**
- **Effect of acute exercise**

![Graph showing the effects of exercise on plasma MOTS-c levels.](image)
MOTS-c is related to Exercise performance

Treated with 15-mg/kg IP MOTS-c daily for 2-weeks

Effect of 2-months MOTS-C treatment on Myostatin levels

In submission (Lee lab and Cohen lab)
MOTS-c K14Q - A Genetic variation in Asians

mt1382A/C SNP found only in 7-9% of North-Asians causes a conformational change
The MOTS-c variation increases risk of diabetes in Japanese Males

No effect of the SNP on risk of diabetes in females
K14Q MOTS-c is less effective *in vivo* and *in vitro*

**Weight Loss (males)**

![Graph showing weight loss (males)]

**Insulin sensitization**

![Graph showing insulin sensitization](image)

**Glucose Tolerance**

![Graph showing glucose tolerance](image)

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*Kim et al.* Submitted to JCI; 2019
Multiple labs are studying the beneficial functions of MOTS-c peptide.
### MOTS-c alleviates various pathological conditions

<table>
<thead>
<tr>
<th>Pathological conditions</th>
<th>MOTS-c action</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aging-associated insulin resistance</td>
<td>Increases glucose uptake in skeletal muscles in aged mice.</td>
<td>[1]</td>
</tr>
<tr>
<td>High-fat diet (HFD)- insulin resistance</td>
<td>Increases insulin sensitivity in HFD-fed mice.</td>
<td>[1]</td>
</tr>
<tr>
<td>Obesity-associated insulin resistance</td>
<td>Increases GLUT-4 expression in skeletal muscle in HFD-fed mice.</td>
<td>[1]</td>
</tr>
<tr>
<td>Obesity-associated insulin resistance</td>
<td>Plasma MOTS-c levels are lower in obese male children and adolescents and negatively correlated with markers of insulin resistance and obesity.</td>
<td>[21,22]</td>
</tr>
<tr>
<td>Nonalcoholic steatohepatitis</td>
<td>Decreases hepatic fat accumulation in HFD-fed mice. MOTS-c analogues prevent NASH in a STAM model</td>
<td>[1,18]</td>
</tr>
<tr>
<td>Endothelial dysfunction</td>
<td>Improves endothelial function in rats. Plasma MOTS-c levels are lower in human subjects with impaired coronary endothelial function.</td>
<td>[23]</td>
</tr>
<tr>
<td>Menopause-associated conditions</td>
<td>Prevents ovariectomy-induced obesity and insulin resistance.</td>
<td>[19]</td>
</tr>
<tr>
<td>Menopause-associated conditions</td>
<td>Alleviates ovariectomy-induced osteoporosis.</td>
<td>[19,20]</td>
</tr>
<tr>
<td>Osteoporosis/Osteopenia</td>
<td>Alleviates bone loss in ovariectomy-induced osteoporosis via AMPK. Promotes rat bone mesenchymal stem cells differentiation to osteoblasts via TGF-β pathway.</td>
<td>[19,20]</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Improves survival in mice during MRSA infection. Enhances bactericidal capacity of macrophages in MRSA-infected mice.</td>
<td>[33]</td>
</tr>
</tbody>
</table>

Kim et al. Journal of Molecular Medicine; 2019
Evidence of MDP efficacy in pre-clinical models of a broad range of diseases

**MOTS-c:**
- NASH
- Obesity
- Diabetes
- Frailty/Sarcopenia
- Osteoporosis
- Infections

**Additional MDPs:**
- Cancer
- Alzheimer’s
- Heart Disease
- Eye Disease
Dr. Kenneth Cundy – Breakthrough Science to Increase Healthy Lifespan
CohBar’s novel improved MDP analogs for treating age-related diseases
CohBar Technology: Novel Improved Analogs of MDPs

<table>
<thead>
<tr>
<th>Mitochondria (Powerhouse of the Cell)</th>
<th>Mitochondria Derived Peptide (“MDP”)</th>
<th>Optimized Peptide (“Analog”)</th>
<th>Mitochondria Based Therapeutic (“MBT”)</th>
</tr>
</thead>
<tbody>
<tr>
<td>![Mitochondria Image]</td>
<td>![Peptide Image]</td>
<td>![Peptide Image]</td>
<td>![Therapeutic Image]</td>
</tr>
</tbody>
</table>

**Identify**
- Identify/characterize peptides with biological activity encoded within mitochondria
- Evaluate therapeutic potential across disease models
- File for Intellectual Property (“Own the Space”)

**Optimize**
- Optimize structures for potency and drug like properties
- Proprietary assays, validated 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

GREEN = CohBar Proprietary Technology
## CohBar R&D Programs for MBTs in Age-Related Diseases

<table>
<thead>
<tr>
<th>Target Indication</th>
<th>Preclinical Discovery and Optimization</th>
<th>IND Enabling Activities</th>
<th>Phase 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>CB4211 (MOTS-c Analog)</td>
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<tr>
<td>NASH</td>
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<tr>
<td>Obesity</td>
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<tr>
<td><strong>New Peptides</strong></td>
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<tr>
<td>NASH and Obesity</td>
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<tr>
<td>Type 2 Diabetes</td>
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<tr>
<td>Cancer</td>
<td></td>
<td></td>
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<tr>
<td>Other Age-Related Diseases</td>
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</tbody>
</table>
CB4211: Lead MBT Program for NASH and Obesity

*Optimized Analog of the Mitochondrial Peptide MOTS-c*

- CB4211 is being developed for the treatment of NASH and Obesity
- Novel Mechanism of Action - regulation of fat cells (adipocytes) leading to reduction of liver fat
- 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
- Phase 1a/1b trial initiated with an activity readout relevant to NASH and obesity
- Intellectual property coverage filed (PCT in 2017)
CB4211: MOTS-c Analog Effective in Mouse NASH Model

NAS is a composite score of liver fat (steatosis), liver cell damage (hepatocyte ballooning) and inflammation.

STAM® mice – use streptozotocin to induce NASH symptoms

CB4211 treatment reduced NAS by 33% after 3 weeks (p<0.01)

Compares favorably with published data:

- Intercept’s Obeticholic acid (FXR agonist): 23% decrease
- Allergan’s Cenicriviroc® (CCR2/5 inhibitor): 25 to 30% decrease
- CB4211 significantly reduced plasma ALT (marker of liver damage) and liver triglyceride (fat) levels


Hambruch E, et al. NASH 2010, Tokyo, Japan.

Friedman SL, et al. HEP DART 2013, Hawaii..
CB4211: Synergy with GLP-1 Agonist in NASH Model

Synergistic Effect of CB4211 and Liraglutide on Body Weight, Fat Mass and Steatosis

(21 Day Diet Induced Obese Mouse Study)

Change in Body Weight at Day 21

<table>
<thead>
<tr>
<th>Condition</th>
<th>% Change in BW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vehicle BID SC</td>
<td>-10</td>
</tr>
<tr>
<td>Liraglutide QD IP</td>
<td>-5</td>
</tr>
<tr>
<td>CB4211A 5 QD SC + Lirag</td>
<td>-15</td>
</tr>
</tbody>
</table>

Change in Fat Mass at Day 21

<table>
<thead>
<tr>
<th>Condition</th>
<th>Change in Fat Mass (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vehicle BID SC</td>
<td>-4</td>
</tr>
<tr>
<td>Liraglutide QD IP</td>
<td>-2</td>
</tr>
<tr>
<td>CB4211A 5 QD SC + Lirag</td>
<td>-3</td>
</tr>
</tbody>
</table>

Liver Fat Deposits at Day 21

- Liraglutide
- Liraglutide + CB4211 5 mg/kg QD SC

† Cundy KC, et al. AASLD 2017, Washington, DC.
CB4211: Synergy with PPARγ Agonist in Diabetes Model

Synergistic Effect of CB4211 and Pioglitazone on Glucose Tolerance
(14 Day Zucker Diabetic Rat Model of Type 2 Diabetes)

Glucose Tolerance Test – Blood Glucose Levels

Area Under GTT Curve

* CohBar internal data
CB4211: Novel Mechanism of Action Relevant to NASH

**Targeting Free Fatty Acid Release from Fat Cells**

*Inhibition/Regulation of Lipolysis*

- NALFD: excess free fatty acid released from abdominal fat cells by lipolysis flows directly to the liver
- Excess fatty acid in liver leads to NASH: liver fat deposits, inflammation, fibrosis, cirrhosis, and ultimately liver cancer
- Inhibiting lipolysis reduces fatty acid release

**Molecular Mechanism – Enhances Insulin Signaling**

*Regulation of Insulin Signaling*

- Insulin receptor and insulin signaling play a central role in metabolic regulation
- CB4211 enhances the action of insulin in vitro:
  - Inhibits lipolysis in fat cells (adipocytes)
  - Decreases free fatty acid release to liver
  - Decreases glucose production by liver cells
  - Decreases glucose consumption by muscle cells.
- Molecular mechanism of action presented at ADA in June 2018: **CB4211 is a Potential Treatment for Metabolic Diseases with Novel Mechanism of Action: Sensitization of the Insulin Receptor**
- Further evidence that some MDP’s are important regulators of key metabolic pathways in the body

Source: Nutrients 2015, 7, 9453–9474
CB4211: Phase 1a/1b Clinical Study Design

**Conventional Phase 1a SAD/MAD in healthy normal subjects** - assess safety, tolerability, PK, selection of maximum dose, cardiovascular safety assessment (randomized, double-blind, placebo controlled)

**Placebo controlled Phase 1b arm in obese subjects with NAFLD** – exploratory activity study
- 4-week treatment period with once daily subcutaneous dosing at maximum well tolerated dose

**Activity assessed by change in liver fat (MRI-PDFF), body weight, and biomarkers**
Data on changes in liver fat (foundational event of NASH), body weight (primary endpoint for obesity), and biomarkers relevant to NASH, obesity, and metabolic disease.

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**Single Ascending Dose**

**Multiple Ascending Dose**

**Phase 1b (NAFLD and Obese)**

- Clinical phase I study ongoing
- Dosing currently suspended pending FDA input on protocol amendment
- Top-line activity data from Phase 1a/1b readout relevant to NASH and obesity
**New Peptides: Type 2 Diabetes – Key Receptor Identified**

**Improved Glucose Tolerance in Diet Induced Obese (DIO) Mice**

<table>
<thead>
<tr>
<th>Area Under GTT Curve</th>
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</thead>
<tbody>
<tr>
<td>AUC (mg/dL/min)</td>
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<tr>
<td></td>
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<tr>
<td>30000</td>
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<tr>
<td>20000</td>
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<tr>
<td>10000</td>
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<td>0</td>
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</table>

**Day 10**

- **Vehicle**
- **Liraglutide**
- **New MBT#1**

New family of peptide analogs with potential for T2DM
- Improved glucose tolerance in DIO mice
- Optimizing potency and drug-like qualities
- Mechanism of action explored by receptor screening
- Recent breakthrough – activity discovered at a key cell surface receptor
  - Plays a key role in several age-related diseases
  - Potential for entirely new class of agents for this receptor
  - Abstract accepted for ADA Meeting (June 2019)
  - Further details embargoed until data are presented
New Peptides: In Vitro Evidence of Cancer Effects

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

<table>
<thead>
<tr>
<th>20 Different Human Tumor Cell Types</th>
<th>In Vitro Effects on Cancer Cell Growth</th>
</tr>
</thead>
<tbody>
<tr>
<td>A B C D E F G H I J K L M N O P Q R S T</td>
<td>• Systematic evaluation of human tumor cell proliferation</td>
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<tr>
<td>1 8 4 5 8 8 4 6 5 5 7 6 10 6 5 7 7 8 8 9 7</td>
<td>• 20 different human tumor cell types screened against 20 CohBar peptides</td>
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<tr>
<td>2 9 5 8 8 8 8 7 7 8 8 7 8 8 6 7 8 8 3 8 8</td>
<td>• Evidence of effects across a broad range of tumor types</td>
</tr>
<tr>
<td>3 8 6 8 3 6 8 8 4 8 5 7 6 7 5 8 8 7 8 8 6</td>
<td>• Currently evaluating CohBar peptides in immuno-oncology setting</td>
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<td>17 7 7 5 4 6 8 5 7 6 6 3 5 4 5 6 8 8 8 8</td>
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<td>18 7 7 6 7 6 7 5 6 5 6 3 6 6 6 7 8 8 8 8</td>
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<td>19 9 7 7 11 8 8 6 5 8 7 6 5 8 8 8 8 8 8 8</td>
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<tr>
<td>20 10 8 7 5 6 8 6 6 8 7 6 8 4 8 9 9 8 8 8</td>
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</tbody>
</table>

Lower number/more red = less proliferation

(Cultured Human Tumor Cells)

In Vitro Effects on Cancer Cell Growth:
- 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
- Currently evaluating CohBar peptides in immuno-oncology setting

(New peptides not yet optimized)
New Peptides: Evidence of Antifibrotic Effects in Lung Cells

Decreased Expression of Fibrosis Biomarkers in Cultured Lung Cells

New peptide analog with potential antifibrotic effects:
- Co-culture of lung epithelial cells and lung fibroblasts (Eurofins)
- Treatment with peptide or vehicle for 3 days
- Protein biomarker expression determined by specific validated assays
- Result compared to vehicle treated control
- Significant reduction in fibrosis biomarkers:
  - Collagen Type I, Collagen Type III and αSMA (alpha-smooth muscle actin)

1CohBar Preliminary Data on File
New MBT #2: Effective in Mouse Model of Pulmonary Fibrosis

**Decreased Lung Fibrosis in Bleomycin-Induced Fibrosis Mouse Model**

- **New Peptide Significantly Reduced Ashcroft Score**
  - Ashcroft Score - objective measure of lung tissue damage and fibrotic changes by microscopy
  - Mice treated with intratracheal bleomycin to induce lung fibrosis
  - Vehicle treatment alone did not prevent fibrosis
  - Preventive treatment with New MBT #2 led to significant reduction in Ashcroft Score @ 3 weeks
  - New MBT #2 also decreased lung collagen deposits (hydroxyproline biomarker)
  - In vivo translation of the in vitro antifibrotic effects

![Graph showing Ashcroft Score comparison between Normal, Vehicle, and New MBT #2 treated groups with p<0.05 significance.](image-url)
New MBT #2: In Vivo Evidence of Effects on Fibrosis

New MBT #2 Improved Lung Histopathology in Mouse Pulmonary Fibrosis Model

- **Lung Tissue of Normal Mice (Day 21)**
  - ID: 102
  - Normal Healthy Lung Tissue

- **Mice Exposed to Bleomycin and Treated with Vehicle (Day 21)**
  - ID: 205
  - Damaged Lung Structure And Fibrous Changes
  - V - blood vessel; red arrows – fibrous knots

- **Mice Exposed to Bleomycin and Treated with New MBT #2 (Day 21)**
  - ID: 404
  - Evidence of Protection from Bleomycin Induced Fibrosis

(Representative Slides at 100x Magnification, V-blood vessel; red arrows – fibrous knots)

\(^1\)CohBar Preliminary Data on File
CohBar Q1 2019 Investor Call Scheduled for May 7, 2019

CohBar to Release First Quarter 2019 Financial Results and Provide Business Update

- The company will release its first quarter 2019 financial results after the market closes on Tuesday, May 7, 2019
- Management will host a conference call with a slide presentation at 5:00 p.m. ET (2:00 p.m. PT)
- For details for the Conference Call and Slide Presentation please visit www.cohbar.com/news-media/events
Questions?