ANNOVIS

Attacks Alzheimer's Disease and Neurodegeneration by Improving the Information Highway of the Nerve Cell

Axonal Transport

Symbol: **ANVS** (NYSE American)

September 2021

FORWARD-LOOKING STATEMENTS

Statements in this presentation contain "forward-looking statements" that are subject to substantial risks and uncertainties. Forward-looking statements contained in this presentation may be identified by the use of words such as "anticipate," "expect," "believe," "will," "may," "should," "estimate," "project," "outlook," "forecast" or other similar words, and include, without limitation, statements regarding Annovis Bio, Inc.'s expectations regarding projected timelines of clinical trials, and expectations regarding current or future clinical trials. Forward-looking statements are based on Annovis Bio, Inc.'s current expectations and are subject to inherent uncertainties, risks and assumptions that are difficult to predict. Further, certain forward-looking statements are based on assumptions as to future events that may not prove to be accurate, including that clinical trials may be delayed; that the data reported herein is interim data, conclusions as to which may be superseded by subsequent data we expect to receive in connection with Phase 2a trials and/or subsequent clinical trials; and that any anticipated meeting with or presentation to the FDA may be delayed. These and other risks and uncertainties are described more fully in the section titled "Risk Factors" in the Annual Report on Form 10-K for the year ended December 31, 2020 and other reports filed with the Securities and Exchange Commission. Forward-looking statements contained in this presentation are made as of this date, and Annovis Bio, Inc. undertakes no duty to update such information except as required under applicable law.

HIGHLIGHTS

A novel approach to treat neurodegeneration is desperately needed

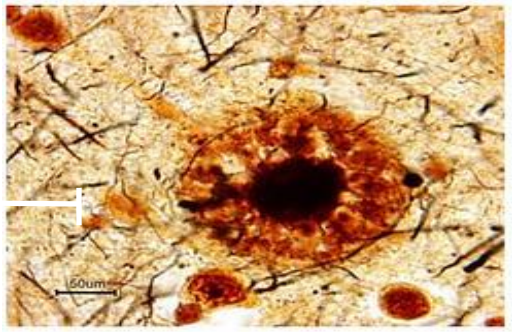
- **Annovis is developing drugs** for Alzheimer's (AD) and Parkinson's disease (PD), including the orphan indication Alzheimer's in Down Syndrome (AD-DS)
- **Lead compound - ANVS401 - in Phase 2a clinical trial,** is the only drug to improve cognition in AD and motor function in PD patients, as recently announced
- **ANVS401 reversed several steps of the toxic cascade,** in AD and PD patients as recently announced. Additional biomarker data to come.
- **Successful completion of phase 2a clinical trials** will validate our approach and allow start of two phase 3 studies

ANNOVIS' NEW APPROACH TO ATTACK AD AND PD

Chronic and acute brain insults lead to high levels of neurotoxic proteins, inflammation and to neurodegeneration

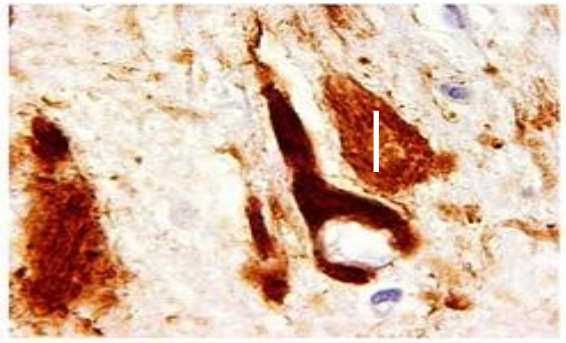
Amyloid β

Alzheimer's - Parkinson's
Aβ Targeting Compounds



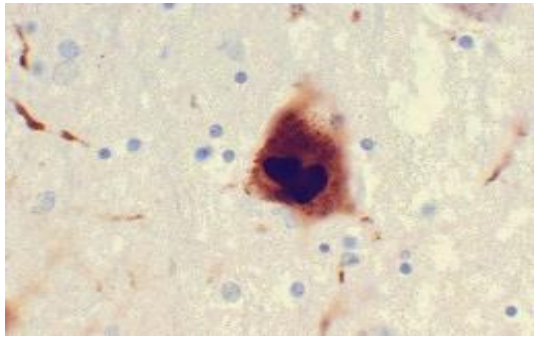
Tau

Tauopathies - Alzheimer's
Tau Targeting Compounds



aSynuclein

Parkinson's - Alzheimer's
aSYN Targeting Compounds



Attacking one neurotoxic protein results in minimal effect

ANVS401 is the only drug to attack multiple neurotoxic proteins simultaneously

NEUROTOXIC PROTEINS IMPAIR AXONAL TRANSPORT AND **CAUSE A TOXIC CASCADE**

HIGH LEVELS OF NEUROTOXIC PROTEINS

- IMPAIRED AXONAL TRANSPORT
- SLOWER SYNAPTIC TRANSMISSION
- INFLAMMATION
- DEATH OF NERVE CELLS
- LOSS OF COGNITIVE AND MOTOR FUNCTION

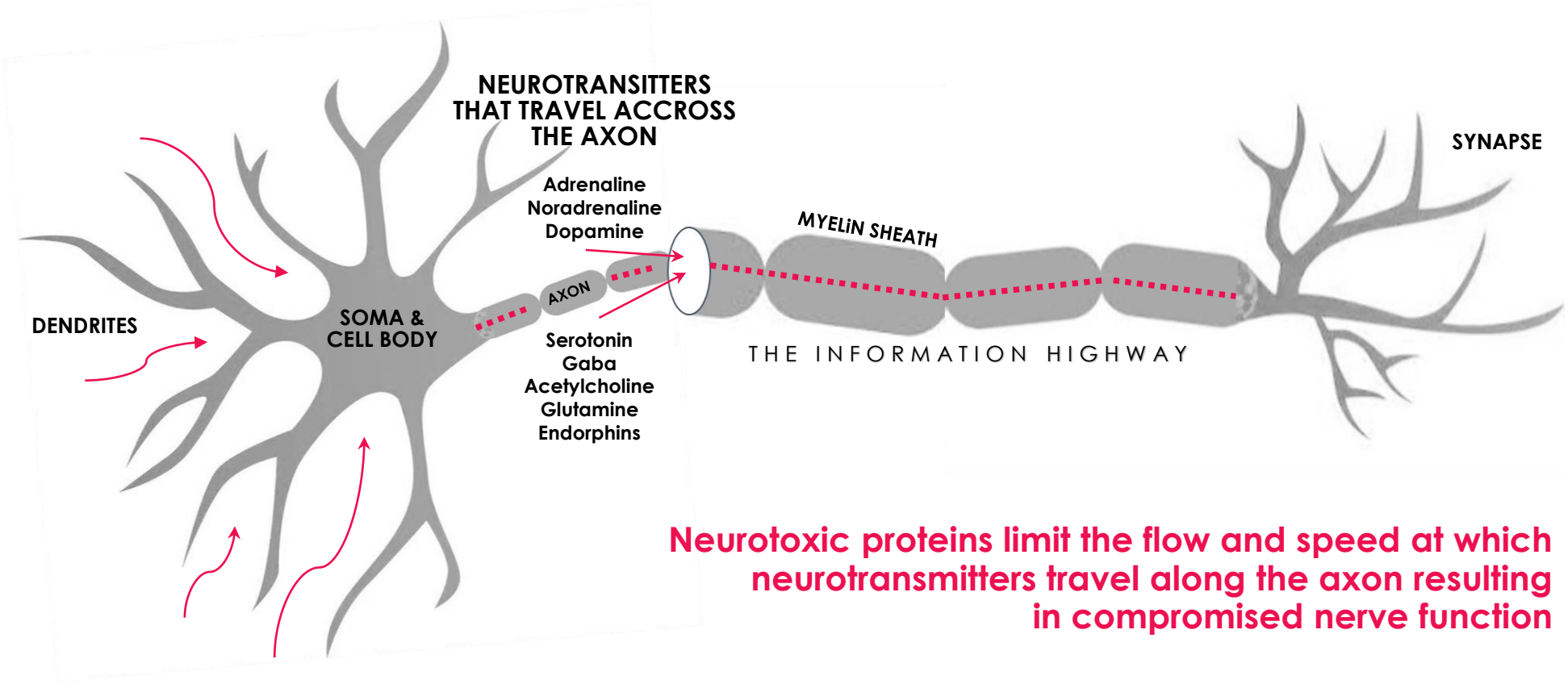
ANVS401 LOWERS LEVELS OF NEUROTOXIC PROTEINS

- IMPROVED AXONAL TRANSPORT
- INCREASED SYNAPTIC TRANSMISSION
- NO INFLAMMATION
- HEALTHY NERVE CELLS
- IMPROVED COGNITIVE AND MOTOR FUNCTION

ANVS401 IMPROVES AXONAL TRANSPORT AND **IMPEDES THE TOXIC CASCADE**

HOW NERVE CELLS WORK

In healthy nerve cells little packages containing neurotransmitters or nerve growth factors travel unimpaired from the cell body through the axon to the synapse.



Neurotoxic proteins limit the flow and speed at which neurotransmitters travel along the axon resulting in compromised nerve function

NEURODEGENERATION IS AN AXONAL TRANSPORT DISEASE

"Axonal transport disruption is linked to human neurological conditions." - Nature Review, September 2019

Axonal transport is responsible for:

- Neurotransmitters GABA (anxiety), ACh (cognition), dopamine (movement), serotonin (mood)
- Neurotrophic factors NGF, BDNF
- All communication within and between nerve cells

Retrograde (0.5 frame/sec)

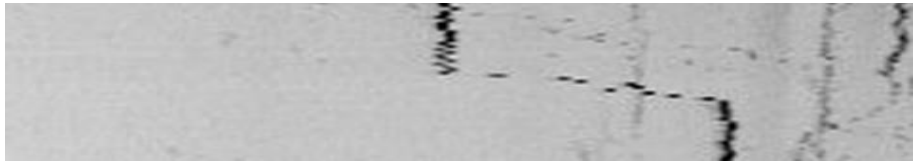
Normal Transport

The ***Normal Flow and Speed*** of vesicles carrying BDNF across the axon.

(88s)

Abnormal Transport

Shows the ***Blockage and Slowing*** of BDNF across the axon. Black areas demonstrate where transport is slowed due to high levels of neurotoxic proteins.



(120s)

TREATED WITH ANVS401

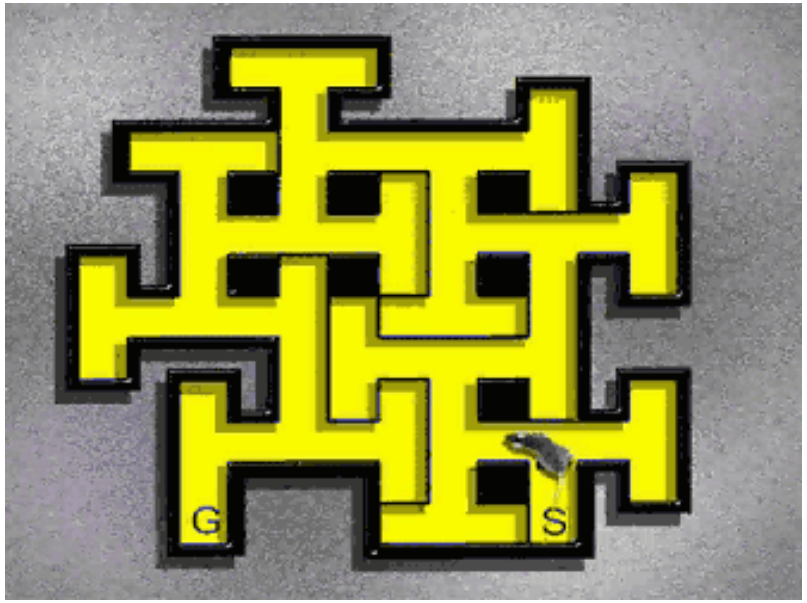
The ***Flow and Speed*** of axonal transport is improved.

(88s)

APP, Ab42, C99 – Mobley, UCSD; aSYN – Isacson, Harvard; Lee, U.Penn;
Tau – U. Muenich & Zuerich; Htt – Mobley, UCSD; TDP43 – Taylor, Northwestern

RESULTS IN ANIMALS

Multiple animal studies showed that ANVS401 fully recovered the affected function



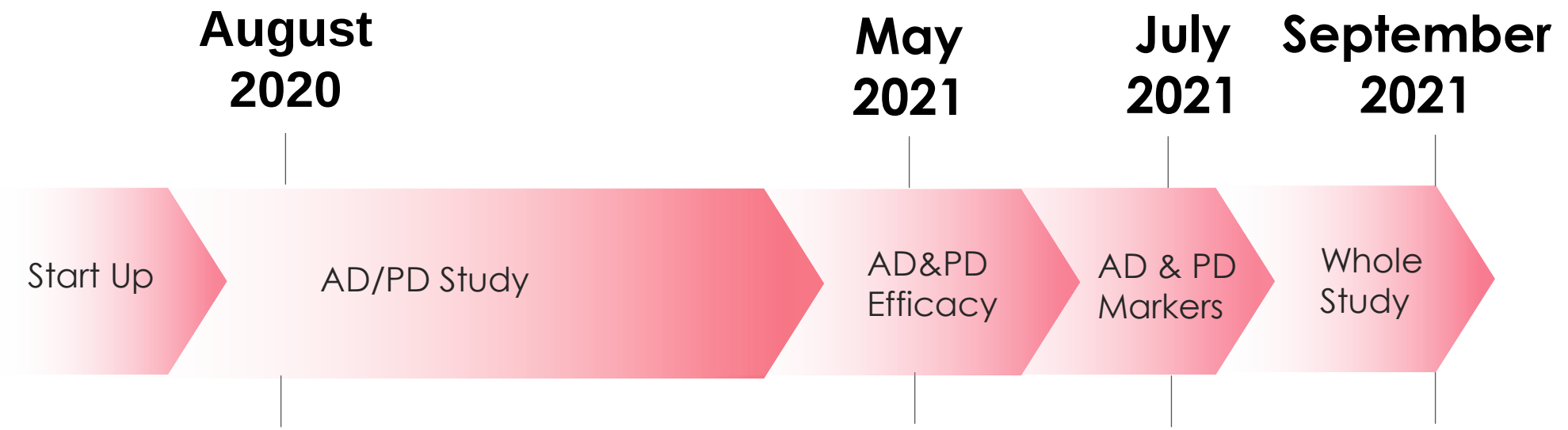
Function		Animal Model
Memory and learning (4)	→	AD mice, DS mice, stroke mice, TBI rats
Movement (2)	→	PD mice, FTD mice
Eyesight (1)	→	Acute glaucoma rats

TWO PHASE 2 CLINICAL TRIALS

	AD Trial	PD Trial
Therapeutic Area	Early to Moderate AD	Early to Moderate PD
Patients	14	14 + 40
Phase	2	
Sites	12	
Country	United States	
Design	Double-Blind, Placebo-Controlled, Biomarker Study	
Endpoints	Reversal of Toxic Cascade	
Exploratory	Efficacy	

TIMELINE OF PHASE 2 CLINICAL TRIAL IN AD and PD

Preliminary data commenced in 1Q2021



A meeting with the FDA to discuss the data from the AD and the PD study as well as from the chronic toxicology in rats and dogs is projected for Fall of 2021

BASELINE DEMOGRAPHICS

	ALZHEIMER			PARKINSON		
Patients Enrolled	Placebo (N=6)	ANVS401 80mg (N=10)	Total (N=16)	Placebo (N=5)	ANVS401 80mg (N=10)	Total (N=15)
Age (years)	68.0 (6.87)	72.8 (6.34)	71.0 (6.75)	75.4 (3.13)	65.0 (9.31)	68.5 (9.18)
Male	3 (50.0%)	2 (20.0%)	5 (31.3%)	3 (60.0%)	8 (80.0%)	11 (73.3%)
Female	3 (50.0%)	8 (80.0%)	11 (68.8%)	2 (40.0%)	2 (20.0%)	4 (26.7%)
HISPANIC	4 (66.7%)	5 (50.0%)	9 (56.3%)	2 (40.0%)	0 (0.0%)	2 (13.3%)
CAUCASIAN	2 (33.3%)	5 (50.0%)	7 (43.8%)	3 (60.0%)	10 (100.0%)	13 (86.7%)
WHITE	4 (66.7%)	8 (80.0%)	12 (75.0%)	5 (100.0%)	10 (100.0%)	15 (100.0%)
AFRICAN AMERICAN	1 (16.7%)	0 (0.0%)	1 (6.3%)	0	0	0
ASIAN	1 (16.7%)	1 (10.0%)	2 (12.5%)	0	0	0
NATIVE HAWAIIAN	0 (0.0%)	1 (10.0%)	1 (6.3%)	0	0	0

SAFETY SUMMARY

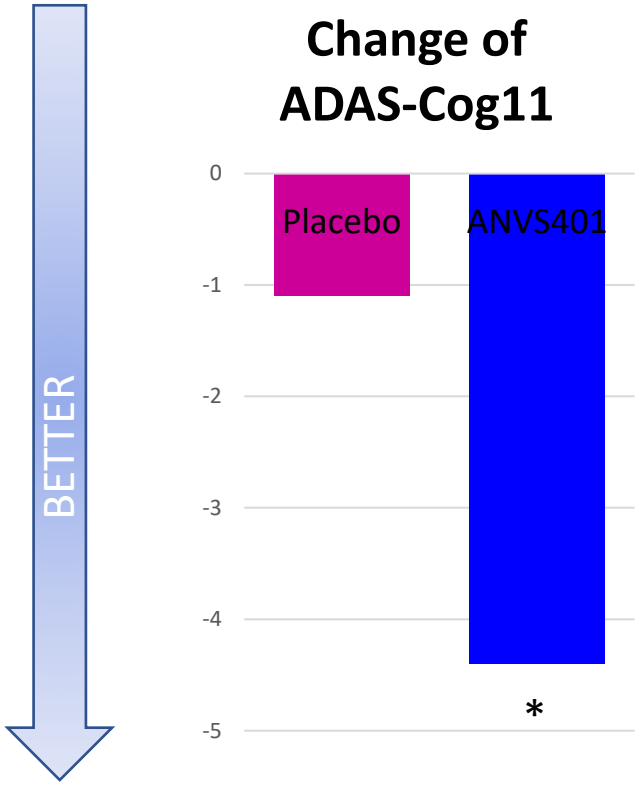
Data from first 14 AD and PD patients

	AD Patients			PD Patients		
	Placebo (N=6)	ANVS401 80mg (N=10)	Total (N=16)	Placebo (N=5)	ANVS401 80mg (N=10)	Total (N=15)
Subjects with any AEs	3 (50.0%)	5 (50.0%)	8 (50.0%)	3 (60.0%)	3 (30.0%)	6 (40.0%)
Number of AEs	4	7	11	5	3	8
Serious AEs	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
AEs that led to Drug Interrupted	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
AEs that led to Drug Withdrawn	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
AEs Suspected Drug Related	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (20.0%)	0 (0.0%)	1 (6.7%)
AEs Study Procedure	3 (50.0%)	4 (40.0%)	7 (43.8%)	2 (40.0%)	1 (10.0%)	3 (20.0%)
CTCAE Grade 1	3 (50.0%)	4 (40.0%)	7 (43.8%)	3 (60.0%)	3 (30.0%)	6 (40.0%)
CTCAE Grade 2	0 (0.0%)	1 (10.0%)	1 (6.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)

Most AEs were due to the spinal fluid collection that resulted in headaches and back aches

IMPROVED COGNITION IN AD PATIENTS – ADAS-Cog11

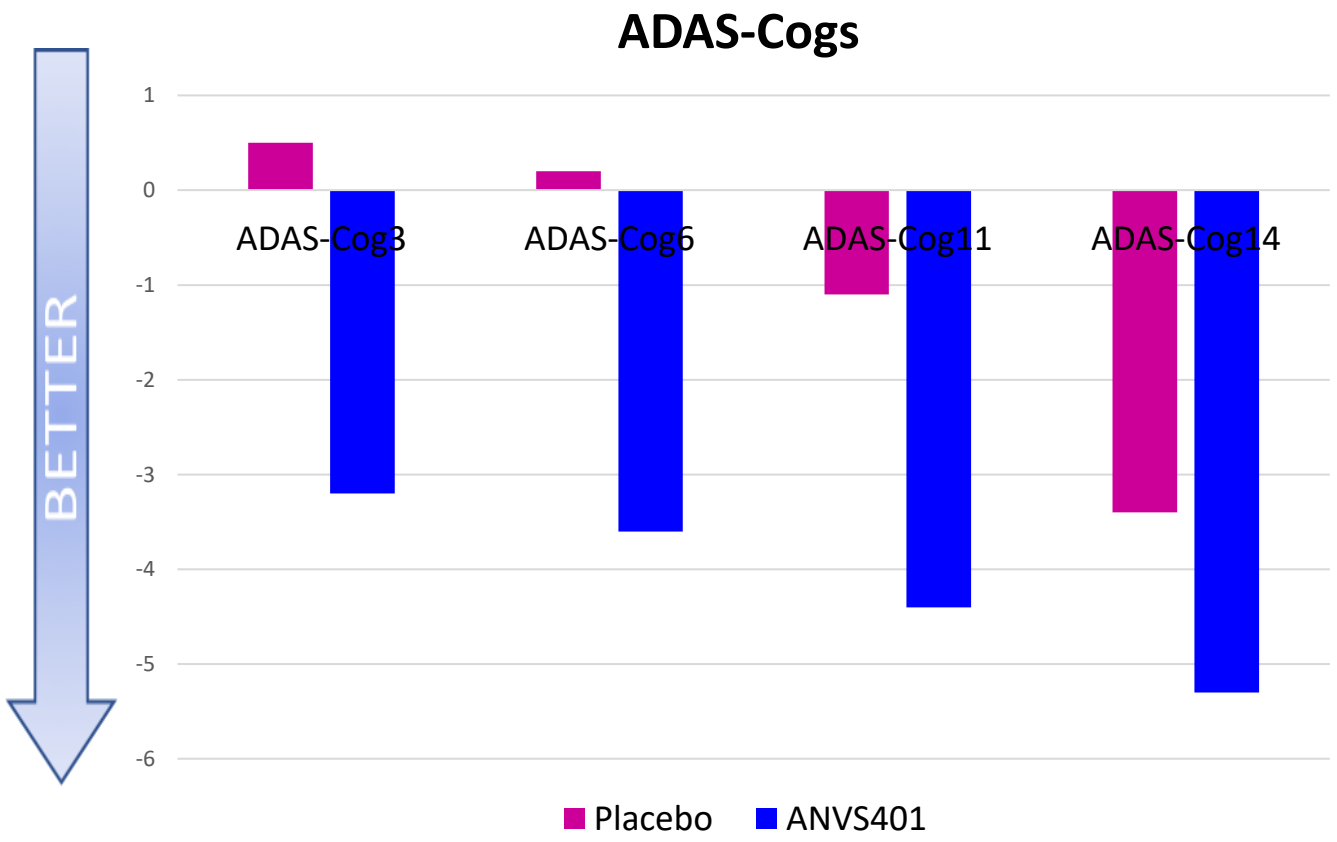
Data from 14 AD patients



From baseline to 25 days in the ANVS401-treated group, ADAS-Cog11 improved by 4.4 points, a statistically significant improvement of 30% ($p<0.05$). Compared to placebo at 25 days the treated group is 3.3 points better than the placebo

IMPROVED COGNITION IN AD PATIENTS – ADAS-Cogs

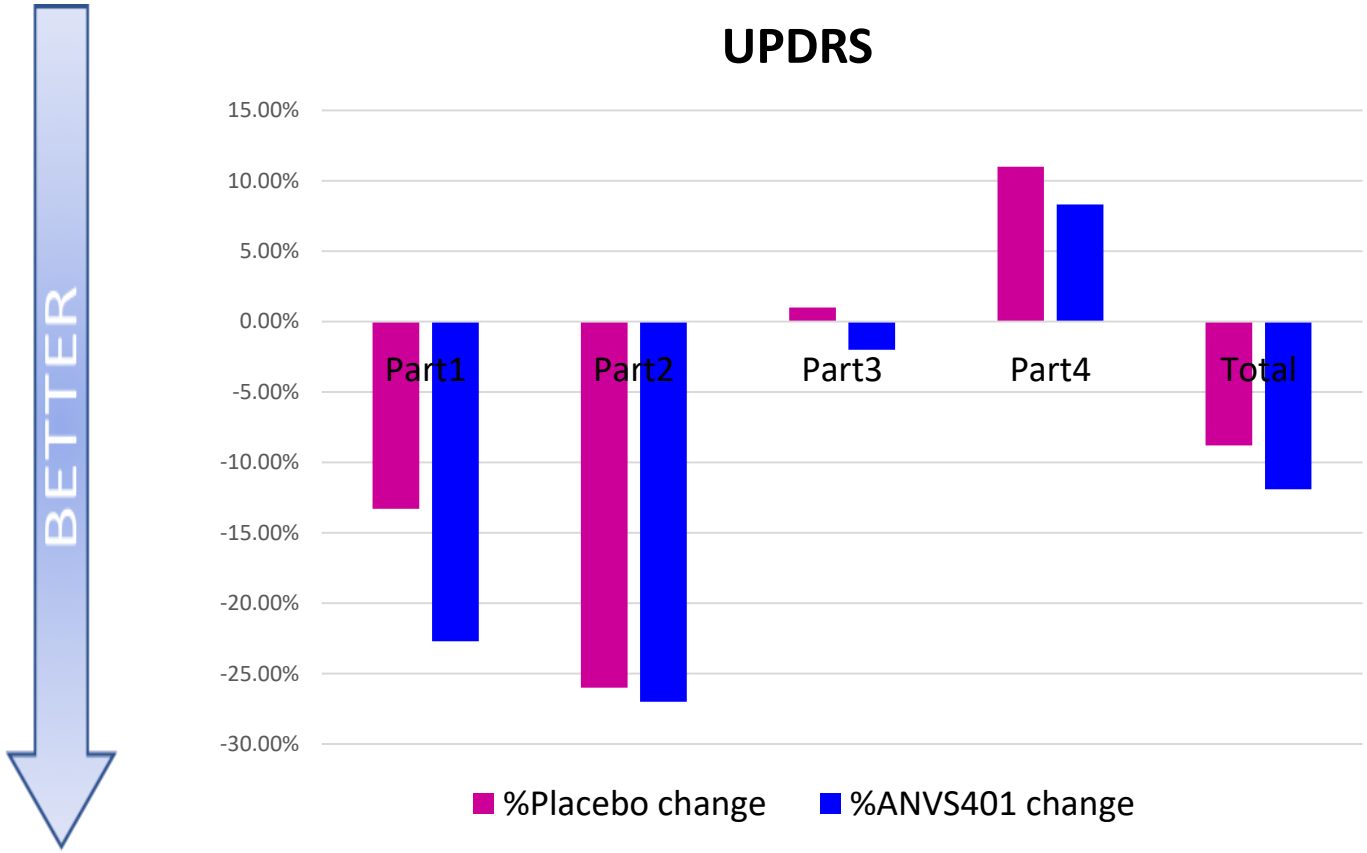
Data from 14 AD patients



While the whole ADAS-Cog 11 test is statistically significantly better in ANVS401-treated patients than at baseline, the ANVS401 group also shows trends of improvement in all four ADAS-Cog tests performed compared to placebo.

EFFICACY IN PD PATIENTS – MDS-UPDRS TEST

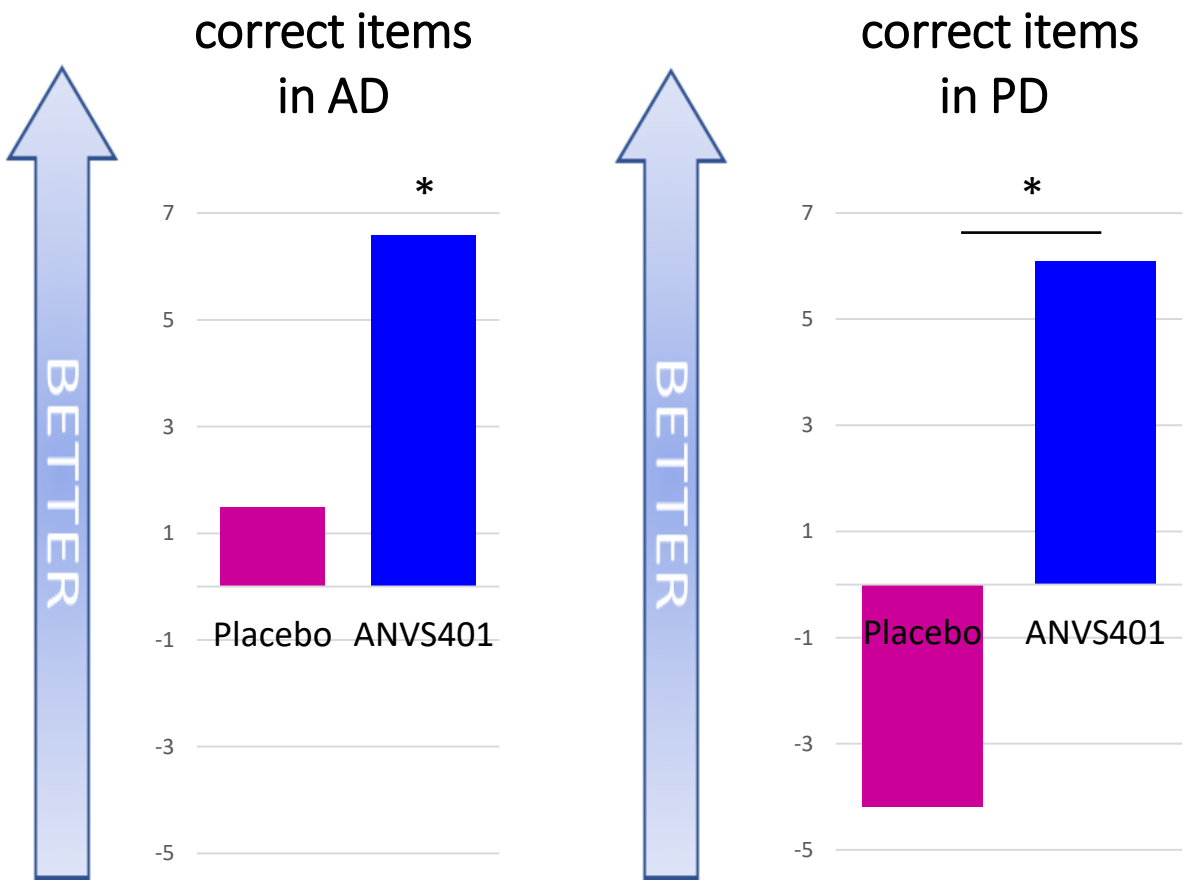
Data from 14 PD patients



ANVS401-treated group showed trends of improvement in all four parts of UPDRS test compared to placebo

IMPROVED SPEED AND ACCURACY IN AD AND PD PATIENTS
WAIS CODING TEST

Data from 14 AD and 14 PD patients



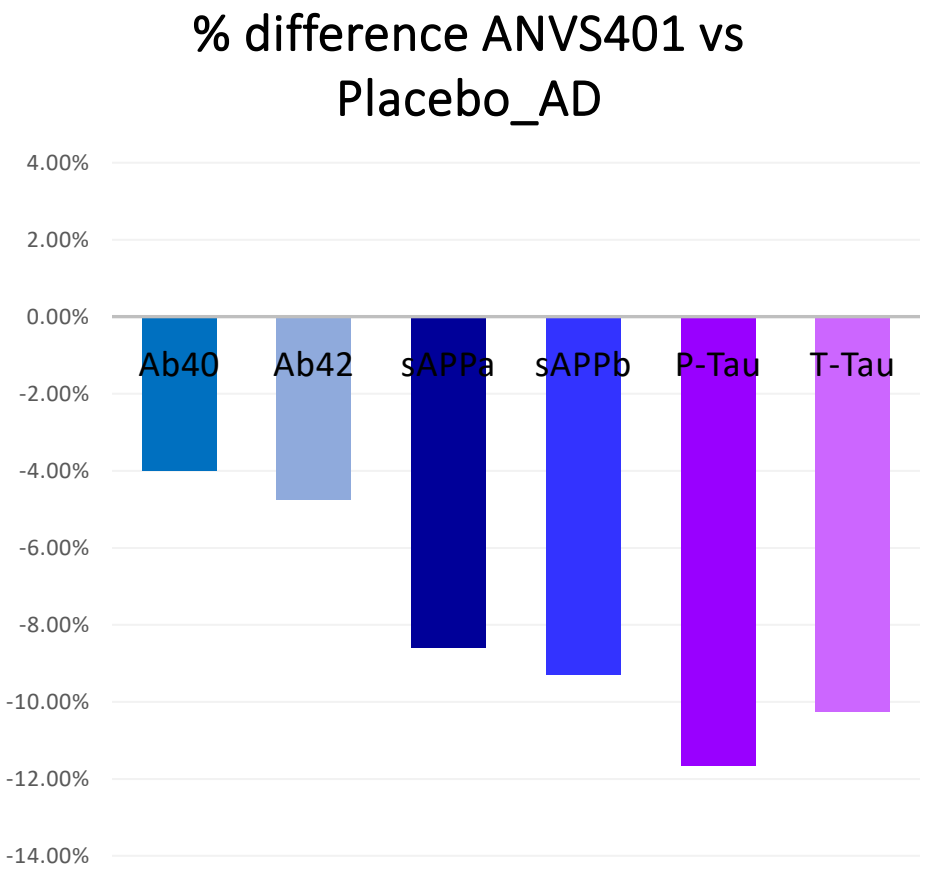
The WAIS coding test measures speed in movement and thinking. Treated AD patients show a statistically-significant 23% improvement and PD patients a statistically-significant 24% improvement compared with placebo

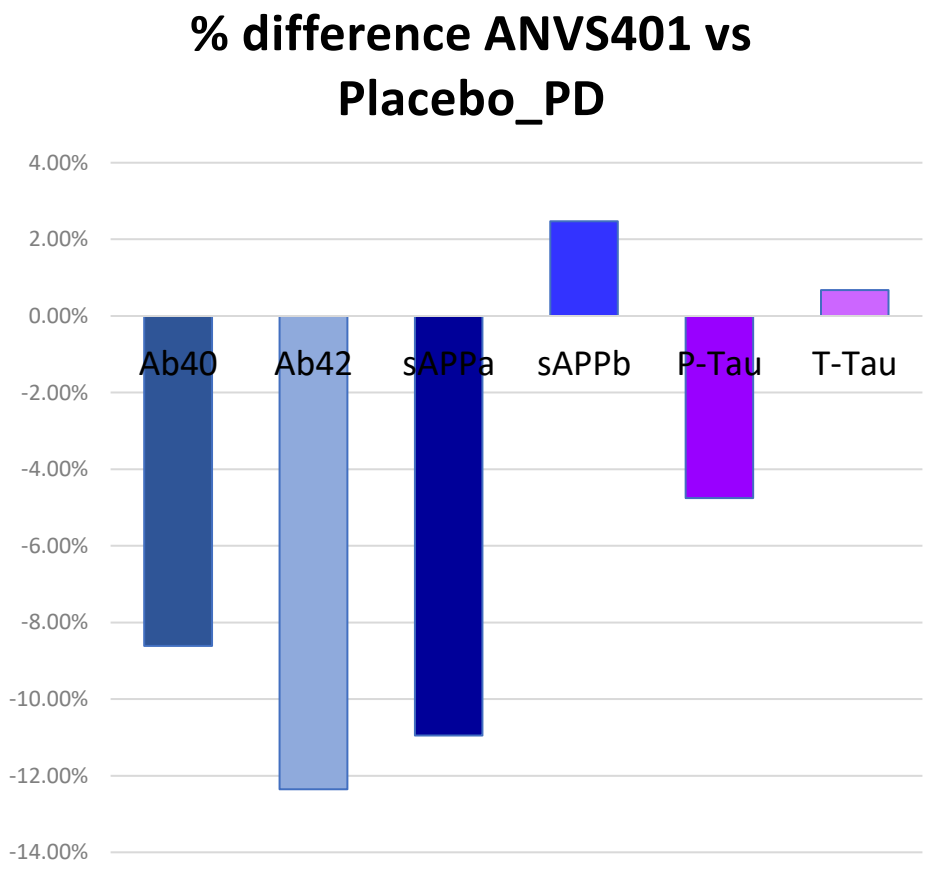
MARKERS OF TOXIC CASCADE

REVERSAL OF TOXIC CASCADE	
Step 1: Lower levels of neurotoxic proteins	sAPPa, sAPPb, Ab42, Ab40, Tau, P-Tau (alpha-Synuclein)
Step 2: Improved axonal transport	(BDNF)
Step 2: Improved axonal health	NFL
Step 3: Lower inflammation	(sTREM2, YKL40 & GFAP)
Step 4: Lower nerve cell death	(SNAP25, Neurogranin)

STEP 1: NEUROTOXIC PROTEINS ARE LOWERED IN AD AND PD PATIENTS

Data from first 14 AD and PD patients





Aβ42/Aβ40 RATIO IN AD PATIENTS

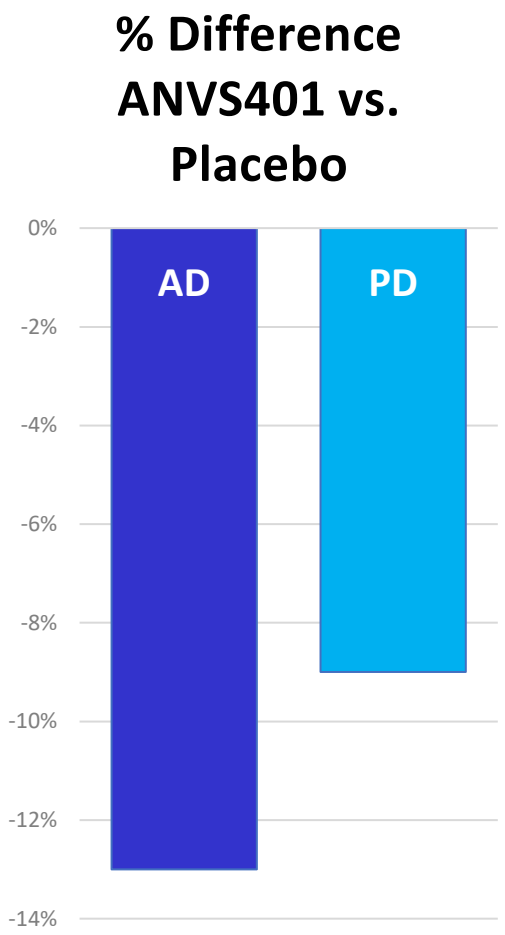
Data from first 14 AD patients

	AD Patients	
	Placebo	ANVS401
Baseline	0.064	0.059
25 Days	0.064	0.062
p-Value		0.0113

The Aβ42/Aβ40 ratio is well-accepted standard for AD. Patients with AD have a ratio **< 0.072** and without >0.072. ANVS401-treated patients improve their ratio statistically significantly after 25 days showing improvement in AD.

NEUROFILAMENT LIGHT IN AD AND PD PATIENTS IS LOWERED

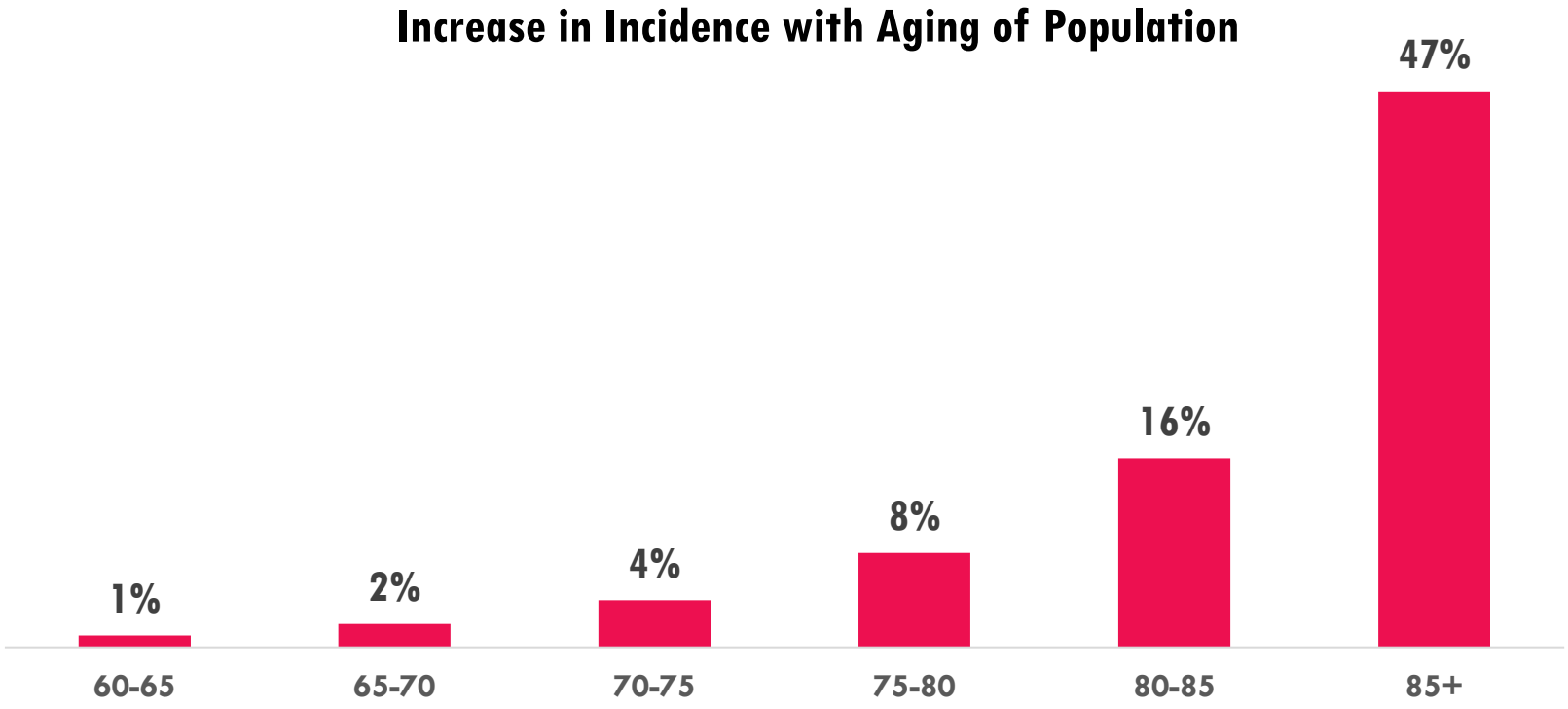
Data from first 14 AD and PD patients



Neurofilament light represents the health of the axon and neuron.

In both Posiphen-treated patient populations, NfL is reduced representing better axonal health.

MARKET PROJECTIONS



Source: Alzheimer's Association 2014; Incidence of AD in Relation to Age

Annual sales potential for US and worldwide are over $100 billion dollars

FINANCIAL HIGHLIGHTS

- Completed $50M equity raise in May 2021
- Cash balance $49 million, debt $0 as of June 30, 2021
- Fully funded through anticipated Phase 3 trial / two years
- NIH grants funding ADCS Phase 2a trial in AD and chronic toxicology study
- ~38% insider ownership
- Shares outstanding 8.1 million; float 5.6 million
- Analyst coverage from ThinkEquity and Maxim Group

MANAGEMENT AND ADVISORY TEAM

Maria L. Maccecchini, PhD, Founder, President & CEO

Dr. Maccecchini founded Annovis in May 2008 to develop better therapeutics for Alzheimer's, Parkinson's and other neurodegenerative diseases. Was partner and director of two angel groups, Robin Hood Ventures and MidAtlantic Angel Group; Founder and CEO of Symphony Pharmaceuticals/Annovis a biotech company that sold in 2001 to Transgenomic; General Manager of Bachem Bioscience, the US subsidiary of Bachem AG, Switzerland and Head Molecular Biology Mallinckrodt; Dr. Maccecchini did one postdoc at Caltech and one at the Roche Institute of Immunology, her PhD in biochemistry is from the Biocenter of Basel with a two-year visiting fellowship at The Rockefeller University.

Jeffrey McGroarty, CPA, MBA, Chief Financial Officer

Mr. McGroarty is a financial executive with experience in investor relations, working with analysts, creditors and financial institutions, planning and analysis, capital allocation, SEC communications and reporting, accounting, acquisitions and turnarounds. He is experienced in effectively managing complex projects, building professional relations and developing staff. Mr. McGroarty was previously employed as CFO of Safeguard Scientifics, Interim Controller at Cephalon, Inc., Vice President-Financial Planning and Analysis of Exide Technologies, Inc., and Senior Manager at PWC. His MBA is from the Wharton School of Business.

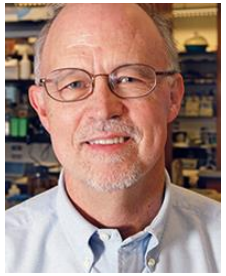
Feng Chang, PhD, VP of Research

Dr. Chang is an experienced neuroscientist with more than a decade of experience in neurodegenerative diseases, with broad scientific knowledge and hands-on experience. Prior to joining Annovis, she was a scientific solution consultant with Clarivate Analytics where she worked on cutting-edge scientific projects with top-50 pharma clients. Previously, Dr. Fang was business development manager for Coriell Institute for Medical Research and an assistant professor at Boston University, where she designed and supervised projects focused on prion diseases and AD as a research team leader.

William Mobley, MD, PhD, Chief Scientific Advisor

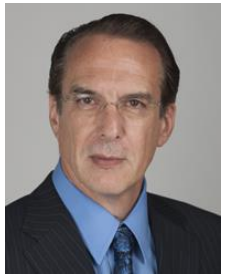
Distinguished Professor, Department of Neurosciences Florence Riford Chair for Alzheimer Research and Associate Dean for Neurosciences Initiatives at UC San Diego. He is a member of the National Academy of Medicine. His research focuses on the neurobiology of neurotrophic factor actions/signaling and on the hypothesis that malfunction of these mechanisms contribute to neuronal dysfunction in developmental and age-related disorders of the neurosystem.

SCIENTIFIC ADVISORY BOARD



Sidney Strickland, PhD, Chairman

Vice President and Dean for Educational Affairs and Research Professor, Patricia and John Rosenwald Laboratory of Neurobiology and Genetics at Rockefeller University. Dr. Strickland's laboratory investigates how dysfunction of the circulatory system contributes to Alzheimer's and other neurodegenerative disorders. He will serve as the Chairman of Annovis Bio's SAB.



Jeffrey Cummings, MD

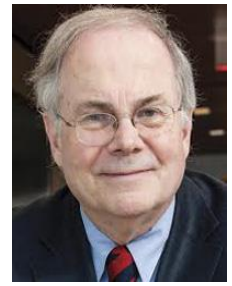
Dr. Cummings completed Neurology residency and a Fellowship in Behavioral Neurology at Boston University, Massachusetts. US training was followed by a Research Fellowship in Neuropathology and Neuropsychiatry at the National Hospital for Nervous Diseases, London, England. Dr. Cummings was formerly Professor of Neurology and Psychiatry, Director of Alzheimer's Disease Research and Director of the Center for Neurotherapeutics at UCLA. He was Director of the Cleveland Clinic Lou Ruvo Center for Brain Health in Las Vegas, Cleveland and Florida.

William Mobley, MD, PhD

Dr. Mobley is Distinguished Professor, Department of Neurosciences Florence Riford Chair for Alzheimer Research and Associate Dean for Neurosciences Initiatives at UC San Diego. He is a member of the National Academy of Medicine. His research focuses on the neurobiology of neurotrophic factor actions/signaling and on the hypothesis that malfunction of these mechanisms contribute to neuronal dysfunction in developmental and age-related disorders of the neurosystem.

Gregory Petsko, PhD

Dr. Petsko is a member of the National Academy of Sciences, the National Academy of Medicine, the American Academy of Arts and Sciences and the American Philosophical Society. His research interests are directed towards understanding the biochemical bases of neurological diseases like Alzheimer's, Parkinson's, and ALS discovering treatments (especially by using structure-based drug design), that could therapeutically affect those biochemical targets, and seeing any resulting drug candidates tested in humans. He has also made key contributions to the field of protein crystallography.



Rudolph E. Tanzi, PhD

Dr. Tanzi has published over 500 research papers and has received the highest awards in his field, including the Metropolitan Life Foundation Award, Potamkin Prize, Ronald Reagan Award, Silver Innovator Award, and many others. He was named to TIME magazine's list of TIME100 Most Influential People in the World (2015), and received the Smithsonian American Ingenuity Award, the top national award for invention and innovation. He co-authored the popular trade books "Decoding Darkness", New York Times bestseller, "Super Brain", and international bestseller "Super Genes".

BOARD OF DIRECTORS

Michael B. Hoffman
Chairman

Mr. Hoffman is the Founder and Managing Partner of Stone Capital Partners, a private equity firm focused on power and renewable energy. He was Partner of Riverstone, senior managing director at the Blackstone Group and managing director at Smith Barney, Harris Upham & Co. He serves as President of Northern Genesis Acquisition Corporation (II and III) and is a Trustee of The Rockefeller University.

Maria L. Maccecchini, PhD
Executive Board Member

Dr. Maccecchini founded Annovis in May 2008 to develop better therapeutics for Alzheimer's, Parkinson's and other neurodegenerative diseases. She was the Founder and CEO of Symphony Pharmaceuticals/Annovis, a company focused on protecting brain cells after stroke which was sold in 2001 to Transgenomic.

Reid S. McCarthy

Mr. McCarthy is experienced in corporate financial management, operations and new venture development. He was CFO of Topaz Pharmaceuticals, Inc. until its sale in 2011 to Sanofi Pasteur. He also served as CFO of JJ Haines & Company, Inc. and provided consulting CFO services to several life sciences companies. He has been a founding executive of several venture capital-backed companies which were successfully sold.

Claudine E. Bruck, PhD

Dr. Bruck is a pharmaceutical executive and scientist with strong entrepreneurial drive. Exhibited successes in building a therapeutic research unit de novo and leading discovery and clinical development of biological (vaccines, biopharmaceuticals) and small molecule medicines as well as an ophthalmic drug portfolio. With creativity and a strong results-focus, she is energized to challenge and lead teams. Extensive Pharmaceutical industry experience spans drug discovery and development across several therapeutic areas.

Mark White

Mr. White is a biopharmaceutical executive with global marketing, business development and sales experience. Currently, he is an independent consultant and a member of Robin Hood Ventures, a Philadelphia based angel investor group. Previously, Mr. White held senior level roles at Pfizer in marketing and commercial development, where he led the successful global launches of Inspira, Revatio, Lyrica and Xeljanz. In his last position, he was Vice President Worldwide Marketing, with global responsibility for new product development and in-line marketing for Pfizer's Inflammation Therapeutic Area.

SUMMARY

A novel approach to treat neurodegeneration is desperately needed

- The markets for AD and PD drugs are in the multibillions of dollars and growing
- Annovis has a novel approach to stop AD and PD
- ANVS401 shows improvements in Phase 2a clinical trials:
  - Cognition in AD patients
  - Motor function in PD patients
  - WAIS coding in AD and PD patients
- The successful completion of our Phase 2 clinical trials is providing validation of our approach in two diseases and allow us to move to Phase 3 trials in both diseases

Additional Slides

CHANGE IN CAUSES OF DEATH FROM 2000 TO 2018

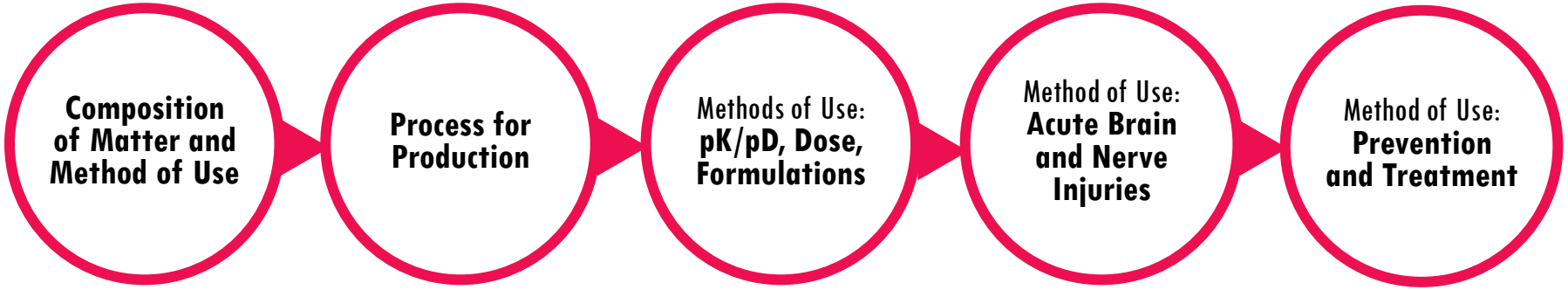
• Breast Cancer	- 13%
• Colon Cancer	- 21%
• Heart Disease	- 21%
• Stroke	- 24%
• HIV	- 67%
• Parkinson's	+ 84%
• Alzheimer's	+ 112%

PIPELINE

Therapy	Diseases/Conditions	PRE-CLINICAL	IND	PHASE I	PHASE II	PHASE III
ANVS401 Oral drug for chronic indications	AD					
	PD					
	AD-DS					
	FTD					
	CTE					
ANVS405 Injectable drug for acute traumatic events	TBI					
	Stroke					
ANVS301 Oral drug for advanced AD and dementia	Advanced AD					

CORPORATE PATENT ESTATE

Multi-layer strategy



Patent/Application	Subject Matter	Status	Expiry
Provisional	ANVS401 to treat viral and bacterial infections of the brain, including Covid19	Pending	**2042**
PCT	ANVS401 and 405 – Mechanism of Action for prevention and treatment of diseases	Pending	**2038**
PCT	ANVS405 - Acute brain and nerve injuries	Granted – Europe and Japan	**2036**
PCT	ANVS401 - pK/pD, low doses, formulations Neurodegenerative Diseases	Granted – US and Europe	**2031**
In-licensed patents	Composition of matter, manufacturing, method for treating AD and DS	Granted	**2022-25**

ANNOVIS

Improves **THE FLOW** of Axonal Transport in Alzheimer's Disease and Neurodegeneration

ANVS401 ANVS405 ANVS301

Symbol: **ANVS** (NYSE American)

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