Our goal is to slow, stop and even prevent the onset and progression of degenerative brain diseases, with particular focus on Alzheimer’s and Parkinson’s Disease.
Forward looking statements

This presentation contains forward-looking statements made within the meaning of the Private Securities Litigation Reform Act of 1995 by Cantabio Pharmaceuticals, Inc. and its representatives. These statements can be identified by introductory words such as “expects,” “plans,” “intends,” “believes,” “will,” “estimates,” “forecasts,” “projects” or words of similar meaning, and by the fact that they do not relate strictly to historical or current facts. Forward-looking statements frequently are used to discuss potential product applications, collaborations, product development activities, clinical studies, regulatory submissions and approvals, and similar operating matters. Many factors may cause actual results to differ from forward-looking statements, including inaccurate assumptions and a broad variety of risks and uncertainties, some of which are known and others of which are not. Known risks and uncertainties include those identified from time to time in the reports filed by Cantabio Pharmaceuticals, Inc. with the Securities and Exchange Commission, which should be considered together with any forward-looking statement. No forward-looking statement is a guarantee of future results or events, and one should avoid placing undue reliance on such statements.

Cantabio Pharmaceuticals, Inc. undertakes no obligation to update publicly any forward-looking statements, whether as a result of new information, future events or otherwise. Cantabio Pharmaceuticals, Inc. cannot be sure when or if it will be permitted by regulatory agencies to undertake clinical trials or to commence any particular phase of clinical trials. Because of this, statements regarding the expected timing of clinical trials cannot be regarded as actual predictions of when Cantabio Pharmaceuticals, Inc. will obtain regulatory approval for any “phase” of clinical trials. We also cannot be sure of the clinical outcome for efficacy or safety of our compounds. Potential investors should refer to the risk factors in our reports filed on Edgar.
Cantabio’s therapeutic strategy – a different approach

• Development of novel therapeutic candidates, which aim to eliminate the root causes of AD & PD: protein aggregation, oxidative and glyoxal stress caused toxicity.

• Pursuing a portfolio of related targets: one novel and two highly validated targets that have been difficult to target

• Applying innovative drug discovery and therapeutic technologies to develop small molecule pharmacological chaperones

• Cantabio’s therapeutic candidates have novel mechanism of action – stabilizing the functional form of the target

• Cantabio’s therapeutic candidates are potentially preventative and are aimed at stopping disease progression
Market opportunity

• Dementia
  • 46.8 million people globally suffer from dementia, rising to 74.7 million by 2030
  • Estimated global cost: $818bn, rising to $2 trillion by 2030
  • 10 million new cases in 2015

• Alzheimer’s disease (AD)
  • 6th leading cause of death in the USA
  • Estimated cost: $236 billion, $605 billion globally
  • 5.4 million Americans are currently living with the disease, with around 44 million diagnosed worldwide

• Parkinson’s disease (PD)
  • Affects 7 to 10 million people worldwide
  • Estimated cost of nearly $25 billion per year
  • 60,000 Americans are diagnosed with PD each year

• Current therapies only temporarily treat symptoms, despite this the dementia drug market is worth U.S. $6.5 billion / year

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2 Michael J Fox Foundation
3 Nature Reviews Drug Discovery Sept 2015
Protein aggregation is the pathological hallmark of diseases such as AD and PD

- **AD and PD**
  - Chronic, degenerative disorders
  - Gradual neuronal death
  - Result in loss of memory, cognition, motor function, behavioral changes and eventually death

- Both diseases are associated with proteins that misfold, aggregate and become toxic.
  - **AD**: Aβ peptide -> amyloid
  - Tau protein -> NFT
  - **PD**: α-synuclein -> Lewy bodies

AD-caused aggregation of Aβ and Tau in the brain detected by PET imaging.
Onset and progression of AD & PD linked to protein aggregation and oxidative stress

**Protein aggregation** causes brain cell death through
- Oxidative stress generation
- Membrane disruption
- Other unknown complex mechanisms

**High oxidative stress** causes brain cell death through
- Amino acid and DNA damage
- Protein loss of function
- Mitochondrial dysfunction
- Altered survival signaling
- Protein aggregation

**High glyoxal levels** causes brain cell death through
- Amino acid and DNA modification
- Protein loss of function
- Mitochondrial dysfunction
- Oxidative stress generation
- Protein aggregation

Cantabio’s therapeutic candidates aim to prevent, reduce and stop protein aggregation, oxidative and glyoxal stress.
CANTABio’s therapeutic technology: small molecule pharmacological chaperones

- **Chaperones act to stabilize the functional form of a protein against misfolding**
- **Support and enhance the body’s existing biological processes**
- **Can be orally administered**
CANTABio’s small molecule drug discovery approach

Identify novel small molecule binders

Mechanism of action studies

In vivo validation

Fragment and Lead-like Molecule Library

Biophysics based high throughput screens

Cell and drosophila based neuro-degeneration models

X-Ray / NMR

PK/PD, BBB permeability

Functional CNS-like Hits

Structure-based design

Medicinal Chemistry

Lead Series

In vivo neurodegenerative models

Toxicity models

IND
CANTABio’s therapeutic pipeline

<table>
<thead>
<tr>
<th>Therapeutic Program</th>
<th>Indication</th>
<th>Pre-clinical</th>
<th>Phase I</th>
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<tbody>
<tr>
<td>DJ-1 small molecule pharmacological chaperone</td>
<td>PD</td>
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<tr>
<td>Cell penetrant engineered DJ-1</td>
<td>PD</td>
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<tr>
<td>Tau small molecule pharmacological chaperone</td>
<td>AD</td>
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<tr>
<td>Aβ small molecule pharmacological chaperone</td>
<td>AD</td>
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</table>

- Cantabio is focused on delivering candidates to clinical trials
- Diversity of programs helps to reduce overall risk
- Separate but potentially complementary approaches

Cantabio’s therapeutic candidates are potentially preventative and are aimed at stopping disease progression
DJ-1 protein: a defensive protein protecting from oxidative stress, glycolation protein aggregation

DJ-1 is genetically linked to familial PD, associated with sporadic PD and other neurodegenerative diseases

DJ-1 has been implicated in a number of diseases

- DJ-1 has multiple functions: REDOX-sensitive antioxidant and chaperone (inhibitor of alpha-synuclein protein aggregation), glyoxolase and deglyoxolas
- DJ-1 as a biomarker for PD: Oxidative state of DJ-1 and its concentration in biofluids is an indicator of PD
- To date there has been limited effort toward studying the biology and therapeutic targeting of this protein by the biotech and pharmaceutical industry
- Therapeutic targeting of DJ-1 could yield added mechanistic benefits: protection from oxidative stress and protein misfolding.

DJ-1 protein is an unique glyoxalase with the ability to metabolize MGO and remove advanced glycolated end-product from proteins and DNA.

DJ-1 neutralizes toxic methyl-glyoxal and repairs MGO caused glycation on proteins and DNA.

MGO caused disfunction in neurons in PD:
- Mitochondrial dysfunction
- Oxidative stress generation
- $\alpha$-synuclein aggregation and dysfunction
Targeting DJ-1 by pharmacological chaperones as a therapeutic approach

- DJ-1 is a dimeric (two-part) protein which helps protect cells from damage by reducing the effects of oxidation and protein aggregation.
- DJ-1 is fragile, and conditions of oxidative stress can cause DJ-1 to break down.
- With less DJ-1, oxidative stress and protein aggregation increases, destroying more DJ-1 in a vicious cycle of run-away oxidation which results in cell death.

Cantabio’s pharmacological chaperone molecules bind to DJ-1 protein in a manner which supports the structural integrity and function of DJ-1 reducing its sensitivity to break down under oxidative stress.

Cantabio’s novel candidate chaperone molecules have shown efficacy in cellular and animal models of PD.
Enhancement of DJ-1 protein levels in vivo as a therapeutic approach to Parkinson’s disease and related conditions

**DJ-1 loses its function in disease conditions**
Superfluous oxidation caused by high oxidative stress during disease conditions leads to DJ-1 inactivity as a result of structural destabilization and aggregation of the protein

**Our approach:**
To enhance functional DJ-1 protein levels in the brain through delivery of brain penetrant DJ-1 into central nervous system

- Deliver more DJ-1 directly to areas which need it to prevent cell damage and increase the cell’s natural defenses.
- Cantabio’s use of protein delivery technology helps administered DJ-1 cross the blood-brain barrier
Supplementing engineered DJ-1 into brain cells as a therapeutics strategy for PD and other neurodegenerative disease

Levels of DJ-1 can also be enhanced directly by administering DJ-1 directly to patients. More DJ-1 in the brain means more protection from oxidative stress and protein aggregation damage. However, due to the size of DJ-1 it is difficult to deliver it directly into cells.

Enabling drugs to cross the blood-brain barrier is a major challenge in CNS medicine.

Cantabio fused cell-penetrant molecule to DJ-1 which allows DJ-1 to cross the blood brain barrier and enter into brain cells. By this approach Cantabio’s cell penetrant DJ-1 therapeutic candidate has shown efficacy in cellular and in vivo models of PD.
Tau aggregation leads to loss of function and gain of toxic function

Tau binds to microtubules and stabilizes them in healthy neurons

Tau protein aggregates and detaches from microtubules in AD and related Tauopathies

1,2: Tau hyper-phosphorylates and detaches from the microtubule
3: microtubule breakdown
4: impaired axonal transport
5: Tau aggregation

1,2,: Tau proteins
3,: Microtubule
4,: Axonal transport with motor proteins
Pharmacological chaperones to prevent Tau protein and Aβ peptide aggregation for AD

The aggregation and amyloid formation of the Aβ peptide has been linked to the onset and progression of AD.

Cantabio has identified novel small molecule pharmacological chaperones that bind to monomeric Tau and or Aβ peptide and prevent aggregation of the proteins.

Tau targeting candidates have shown efficacy in cellular and in vivo models of AD.
## Competitive analysis of Cantabio’s therapeutic programs

<table>
<thead>
<tr>
<th>Therapeutic Program</th>
<th>Competitive Advantage</th>
<th>Intellectual Property</th>
</tr>
</thead>
<tbody>
<tr>
<td>DJ-1 targeting small molecule pharmacological chaperone for PD</td>
<td>• no direct competition&lt;br&gt;• global leader in DJ-1 drug discovery&lt;br&gt;• novel mechanism of action</td>
<td>• Cantabio has full IP rights&lt;br&gt;• patenting in progress</td>
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<td>Cell penetrant DJ-1 protein for PD</td>
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<td>• Cantabio has full IP rights&lt;br&gt;• patenting in progress</td>
</tr>
<tr>
<td>Tau targeting small molecule pharmacological chaperone for AD</td>
<td>• novel mechanism of action&lt;br&gt;• limited of direct competition&lt;br&gt;• lack of competition in clinical trials</td>
<td>• Cantabio has IP rights&lt;br&gt;• patent in progress</td>
</tr>
<tr>
<td>Aβ peptide targeting small molecule pharmacological chaperone for AD</td>
<td>• novel mechanism of action&lt;br&gt;• limited direct competition&lt;br&gt;• lack of competition in clinical trials</td>
<td>• Cantabio has full IP rights</td>
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</table>

- Cantabio is in a leading position with its *DJ-1 targeting small molecule pharmacological chaperone* and *cell penetrant DJ-1 protein* therapeutic programs.

- Small molecule chaperones penetrate neurons and are orally bioavailable and have economic benefit over biologic approaches.
Our R&D capabilities

- Modern lab facilities in new research facility in Budapest
- Multi disciplinary team
  - Biophysics, biochemistry
  - Computational drug design
  - Cellular assays
  - In vivo
- World leading in DJ-1 research
CANTABio R&D in the news and in scientific publications

Cantabio’s DJ-1 pharmacological chaperone program was featured as the cover article in BioCentury Innovations on October 20th 2016.


Cantabio’s Tau pharmacological chaperone program was featured in the magainze ‘The Scientist’ in October 20th 2017.


- The drug discovery approach taken for Cantabio’s Tau therapeutic program published

Identification of Small Molecule Inhibitors of Tau Aggregation by Targeting Monomeric Tau As a Potential Therapeutic Approach for Tauopathies

- A number of publication published recently by our team

- Periodic update on state of therapeutic programs at conferences:

The 13th International Conference on Alzheimer’s & Parkinson’s Diseases
Mechanisms, Clinical Strategies, and Promising Treatments of Neurodegenerative Diseases
March 29 - April 2, 2017 I Vienna, Austria
CANTABio’s published results highlights

**CB101** - first to show generally that DJ-1 is targetable by small molecules.
- Identified CNS drug-like compounds that protect cells, primary neurons, *in vivo* from oxidative stress and protein aggregation toxicity

**CB201** - demonstrated that CNS penetrating DJ-1 protects cells and primary neurons from oxidative stress and protein aggregation toxicity

**CB301** - pioneered approach targeting intrinsically disordered protein Tau with small molecules.
- Identified CNS drug-like compounds that inhibit Tau aggregation and related toxic effects in cells and primary neurons
CANTABio’s strategic partners

**University of Cambridge** – Cantabio’s Tau program for Alzheimer’s was developed at the University of Cambridge in conjunction with the Max Planck Institute

**NovAliX** – our industrial partner in a number of research applications spanning from high throughput screening to x-ray crystallography

**Purdue University** - work on our DJ-1 small molecule program has been validated by the Rochet Lab at Purdue University, funded by grants from the Michael J Fox Foundation

**University of Antioquia** – validation of the company’s DJ-1 protein-targeting small molecule pharmaceutical chaperone drug candidates in development for the treatment of Parkinson’s disease

**Hallym University** – further validation of cell penetrant DJ-1 protein for PD and other diseases
CANTABio research summary

• Novel approach to the development of therapeutics based on underlying biology and biophysics for AD & PD

• Focus on specific proteins that lose function or become toxic in brain cells due to oxidative and glyoxal stress and protein misfolding

• Pursue a therapeutic technology based on small molecule pharmacological chaperones, and protein brain delivery technology

• Strong pipeline with a portfolio of therapeutic projects with novel mechanism of action and disease modifying potential

• Aim to bring drug candidates to clinical trials within three years
CANTABio Leadership Teams

Management team

Gergely Toth PhD MBA, Founder & CEO
- UC at Berkeley
- University of Cambridge (MBA)
- Affiliate of University of Cambridge, Clinical Neurosciences
- Founded Gardedam Therapeutics
- In biopharma R&D and business since 2002

Simon Peace ACMA MBA, CFO
- University of Durham, University of Bradford
- University of Cambridge (MBA)
- CIMA qualified accountant
- M&A at GE Healthcare (>$1.5B of acquisitions)
- Finance and Tax Advisory Committee of the UK BioIndustry Association

Thomas Sawyer PhD MBA, COO
- University of Glasgow
- University of Cambridge (MBA)
- Entrepreneur, private equity CEO/CTO since 2002
- PE and VC funded private and public companies in US, EU and Africa

Scientific Advisory Board

Prof. Peter St. George Hyslop
- University of Cambridge, Wellcome Trust Principal Research Fellow
- University of Toronto, Director for Tanz Centre for Research in Neurodegenerative Diseases

Professor Jean-Christophe Rochet
- Purdue University, Department of Medicinal Chemistry and Pharmacology

Professor Lisa McConlogue
- UC, San Francisco, Department of Biochemistry and Biophysics,
- Gladstone Institute for Neurodegenerative Diseases

Prof. Franklin Aigbirhio
- University of Cambridge, Co-director of Wolfson Brain Imaging Centre

Dr. Manuel Buttini
- University of Luxembourg, Luxembourg Centre for Systems Biomedicine, Head Neuropathology Core Unit
<table>
<thead>
<tr>
<th>Date</th>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jun 2009</td>
<td>Research pipeline established by Gardedam Therapeutics Inc.</td>
</tr>
<tr>
<td>Jul 2010</td>
<td>Relationship with Purdue University established</td>
</tr>
<tr>
<td>Aug 2013</td>
<td>Further $75k grant from the Michael J. Fox Foundation</td>
</tr>
<tr>
<td>Apr 2015</td>
<td>Raised $1.1m in seed funding</td>
</tr>
<tr>
<td>Dec 2015</td>
<td>CANTABio merged with Gardedam Therapeutics and lists on OTCQB</td>
</tr>
<tr>
<td>Jan 2016</td>
<td>Multi-disciplinary research facility established in Budapest</td>
</tr>
<tr>
<td>Apr 2016</td>
<td>A-Beta license signed with NovAliX</td>
</tr>
<tr>
<td>Jan 2017</td>
<td>Raised $0.6m further financing</td>
</tr>
<tr>
<td>Aug 2009</td>
<td>Relationship with Graffinity/NovAliX established</td>
</tr>
<tr>
<td>Oct 2010</td>
<td>Relationship with Purdue University established</td>
</tr>
<tr>
<td>May 2015</td>
<td>Gardedam directors take control of a US OTC QB company to create CANTABio Pharmaceuticals Inc.</td>
</tr>
<tr>
<td>Sep 2015</td>
<td>First in house R&amp;D facility established</td>
</tr>
<tr>
<td>Jul 2016</td>
<td>DJ-1 license signed with Purdue University</td>
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<tr>
<td>Sep 2016</td>
<td>Tau license signed with University of Cambridge</td>
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</table>
CANTABio Contact:

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