MyMD® PHARMACEUTICALS

New approaches to treat autoimmune diseases and combat aging

Chris Chapman, M.D.

President, Director and Chief Medical Officer

April 2023

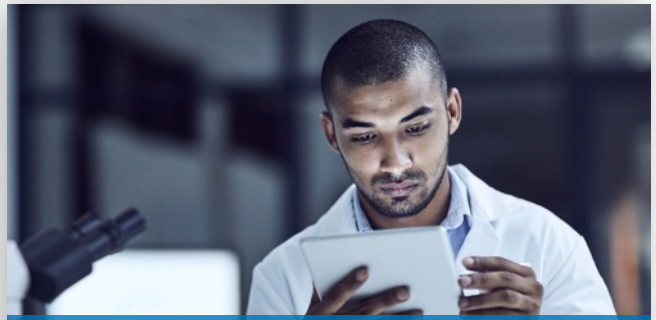
NASDAQ: MYMD

Forward-Looking Statement

This presentation may contain forward-looking statements. These forward-looking statements involve known and unknown risks, uncertainties and other factors which may cause actual results, performance or achievements to be materially different from any expected future results, performance, or achievements. Forward-looking statements speak only as of the date they are made and none of MyMD nor its affiliates assume any duty to update forward-looking statements. Words such as "anticipate," "believe," "could," "estimate," "expect," "may," "plan," "will," "would" and other similar expressions are intended to identify these forward-looking statements. Important factors that could cause actual results to differ materially from those indicated by such forward-looking statements include, without limitation: the timing of, and MyMD's ability to, obtain and maintain regulatory approvals for clinical trials of MyMD's pharmaceutical candidates; the timing and results of MyMD's planned clinical trials for its pharmaceutical candidates; the amount of funds MyMD requires for its pharmaceutical candidates; increased levels of competition; changes in political, economic or regulatory conditions generally and in the markets in which MyMD operates; MyMD's ability to retain and attract senior management and other key employees; MyMD's ability to quickly and effectively respond to new technological developments; MyMD's ability to protect its trade secrets or other proprietary rights, operate without infringing upon the proprietary rights of others and prevent others from infringing on MyMD's proprietary rights; and the impact of the COVID-19 pandemic or similar public health emergencies on MyMD's results of operations, business plan and the global economy. A discussion of these and other factors with respect to MyMD is set forth in the Company's Annual Report on Form 10-K for the year ended December 31, 2022, filed by MyMD on March 31, 2023 as may be supplemented or amended by the company's quarterly reports on Form 10-Q. Forward-looking statements speak only as of the date they are made and MyMD disclaims any intention or obligation to revise any forward-looking statements, whether as a result of new information, future events or otherwise.


Targeting large markets
with groundbreaking,
next-generation approaches
MyMD®
PHARMACEUTICALS

The MyMD Opportunity



NEXT-GENERATION APPROACH

Positive Data

MYMD-1® first oral, selective TNF-alpha inhibitor

SUPERA-CBD™ novel, potent synthetic cannabidiol (CBD) analog

PIPELINE WITH BROAD POTENTIAL

Two Candidates Targeting Large Markets

- Inflammatory/Autoimmune (RA)
- Sarcopenia/frailty (Aging)
- Neurologic (Epilepsy, chronic pain, anxiety)

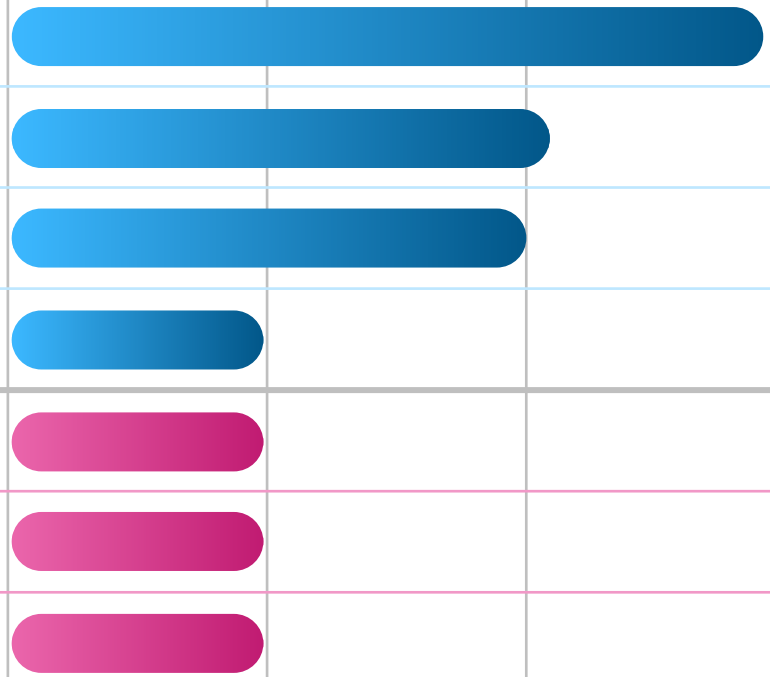
POSITIONED FOR SUCCESS

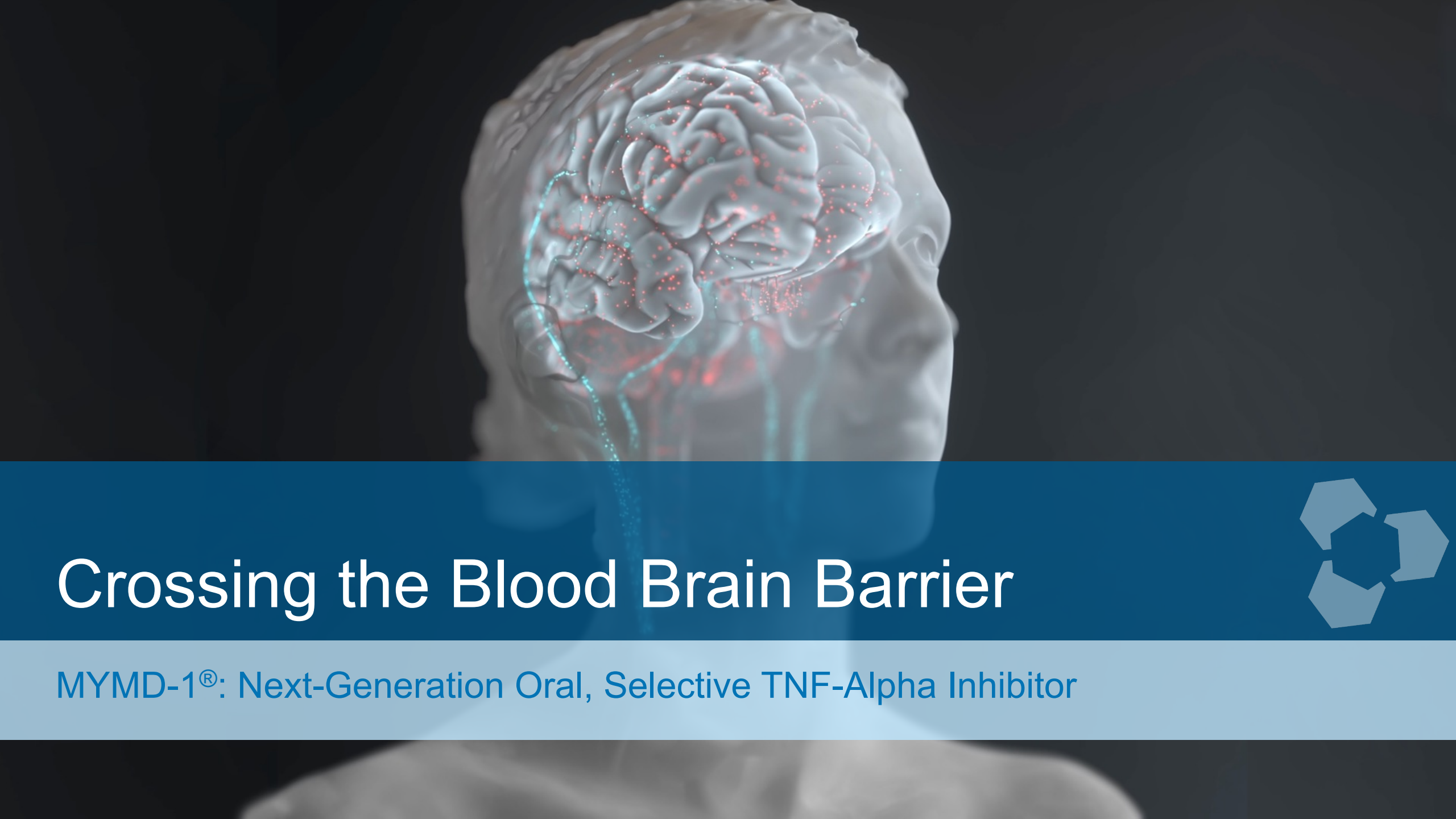
The Right Team to Execute

- High-value IP portfolio
- Experienced team
- Prominent advisors
- Reputable collaborations

Pipeline with Broad Potential

DRUG CANDIDATE	INDICATION	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	MARKET
MYMD-1® Immune Regulator	**Sarcopenia (Aging)**					
	Rheumatoid Arthritis					
	Hashimoto's Thyroiditis					
	Additional Programs					
Supera-CBD™ Synthetic CBD Analog	**Epilepsy**					
	Chronic Pain					
	Anxiety					





Crossing the Blood Brain Barrier

MYMD-1®: Next-Generation Oral, Selective TNF-Alpha Inhibitor

MYMD-1®: Next-Generation, Oral TNF-Alpha Inhibitor

TNF-alpha

An immune modulating cytokine

Dysregulation can lead to **prolonged inflammatory responses**

Known to drive disease in a range of autoimmune and inflammatory disorders

Current **TNF-alpha** inhibitors

Not all patients respond

Black box warnings around the risk of **serious infections** leading to hospitalization and death

Biologics administered by **infusion or injection**

Reducing pathological inflammation without completely blocking TNF-alpha activity thereby mitigating the risk of life-threatening infections

MYMD-1®

The potential to provide **meaningful therapeutic solutions** to patients not served by current TNF-alpha inhibitors

Improved ease of administration

Works by **preventing** the production of excess TNF-alpha in response to inflammatory signals

Better safety profile compared to currently approved anti-TNF therapies

Crosses the blood brain barrier **opening the potential to treat a wide range of disease**

Phase 2 study for potential novel therapy in RA

Potential to **slow the aging process** and **extend healthy lifespan**

Phase 2 studies for sarcopenia/frailty

Targeting Rheumatoid Arthritis with Potential Best-in-Class Approach

MYMD-1®: Next-Generation Oral, Selective TNF-alpha Inhibitor

Opportunity in RA

RA is an **autoimmune disorder** characterized by inflammation and bone erosion

It can occur at **any age**

Treatment is based on a **"trial-and-error"** approach

TNF-alpha plays a central role in RA

RA affects up to **14 million** people around the world

Studies estimate RA incidence may rise to **78 million** by 2040

1.3 M In the U.S.

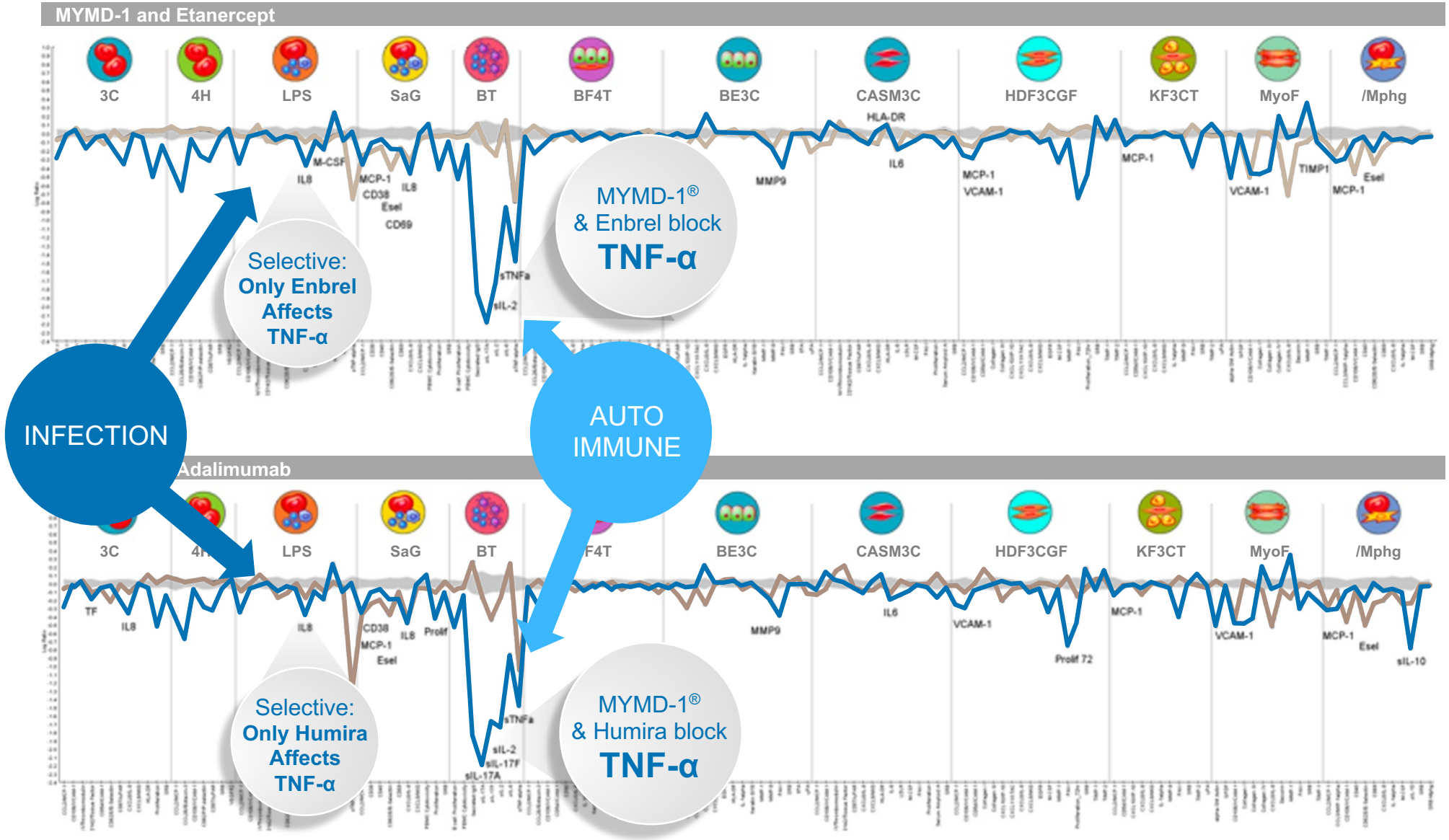
Current treatments that inhibit TNF-a have been available since 1998, all of which are administered via **injection or infusion**

References
https://link.springer.com/article/10.1007/s40744-020-00252-1, https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7085464/, https://www.arthritis.org/diseases/rheumatoid-arthritis
https://health.gov/healthypeople/about/workgroups/arthritis-workgroup, https://arthritis-research.biomedcentral.com/articles/10.1186/s13075-020-02354-1, www.yahoo.com/now/abbvies-humira-still-number-1-224014673.html

Invitro Studies Demonstrated Quantitative Measures of the Inhibition of TNFα, IL-6 and IL-17

MyMD PHARMACEUTICALS

- MYMD-1® 2000 µM
- Enbrel 1000 ng/ml
- Range of variants



- MYMD-1® 2000 µM
- Humira 3000 ng/ml
- Range of variants

A Naturally Occurring Novel Therapeutic and Oral Selective Inhibitor of TNF-α, MYMD-1® (Isomyosamine), Significantly Reduced the Inflammation and Disease Severity in Murine Model of Collagen Antibody Induced Arthritis

1 INTRODUCTION

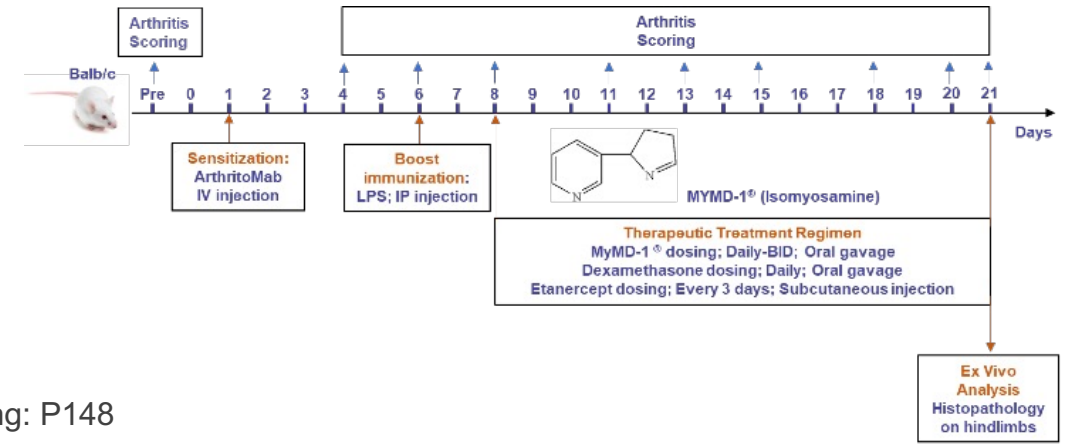
Rheumatoid arthritis (RA) is the most prevalent chronic inflammatory disease and is characterized by inflammation of the synovium of the joints, resulting in joint destruction. It is associated with chronic pain, loss of function, and disability. The murine model of Collagen Antibody Induced Arthritis (CAIA) mimics many of the features of arthritis in humans and has been used successfully in addressing questions of disease pathogenesis and to screen candidate therapeutic agents. Tumor necrosis factor-alpha (TNF-α) is a proinflammatory cytokine that plays a pivotal role in regulating the inflammatory response in chronic autoimmune diseases such as RA. The discovery of the role of TNF-α in the pathogenesis of RA has led to anti-TNF biological therapies as a breakthrough in the treatment of RA. The objective of this study was to investigate anti-inflammatory effects of MYMD-1®, a small molecule selective inhibitor of tumor necrosis factor alpha (TNF-α) with easy access to the body including the brain, in the murine CAIA model.

Adapted from poster presentation at the 2023 SOT Annual Meeting: P148

A Naturally Occurring Novel Therapeutic and Oral Selective Inhibitor of TNF-α, MYMD-1® (Isomyosamine), Significantly Reduced the Inflammation and Disease Severity in Murine Model of Collagen Antibody Induced Arthritis

2 EXPERIMENTAL PROCEDURES

The CAIA model was induced in female Balb/c mice by an intravenous injection of a monoclonal antibodies cocktail that are directed to collagen type II on Day 1 (sensitization), followed by an intraperitoneal injection of the endotoxin LPS on Day 6 (boost immunization). Three oral doses of MYMD-1® (50, 250 and 450 mg/kg/day) given BID (two times a day) were tested starting at the onset of the disease (Day 8 in this study). In addition, Dexamethasone was given daily by oral gavage at 0.3mg/kg and Etanercept was administered subcutaneously twice weekly at 10 mg/kg, both as positive controls. The therapeutic effect of MYMD-1® on inflammation was assessed by measuring the clinical score and paw inflammation (volume). At termination, the histopathological features such as infiltration of polymorphonuclear and mononuclear cells, pannus formation, cartilage degradation and bone resorption of the affected joints were analyzed. Statistical analysis were performed using Unpaired student t-test, One-Way or Two-way ANOVA in comparison to the CAIA/vehicle control. *;+p<0.05; **; ++p<0.01; ***;+++p<0.001; ****;++++p<0.0001.



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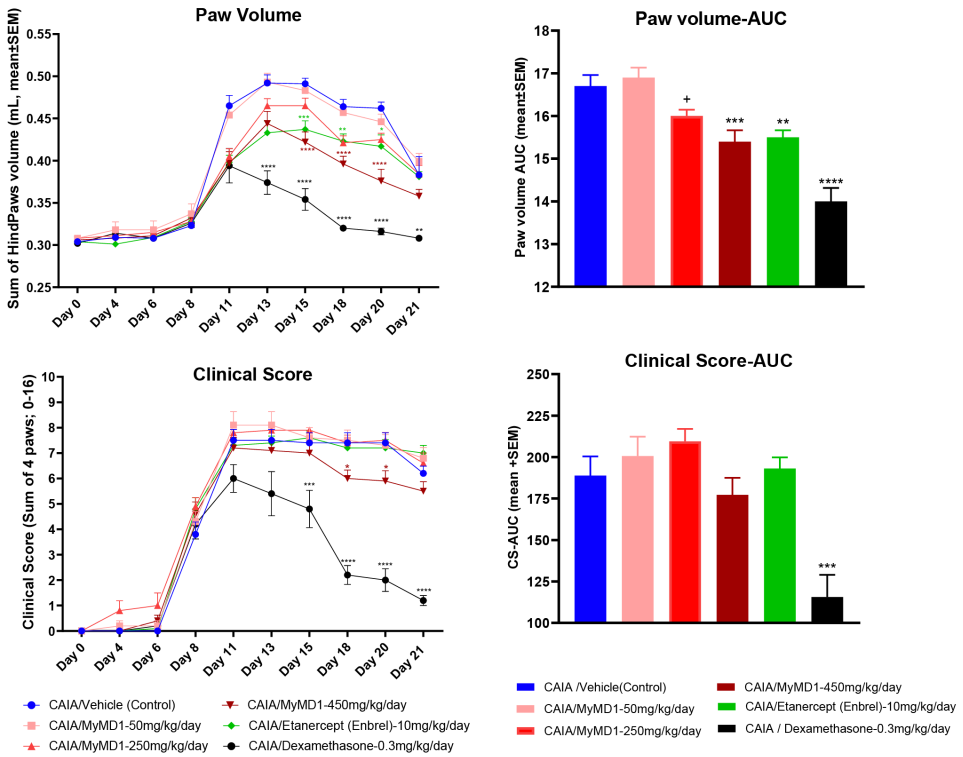
3 IN-LIFE RESULTS

Following arthritis induction, paw inflammation was observed starting from Day 8, peaked on Days 11 to 13 and then slowly decreased towards the end of the study (Days 20 to 21). Treatment with MYMD-1® 450 mg/kg/day significantly reduced the clinical score and the paw volume in BALB/c arthritic mice when compared to CAIA disease control (Figure 1). A similar observation was noted with MYMD-1® at 250 mg/kg/day but at lesser extent. There was no clinical signs and no effect on body weights associated with MYMD-1® treatment.

Figure 1: Clinical Score and Paw Volume Measurements



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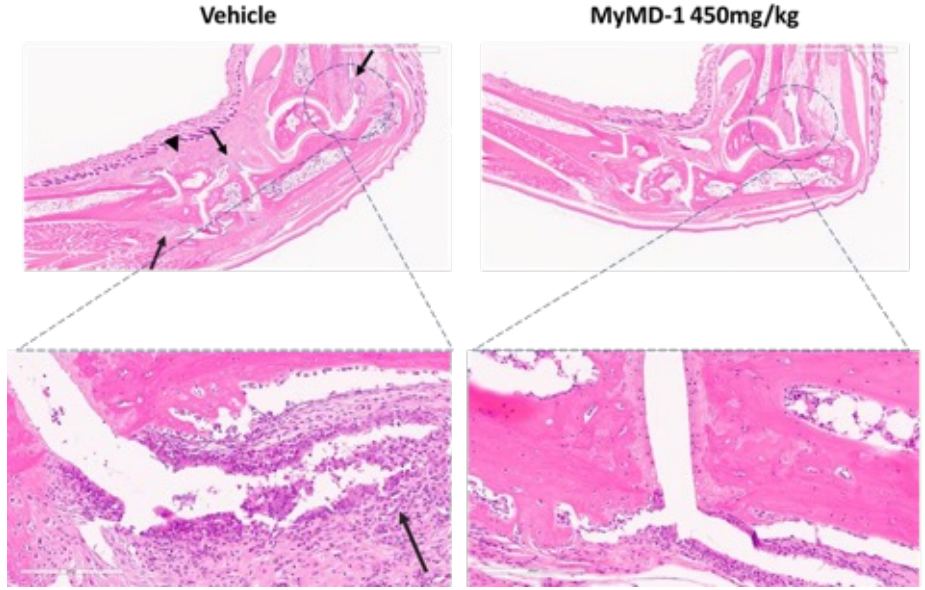
4 HISTOPATHOLOGY RESULTS

Histopathological changes associated with arthritis (inflammation, erosion, synovial hyperplasia, bone degeneration and periosteal changes) were observed in CAIA/vehicle control animals. Disease severity (total composite score) was reduced by 47% with MYMD-1® at 450 mg/kg/day while the reduction was 37% with Etanercept at 10 mg/kg (Figure 2). MYMD-1® at 50mg/kg/day had no reductive effect on the disease state. Scanned images obtained from decalcified left hindlimbs stained with H&E show the thickenir of the joint space by pannus and inflammation in the vehicle control when compared to MYMD-1® (450mg/kg) treatment (Figure 3).



Adapted from poster presentation at the 2023 SOT Annual Meeting: P148

Figure 2: Effect of MYMD-1® on histopathology changes

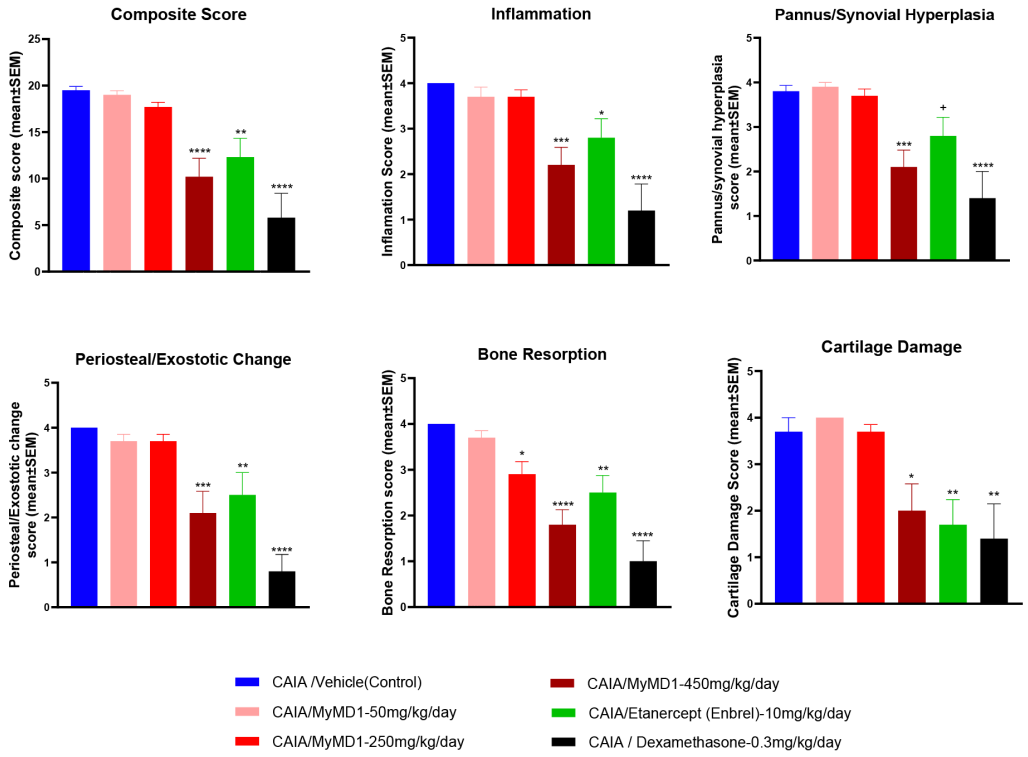


Figure 3: Representatitve Hematoxilin Eosin (H&E) staining of decalcified left hindpaw

Upon low and high magnification in the tibiotarso-metatarsal joint, joint space is thickened by pannus and inflammation (arrows) in vehicle control when compared to MYMD-1® treated animal. Periosteal reaction (bone exostosis) is also noted (arrowhead) in the vehicle control.

A Naturally Occurring Novel Therapeutic and Oral Selective Inhibitor of TNF-α, MYMD-1® (Isomyosamine), Significantly Reduced the Inflammation and Disease Severity in Murine Model of Collagen Antibody Induced Arthritis

5 CONCLUSION

MYMD-1® administration at 450 mg/kg/day inhibited arthritis development in Collagen Antibody Induce Arthritis murine model, with in-life data consistent with histopathological findings. Moreover, no clinical signs or body weight loss was associated with MYMD-1® treatment at 450mg/kg/day. Unlike currently available TNF-α inhibitors, MYMD-1® can be given orally and is a promising drug for rheumatoid arthritis.

Adapted from poster presentation at the 2023 SOT Annual Meeting: P148

MYMD-1® Regulatory Pathway in RA

Double-Blind, Placebo-Controlled Study: Recruiting 3rd Quarter of 2023

12 weeks | Oral Capsule | Male and Female Adults | Rheumatoid Arthritis | United States

Phase 2 study investigating the efficacy, tolerability and pharmacokinetics of MyMD-1® in the treatment of participants with RA

- **Planned IND Submission** 2Q2023
- **Planned IRB Protocol Approval** 2Q2023
- **Planned Patient Recruitment** 3Q2023
- **Planned Enrollment/Dosing to Begin** 3Q2023

OBJECTIVES

Primary

- Demonstrate that MYMD-1® added to MTX, in participants with moderate-to-severe active RA, is effective for reduction of signs and symptoms of rheumatoid arthritis at 28 days.
- To evaluate the biological activity of MyMD-1® added to MTX, in participants with moderate-to-severe active RA.

ENDPOINTS

Primary Efficacy Endpoint

- Percentage of Participants Achieving American College of Rheumatology 20% (ACR20) Response at 28 days
- Percent change from baseline: TNF-α

Tackling Sarcopenia in Aging Populations

MYMD-1®: Next-Generation Oral TNF-alpha Inhibitor

Opportunity in Aging

Aging is closely linked to **frailty, multiple morbidities, and death**

due to conditions such as **neoplastic, cardiovascular, neurodegenerative, metabolic**, or **autoimmune diseases**

Cost of hospitalizations for individuals with sarcopenia in the U.S. was estimated at **$40.4 Billion**

25% of +65 year olds are affected by sarcopenia

Global Population +65 is **~700 million**

Americans 65+ projected to grow from **52 M in 2018** to **95M** by 2060

16% to 23% of total population

Sarcopenia, or age-related frailty and decline in physical function, leads to **increased hospitalization, disability, and death**

References

1. St Sauver JL, Boyd CM, Grossardt BR, Bobo WV, Finney Rutten LJ, Roger VL, et al. Risk of developing multimorbidity across all ages in an historical cohort study: differences by sex and ethnicity. BMJ Open. 2015;5:e006413 2. Kochanek KD, Murphy SL, Xu J, Arias E. Deaths: Final Data for 2017. Natl Vital Stat Rep. 2019;68:1-77; 3. https://www.prb.org/resources/fact-sheet-aging-in-the-united-states/ 4. United Nations World Population Aging 2019; 5. Economic Impact of Hospitalizations in US Adults with Sarcopenia

MYMD-1®: Published Data

Published in *Journal of Drug Research*

- Shown to be safe and well tolerated.
- Pharmacokinetics of Oral Dose in Capsules in Healthy Subjects
- Decrease in TNF-α levels in MYMD-1® over placebo

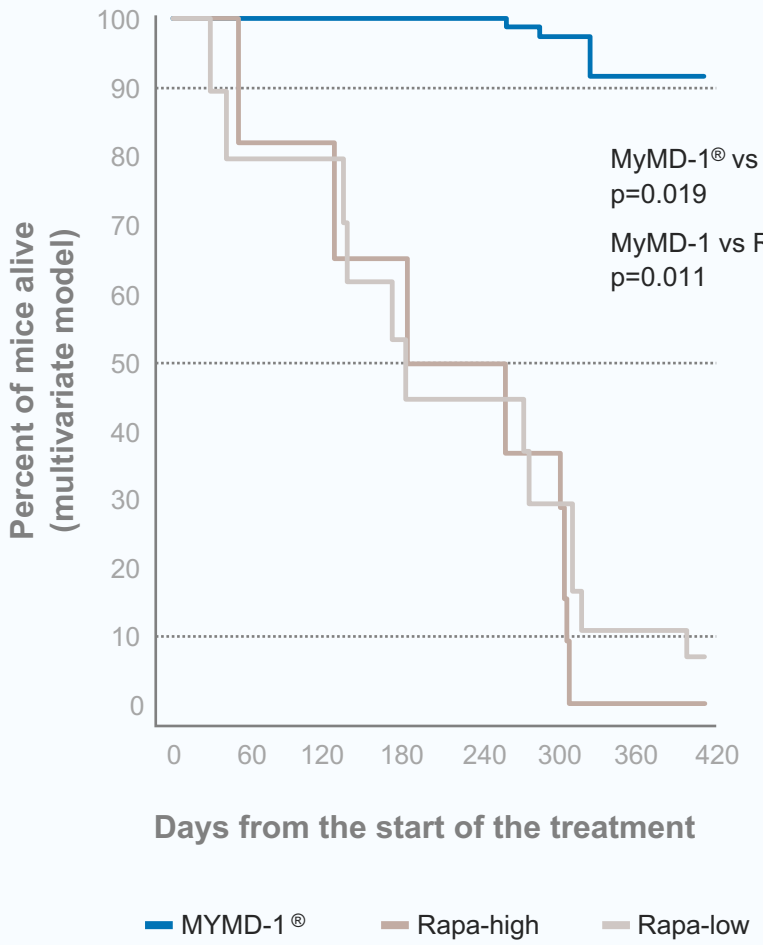
Conducted at Johns Hopkins School of Medicine and published in Journal of Gerontology: Biological Sciences

- 4x more effective than rapamycin in delaying aging
- Extended life of mice, even at advanced age
- Improved health span characteristics in terms of weight loss, muscle strength, and frailty progression

References
https://www.thieme-connect.de/products/ejournals/abstract/10.1055/a-1962-6834
https://doi.org/10.1093/gerona/glac142

MYMD-1®: Significant Four-Fold Improvement with MYMD-1® vs Rapamycin

MYMD-1 vs Rapa-high and Rapa-low



- Preclinical data, conducted at Johns Hopkins School of Medicine with exciting implications
  - ✓ Delayed aging and extended the lifespan of mice, even at advanced age
  - ✓ Improved health span characteristics
    - ✓ Milder weight loss
    - ✓ Greater muscle strength maintenance
    - ✓ Amelioration of progression to frailty
- No concerns of increased beta amyloid accumulation in the brain in the study

MYMD-1® Regulatory Pathway in Sarcopenia/Frailty

Double-Blind, Placebo-Controlled Study: Recruiting Active/Ongoing

28 days

Oral Capsule

Male and Female
aged 65 years or older

Sarcopenia/Frailty

United States

Phase 2 study investigating the efficacy, tolerability and pharmacokinetics of MYMD-1® in the treatment of participants with with chronic inflammation associated with sarcopenia/frailty

IND Cleared FDA
October 2021

Screening
February 2022

Enrollment/Dosing
February 2022

Last Patient Enrolled
2nd Quarter 2023
(anticipated)

OBJECTIVES

Primary

- Demonstrate reduction of chronic inflammatory markers in participants treated with MYMD-1®
- Evaluate the PK of oral doses of MYMD-1® capsules

ENDPOINTS

Primary Efficacy Endpoint

- Effect on serum levels of sTNFR1, IL-6, and TNFα over 28 days of treatment
- Plasma concentrations and parameters of MYMD-1®
- Urine parameters of MYMD-1®

Jeremy D. Walston, MD
Lead PI
Scientific Advisor

JOHNS HOPKINS
CENTER on AGING & HEALTH

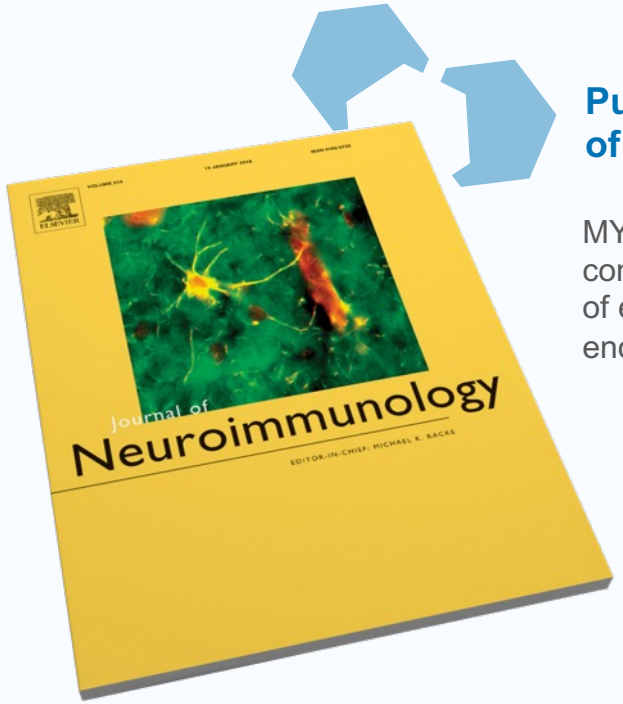
Additional Preclinical Targets

MYMD-1 ®: Next-Generation TNF-Alpha Inhibitor

MYMD-1® Proof of Concept Publications from Johns Hopkins School of Medicine Demonstrating MYMD-1® Regulation of TNFα

Published in The Journal of Immunology

MYMD-1 ®, a Novel Immuno-Regulator, Ameliorates Autoimmune Thyroiditis via Suppression of Th1 Responses and TNF-α Release



Published in Journal of Neuroimmunology

MYMD-1®, a novel alkaloid compound, ameliorates the course of experimental autoimmune encephalomyelitis (MS Model)

References
https://www.jimmunol.org/content/202/5/1350
https://www.jni-journal.com/article/S0165-5728(19)30220-6/fulltext

Additional Programs Underway

Bascom Palmer Collaboration

- On July 12, 2022, we announced a new collaboration with Bascom Palmer Eye Institute of Miami, Florida to collaborate on a pre-clinical study using MYMD-1® as a potential treatment for traumatic optic neuropathy (TON). To date, our collaboration with Bascom Palmer has included pre-clinical and clinical investigations.

Pre-Clinical

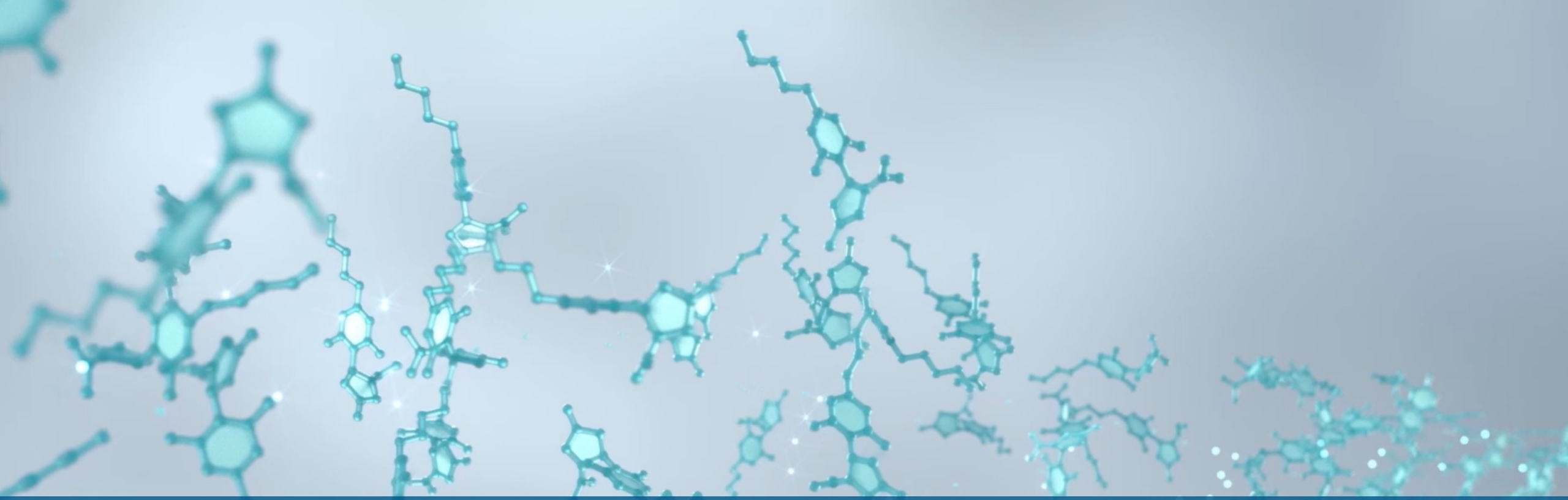
- In July 2022 we entered into a Material Transfer Agreement with Bascom Palmer. Our collaboration was announced in a press release and in an article in *Ophthalmology Times*. Bascom Palmer confirmed in August 2022 that it had received a quantity of our MYMD-1® product candidate and MyMD provided a material safety datasheet and certification of analysis.
- In August 2022, Bascom Palmer researchers conducted a preliminary introductory study of TON in mice. Investigators ran a crush injury of the mice's optic nerves with and without MYMD-1®. The study drug was given once per day via oral gavage at a dosage of 30 mg/kg of body weight. The mice were treated for five days, untreated for two days, and then sacrificed, and their TNF-α levels were measured. Data from this study is pending. We intend to plan additional pre-clinical studies.

Clinical

- In addition to the pre-clinical study described above, we are collaborating with Bascom Palmer to plan future a clinical study. In August 2022, Bascom Palmer researchers executed a confidentiality and non-disclosure agreement, and Bascom Palmer produced a draft protocol synopsis entitled, Assessment of the Anti-Inflammatory Effects of MYMD-1® in Non-Infectious Anterior Uveitis: A Randomized Controlled, Double Blind Clinical Study.

Additional Programs Completed

Neurology/Depression Program	
Pre-Clinical Completed	Immune-Mediated Depression
Pre-Clinical Completed	Multiple Sclerosis
Pre-Clinical Completed	MS Depression



Exploring Unmet Needs In Epilepsy, Chronic Pain And Anxiety

Supera-CBD™ : Next-Generation Synthetic Cannabinoid

DEA scientific review concluded Supera-CBD will not be **considered a controlled substance or listed chemical**

Opportunity in Epilepsy, Chronic Pain, and Anxiety for Supera-CBD™

Epilepsy

Adults with epilepsy **~3M** in the U.S.

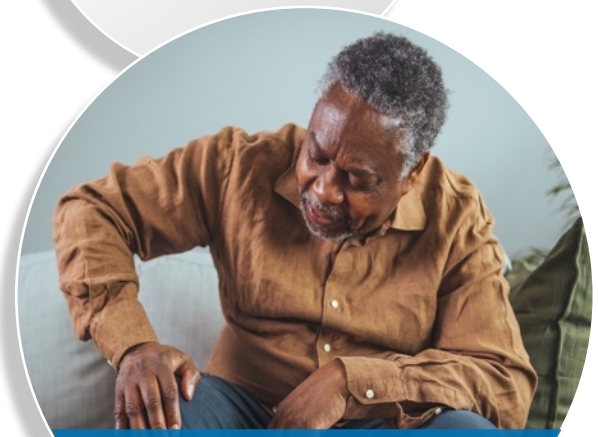
Epilepsy in seniors is up to **240** per 100,000 per year

~470,000 Children have active epilepsy in the U.S.

Chronic Pain

50.2 Million (20.5%) U.S. adults experience chronic pain based on analysis of **NHIS** data

It is estimated that the **total value of lost productivity** due to chronic pain is **$300B** annually



Anxiety

Generalized anxiety disorder (GAD) affects **6.8M** adults

3.1% of the U.S. population

An estimated **31.1%** of U.S. adults experience **an anxiety disorder** at some time in their lives

only **43.2%** are receiving treatment

References

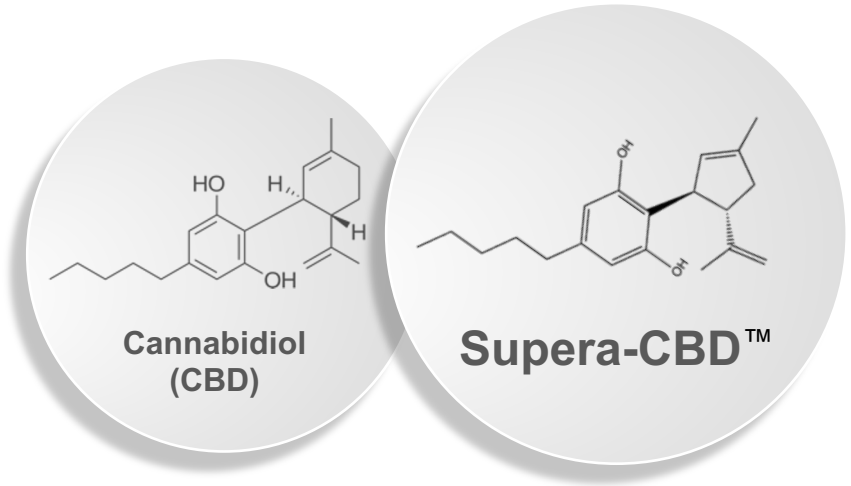
CDC 2020, Neuropsychiatric Disease and Treatment, 2016, Harvard Medical School, 2007. National Comorbidity Survey (NCS). (2017, August 21). Retrieved from https://www.hcp.med.harvard.edu/ncs/index.php. Data Table 1: Lifetime prevalence DSM-IV/WMH-CIDI disorders by sex and cohort.

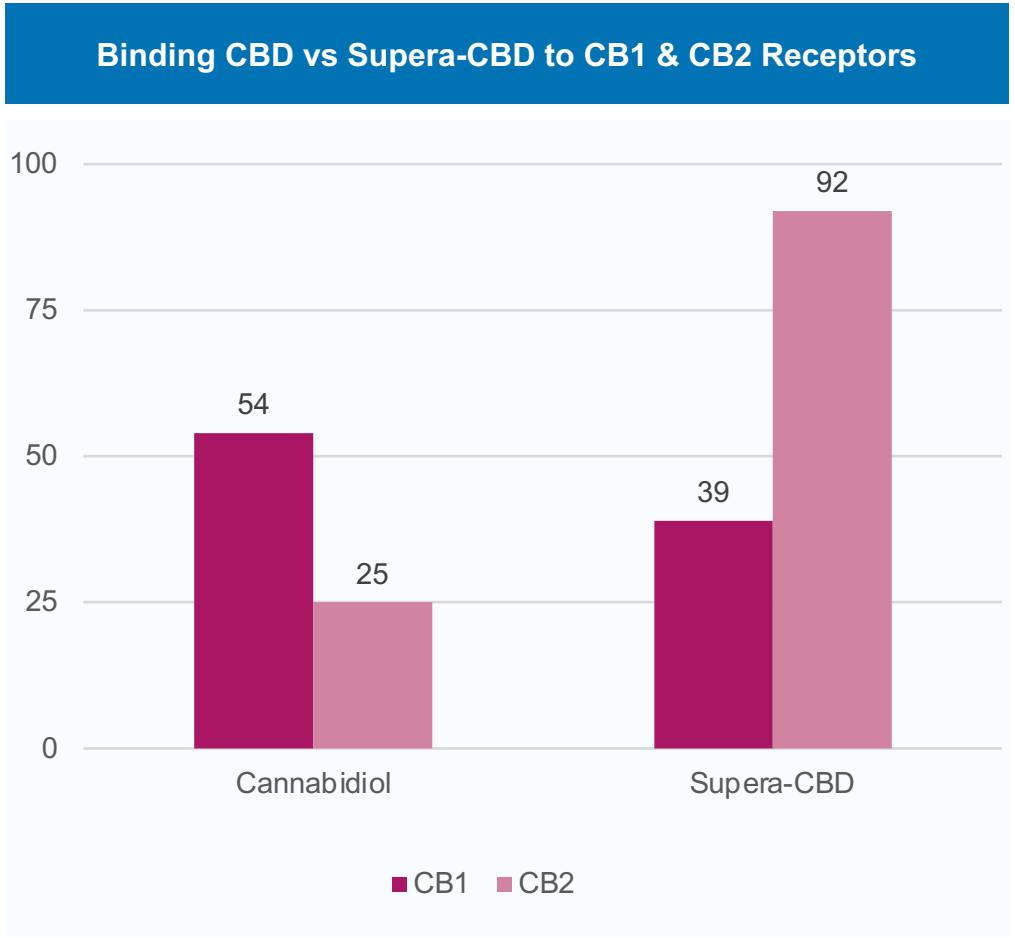
Positive Preclinical Data Supports Development

Studies have shown **Supera-CBD™** to be **dramatically more potent** compared to plant-derived CBD in its ability to **effectively target CB2 receptors**.

Agonists targeting CB2 receptors have the potential to treat acute, chronic and inflammatory pain, as well as neurological diseases.

Supera-CBD™ can be **synthesized** at a **fraction of the cost** of plant-derived CBD purification.





Supera-CBD™ vs. Plant-Derived CBD

Supera-CBD™

- Potent agonist at the CB2 Receptor
- The EC50 for Supera-CBD™ is 3.7 nM
- The EC50 is >8000 times greater than CBD which is >30,000 nM
- EC50=concentration that gives half-maximal effect at receptor activation

Plant-Derived CBD

- Has no physiological agonist activity at the CB2 receptor
- EC50 is >30 uM

Since CB2 receptor is the primary anti-inflammatory mechanism of action of cannabinoids, this suggests that **Supera-CBD™ could have dramatic therapeutic applications for diseases involving immune activation, such as autoimmune diseases, dementias and epilepsy.**

Supera-CBD is >8,000 Times More Potent a CB2R Agonist than CBD

Compound	Assay Name	Assay Format	Assay Target	EC50	Unit	Hill	Curve Bottom	Curve Top	Max Response
Supera-CBD™	cAMP	Agonist	CNR2	**0.00368**	uM	1.0761	0	90.158	94.699
CBD	cAMP	Agonist	CNR2	**>30**	uM	98.94			

Since CB2 receptor is the primary anti-inflammatory mechanism of action of cannabinoids, this suggests:

Supera-CBD™ could have dramatic therapeutic applications for diseases involving immune activation, such as autoimmune diseases, dementias and epilepsy.

Positioned for Significant Value and Growth

Corporate Overview

World-Class Leadership Team With A Proven Track Record

Chris Chapman, MD

President, Director and Chief Medical Officer

Adam Kaplin, MD, PhD

Chief Scientific Officer

Paul Rivard, Esq.

Chief Legal Officer

Jenna Brager, PhD, RN, MS

VP of Drug Development

Excellent IP Portfolio

Molecule	Program	Base Composition	Extensions
MYMD-1®	Rheumatoid Arthritis	March 31, 2036	TBD
	Sarcopenia	March 31, 2036	
	Type 2 Diabetes	April 16, 2037	
	Uveitis	March 31, 2036	
SUPERA CBD™	Chronic Pain	February 11, 2039	TBD
	Epilepsy	February 11, 2039	
	Addiction	February 11, 2039	
	Epilepsy	February 11, 2039	

Funded Through Value-Generating Milestones

MYMD-1®

Sarcopenia (Aging)	
Data readout	3Q2023 (anticipated)
Rheumatoid Arthritis	
Planned IND Submission	2Q2023
Planned IRB Protocol Approval	2Q2023
Planned Patient Recruitment	3Q2023
Planned Enrollment/Dosing to Begin	3Q2023
Hashimoto's Thyroiditis	
IND Cleared	2Q 2020
Phase I Completed	3Q 2021 (Ready to proceed to Phase 2)

SUPERA-CBD™

✓ POC Studies completed in Epilepsy, Chronic Pain, Anxiety	
Genotoxicity Completed	3Q 2022
File IND	TBD

PHASE 2 STUDY FOR SARCOPENIA

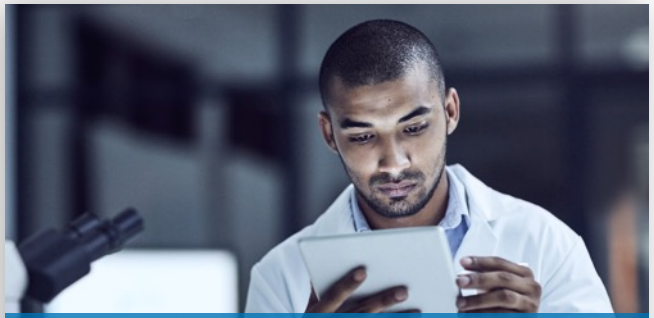
Johns Hopkins University

Clinical Research of West Florida

Tampa

Clearwater

The MyMD Opportunity

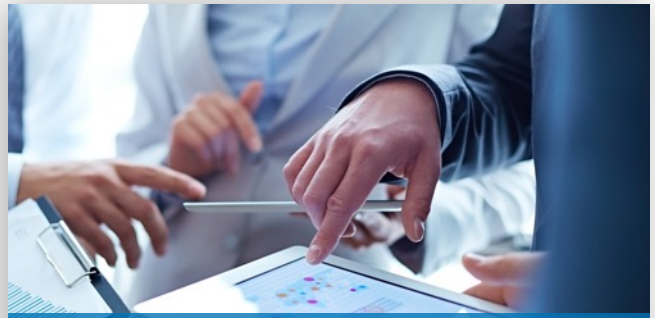


NEXT-GENERATION APPROACH

Promising Data

PIPELINE WITH BROAD POTENTIAL

Two Candidates
Targeting Large Markets



POSITIONED FOR SUCCESS

The Right Team
to Execute

Groundbreaking biotech research on first-in-class drug therapies

MYMD-1®: Orally Available Next-Generation TNF-Alpha Inhibitor

- Works by preventing the production of excess TNF-alpha in response to inflammatory signals
- Improved ease of administration
- Better safety profile compared to currently approved anti-TNF therapies
- Crosses the blood brain barrier opening the potential to treat a wide range of disease (autoimmune and inflammatory)
  - Phase 1 study for potential novel therapy in RA
- Potential to slow the aging process and extend healthy lifespan
  - Phase 2 studies for sarcopenia/frailty

Reducing pathological inflammation without completely blocking TNF-alpha activity thereby mitigating the risk of life-threatening infections

MYMD-1®: Potential to Transform the Way That TNF-Alpha Based Diseases Are Treated

- TNF-alpha is an immune modulating cytokine
  - Dysregulation can lead to prolonged inflammatory responses
  - Known to drive disease in a range of autoimmune and inflammatory disorders

- Current TNF-alpha inhibitors
  - Black box warnings around the risk of serious infections leading to hospitalization and death
  - Biologics administered by infusion or injection
  - Not all patients respond

MYMD-1®: The potential to provide meaningful therapeutic solutions to patients not served by current TNF-alpha inhibitors

Committed Board

Joshua Silverman

Chairman of the Board of Directors

Bill J. White

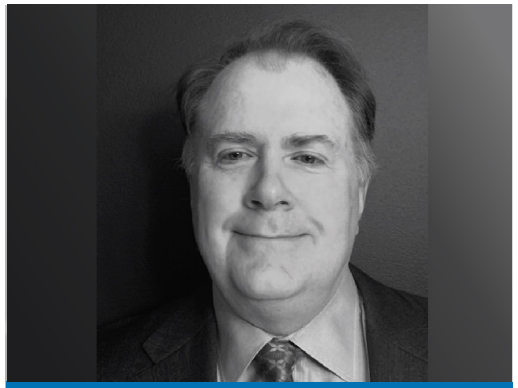
Director

Craig Eagle

Director

Chris Chapman, M.D.

President, Director and Chief Medical Officer



Chris Schrieber

Director

Jude Uzonwanne

Director


Patient
Aging
Approach
MyMD®
PHARMACEUTICALS