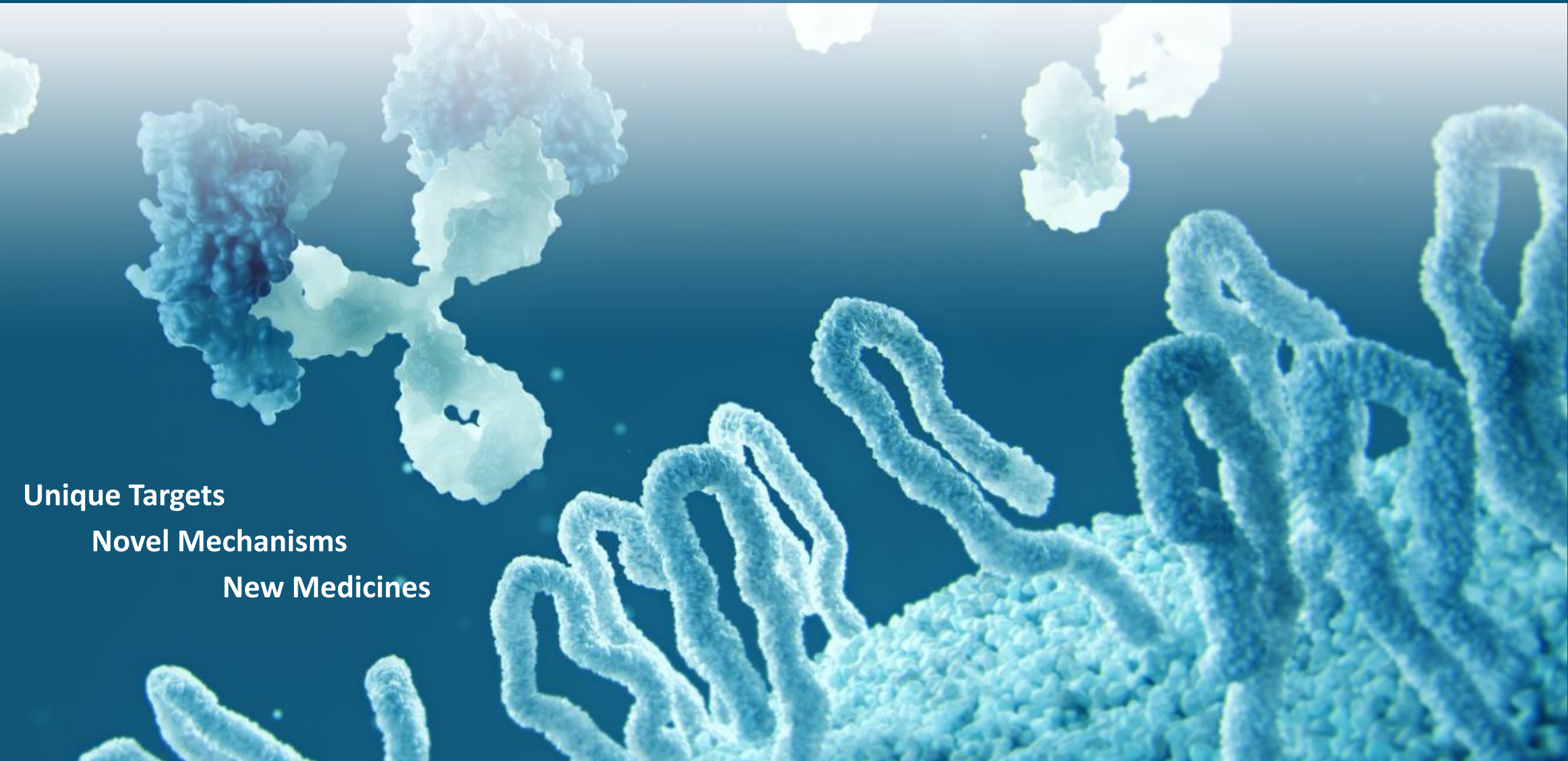
VX15 (pepinemab) Antibody
Treatment for Cancer and Neurodegenerative Disease



Unique Targets

Novel Mechanisms

New Medicines

Forward Looking Statements

To the extent that statements contained in this presentation are not descriptions of historical facts regarding Vaccinex, Inc. ("Vaccinex," "we," "us," or "our"), they are forward-looking statements reflecting management's current beliefs and expectations. Words such as "may," "will," "expect," "anticipate," "estimate," "intend" and similar expressions or their negatives (as well as other words and expressions referencing future events, conditions, or circumstances) are intended to identify forward-looking statements. Examples of forward-looking statements in this presentation include, among others, statements regarding: (i) the timing and success of the commencement, progress and receipt of data from preclinical and clinical trials; (ii) our expectations regarding the potential safety, efficacy or clinical utility of our product candidates; (iii) the expected results of any clinical trial and the impact on the likelihood or timing of any regulatory approval; (iv) financial and financing expectations and opportunities and (v) the performance of third parties.

Forward-looking statements in this presentation involve substantial risks and uncertainties that could cause our research and pre-clinical development programs, clinical development programs, future results, performance, or achievements to differ significantly from those expressed or implied by the forward-looking statements. Such risks and uncertainties include, among others, uncertainties inherent in the execution, cost and completion of preclinical and clinical trials, risks related to our dependence on third parties, uncertainties related to regulatory approval, risks related to our dependence on our lead product candidate VX15 (pepinemab), risks related to competition, other matters that could affect our development plans or the commercial potential of our product candidates, expectations regarding the use of proceeds from our initial public offering, changes in costs and operations, and other risks regarding our capital requirements and results of operations. Except as required by law, we assume no obligation to update these forward-looking statements, or to update the reasons why actual results could differ materially from those anticipated in the forward-looking statements, even if new information becomes available in the future. For a further discussion of these and other factors that could cause future results to differ materially from any forward-looking statement, see the section titled "Risk Factors" in our quarterly report on Form 10-Q filed with the Securities and Exchange Commission ("SEC") and the other risks and uncertainties described in our prospectus for our initial public offering dated August 9, 2018, filed with the SEC pursuant to Rule 424(b) under the Securities Act of 1933, as amended,.

No representations or warranties are offered in connection with the data or information provided herein. This presentation is intended for informational purposes only and may not be relied on in connection with the purchase or sale of any security. Any offering of our securities will be made, if at all, only upon the registration of such securities under applicable securities laws or pursuant to an exemption from such requirements.

Novel antibody therapeutics target unmet needs

✓ **Novel Mechanistic Approach**

Lead product: Pepinemab

Humanized IgG4 monoclonal antibody, first in class

Blocks a unique target, Semaphorin 4D (SEMA4D)

✓ **Advanced clinical programs with near term opportunities for monetization by partnering**

✓ **Proprietary Drug Discovery Platform**

ActivMAb Technology

SEMA4D signals through receptors (PLXNB1/B2) to trigger collapse of cell's cytoskeleton

The cell cytoskeleton regulates process extensions which enable cellular movement and direct cell to cell interactions

SEMA4D/PLXN pathways are activated in immune and central nervous systems in response to stress/injury

Cancer Immunotherapy

In cancer, tumors utilize these pathways to restrict movement of immune cells into the tumor microenvironment

Neurodegenerative Disease

In brain, this pathway is upregulated in response to damage and triggers loss of normal functions and transition to chronic neuroinflammation, which is believed to aggravate neurodegeneration

Clinical Pipeline

Research/Preclinical	Phase 1	Phase 2	Phase 3

Pepinemab Antibody Platform (anti-Semaphorin 4D Mab)

Neurology

Pepinemab in Huntington's Disease (Orphan Drug and Fast Track Designations) — **Complete** — SIGNAL

Pepinemab in Alzheimer's Disease — Initiated 2021 — SIGNAL-AD

Oncology

Pepinemab COMBO with avelumab in NSCLC (Collaboration with Merck KGaA, Darmstadt, Germany) — **Complete** — **CLASSICAL – Lung**

Pepinemab COMBO with pembrolizumab in first-line HNSCC — Initiated 2021 — **KEYNOTE B84**

ActivMAb® Antibody Platform

ActivMAb Technology

Drug Discovery, Complex Membrane Protein Receptors

VX5 (Anti-CXCL13) for Autoimmune Diseases and Cancer

All studies Sponsored by:

Additional Funding &/or Support by:

Alzheimer's Drug Discovery Foundation

Merck KGaA, Darmstadt

Merck, MSD

ASSET: PEPINEMAB NEURODEGENERATIVE DISEASE

Pepinemab Mechanism of Action

- SEMA4D is upregulated in response to stress/disease to trigger neuroinflammatory gliosis
- Pepinemab blocks chronic glial activation and restores their normal support functions.

Broadly applicable approach - does not target disease-specific insult, instead targets common trigger of neuroinflammation which contributes to and amplifies neurodegeneration

STATUS		SUMMARY
Phase 2 Huntington's Disease Complete 2020 Double-blind, Placebo-controlled	Sponsored by: VACCINEX Granted Orphan Disease and Fast Track Designation by FDA	Huntington's Disease (SIGNAL) • Well tolerated • Cognitive benefit to patients • Reduced brain atrophy (vMRI) and restored loss of metabolic activity (FDG-PET) • *Phase 3-ready asset*
Phase 1b/2a Alzheimer's Disease Initiated Q2 2021 Data expected late 2022/early 2023 Double-blind, Placebo-controlled	Sponsored by: VACCINEX Funding by: alzheimer's association; Alzheimer's Drug Discovery Foundation	Alzheimer's Disease (SIGNAL-AD) • Primary endpoint: Safety • Key efficacy endpoints: Cognition and metabolic activity

Currently exploring pharma collaboration in HD and AD

ASSET: PEPINEMAB IMMUNO-ONCOLOGY

Pepinemab Mechanism of Action

Overcome Immune resistance

- neutralizes the SEMA4D barrier at the tumor boundary to facilitate movement of anti-tumor immune cells
- Inhibits immune suppressor cells

Novel and independent mechanism → Synergy with immune checkpoint therapy

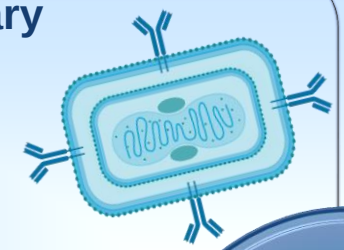
Well tolerated

STATUS		SUMMARY
Phase 1b/2 Non Small Cell Lung Cancer (NSCLC) Complete 2020 Data published in Clinical Cancer Research, 2021 Pepinemab Combination with Bavencio ™	Sponsored by: VACCINEX Co-funded by: EMD Serono/Merck KGaA, Darmstadt MERCK	CLASSICAL-Lung • Well tolerated • Anti-tumor activity in some patients whose cancer was resistant to prior therapy with single-agent checkpoint inhibitors • Anti-tumor activity in some patients with challenging PD-L1 negative or low tumors • Increased penetration of cytotoxic T cells following treatment
Phase 1b/2 Head and Neck Cancer (R/M HNSCC) Initiated Q2 2021 Data expected mid 2022 Pepinemab Combination with Keytruda ™	Sponsored by: VACCINEX Drug provided by: Merck, MSD MERCK	Head and Neck Cancer (Keynote B84) • High levels of myeloid derived suppressor cells (MDSC) that are induced by SEMA4D and a source of resistance to immune checkpoint therapy

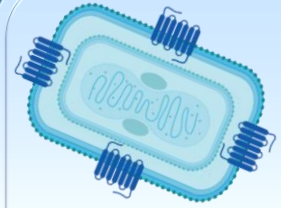
ASSET:
ActivMab Discovery Solutions

Antibody Library

Diverse Antibody Library (10^10) Screened as Human IgG in Mammalian Cells



Antigen Virus



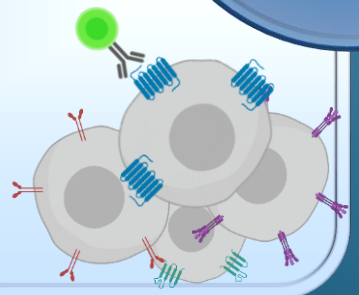
Virions expressing Complex Membrane Proteins for Antibody Discovery

ActivMAb Technology

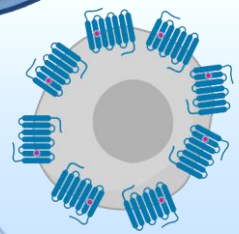
Discovery Solutions

Target Identification

Large cDNA library expressed in Mammalian Cells



Protein Optimization



Mammalian expression of mutant libraries to enhance expression

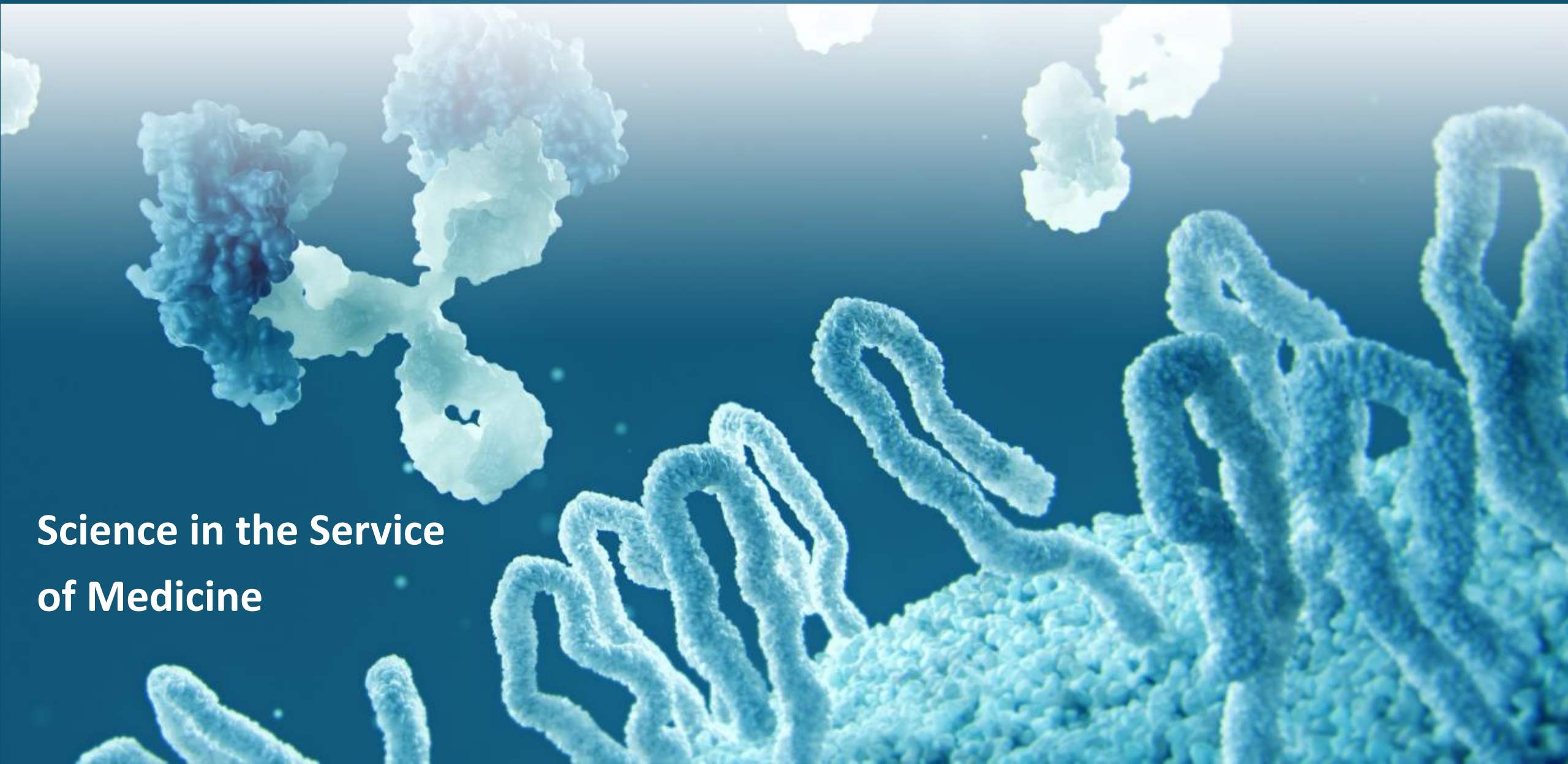
Poxvirus based antibody and protein engineering solutions

Unique capability for selection of high value antibodies against hard-to-target multi-pass membrane receptors
(i.e. GPCRs, ion channels)

Sustainable engine for value creation through pipeline expansion and strategic collaborations

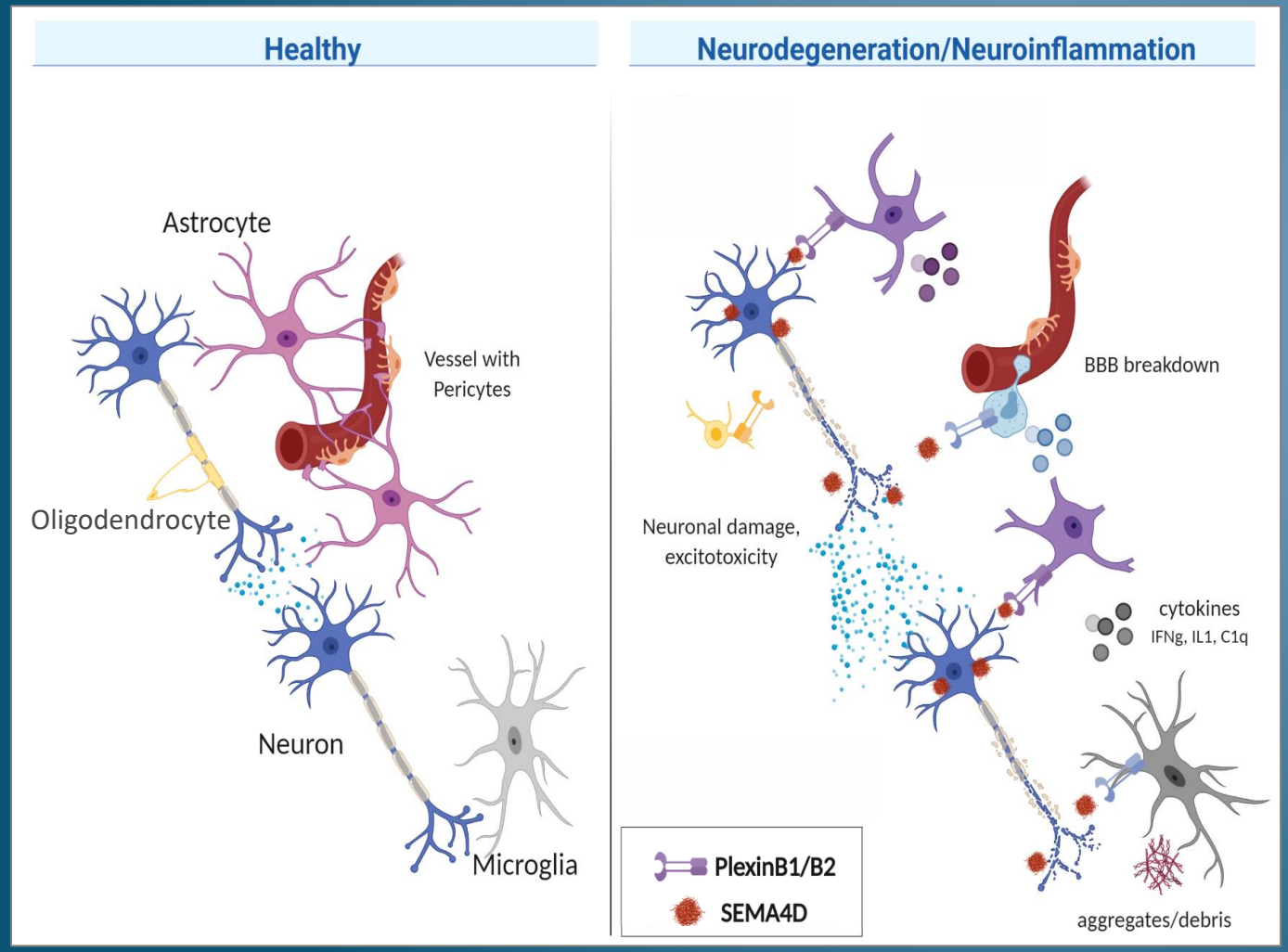
Active collaborations with two major pharma and multiple biotech partners

Pepinemab Antibody
Neurodegenerative Disease



Science in the Service of Medicine

Glial cells respond to damage in the brain



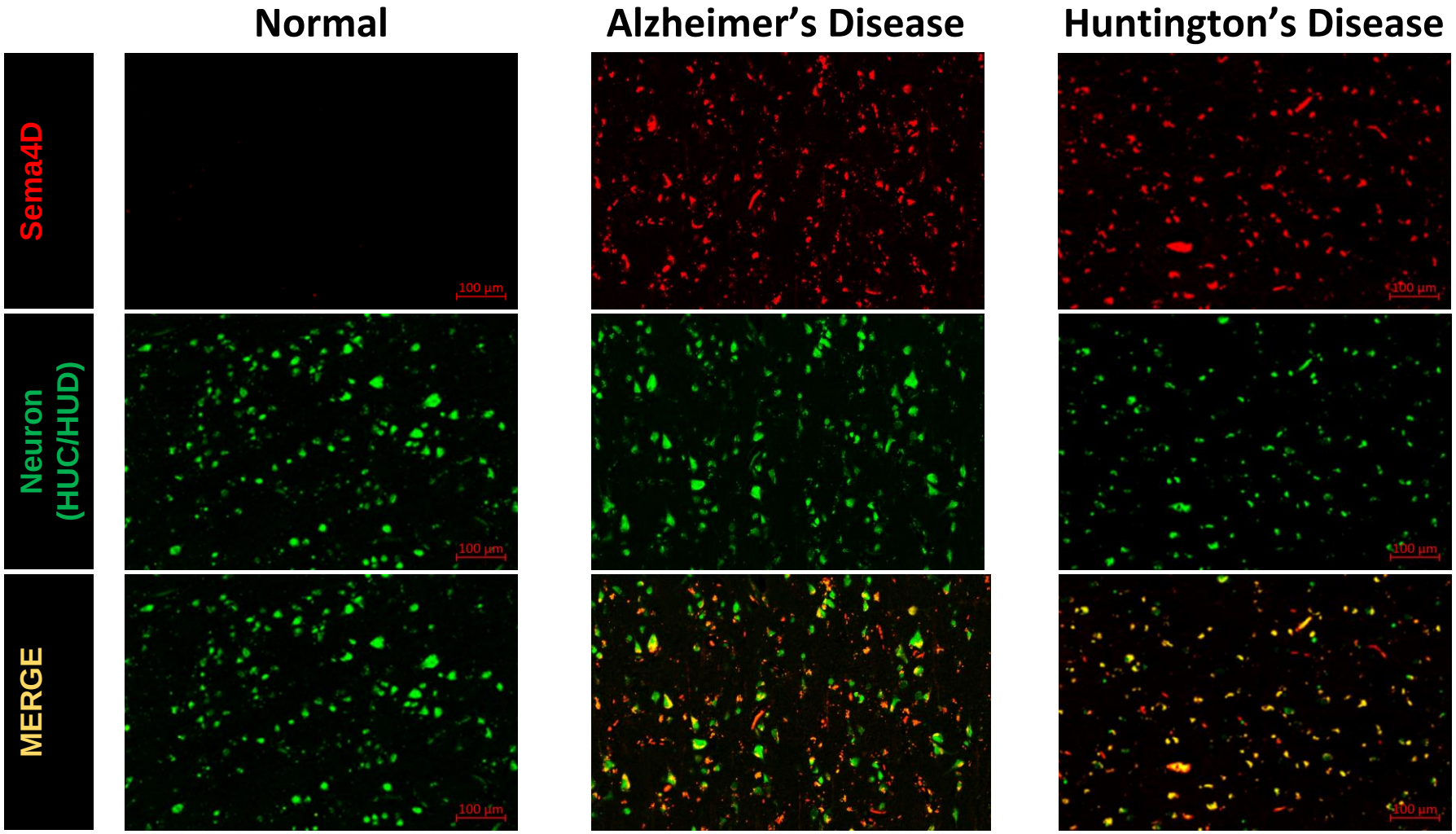
SEMA4D is upregulated on damaged neurons

SEMA4D binding to Plexin receptors on glial cells to triggers collapse of cytoskeleton and transformation to reactive inflammatory state

Chronic activation contributes to and exacerbates neurodegeneration

Smith et al. 2014 Neurobiology of Disease
Southwell et al. 2015. Neurobiology of Disease
Schematics created with BioRender.com

SEMA4D is upregulated in neurons during Human AD and HD disease progression



Human autopsy sections of frontal lobe

Huntington's is a Genetically Inherited Disease

HD is caused by dominant mutation in a single gene.

There are currently no approved treatments to alter the course of Huntington's Disease.

Estimated patient population in the US is ~40,000 individuals with manifest disease and >150,000 at risk of having inherited the HD mutation.

Neuronal degeneration, neuroinflammation, and severe atrophy is observed in multiple brain regions affecting cognition, emotion, and motor function.

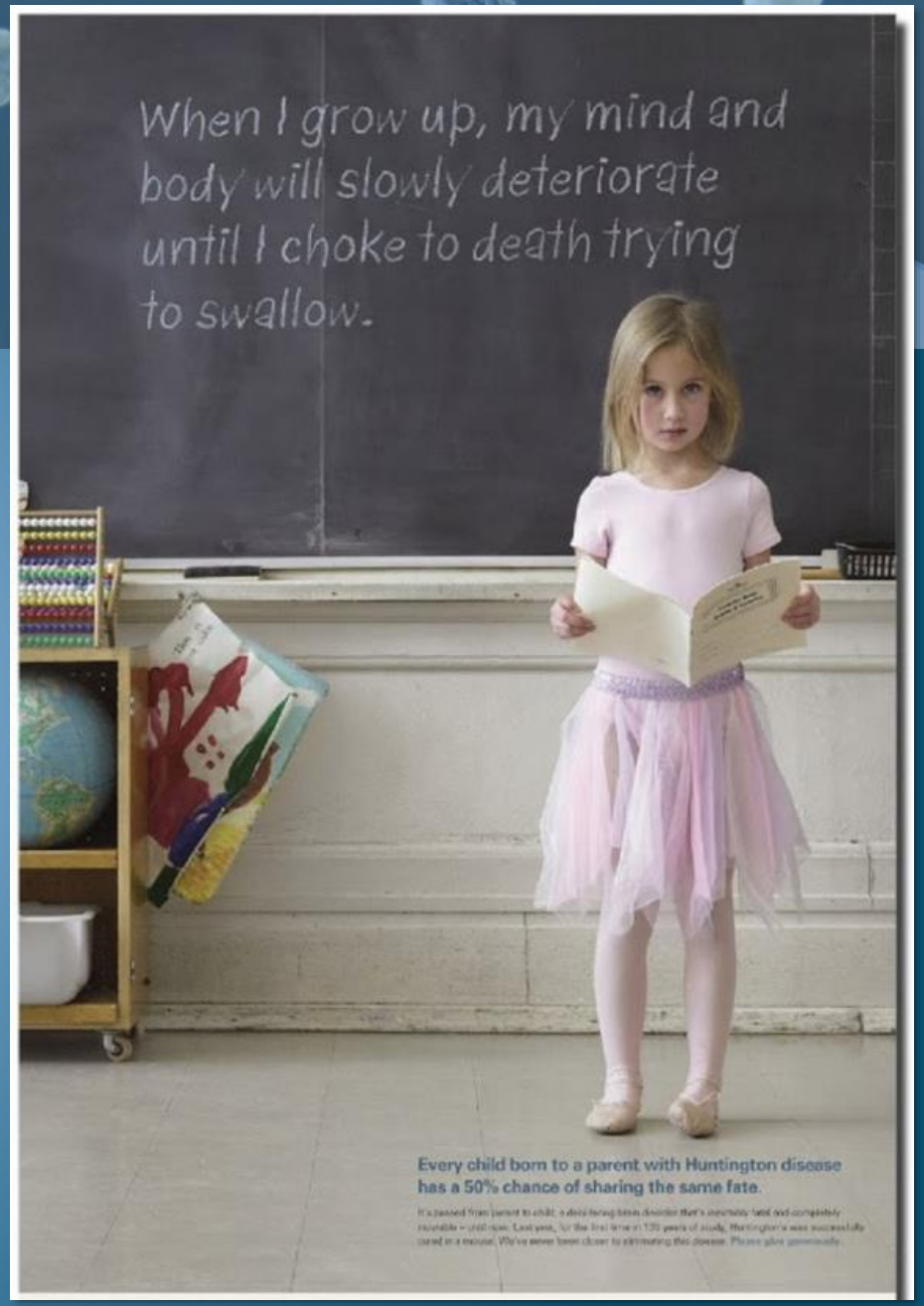
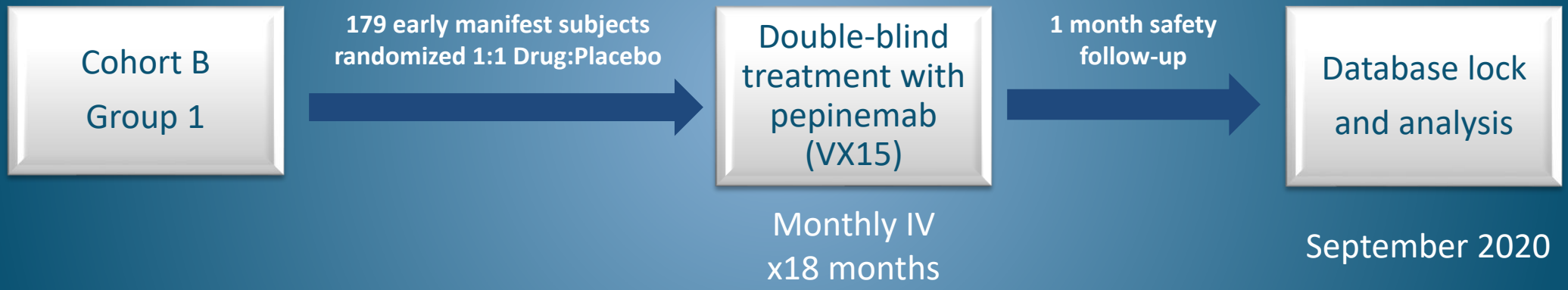


Photo credit: Huntington Society of Canada

Clinical Trial Design – Group B1, Early Manifest HD

Orphan Disease and Fast Track designations



Study Objectives

- Safety and tolerability
- Cognitive Function and Clinical global impression of change (CGIC)
- Brain imaging measures

Abbreviated Baseline Characteristics and Safety – Cohort B1, ITT population

Pepinemab (PEPI) SEMA4D blocking antibody is well tolerated.

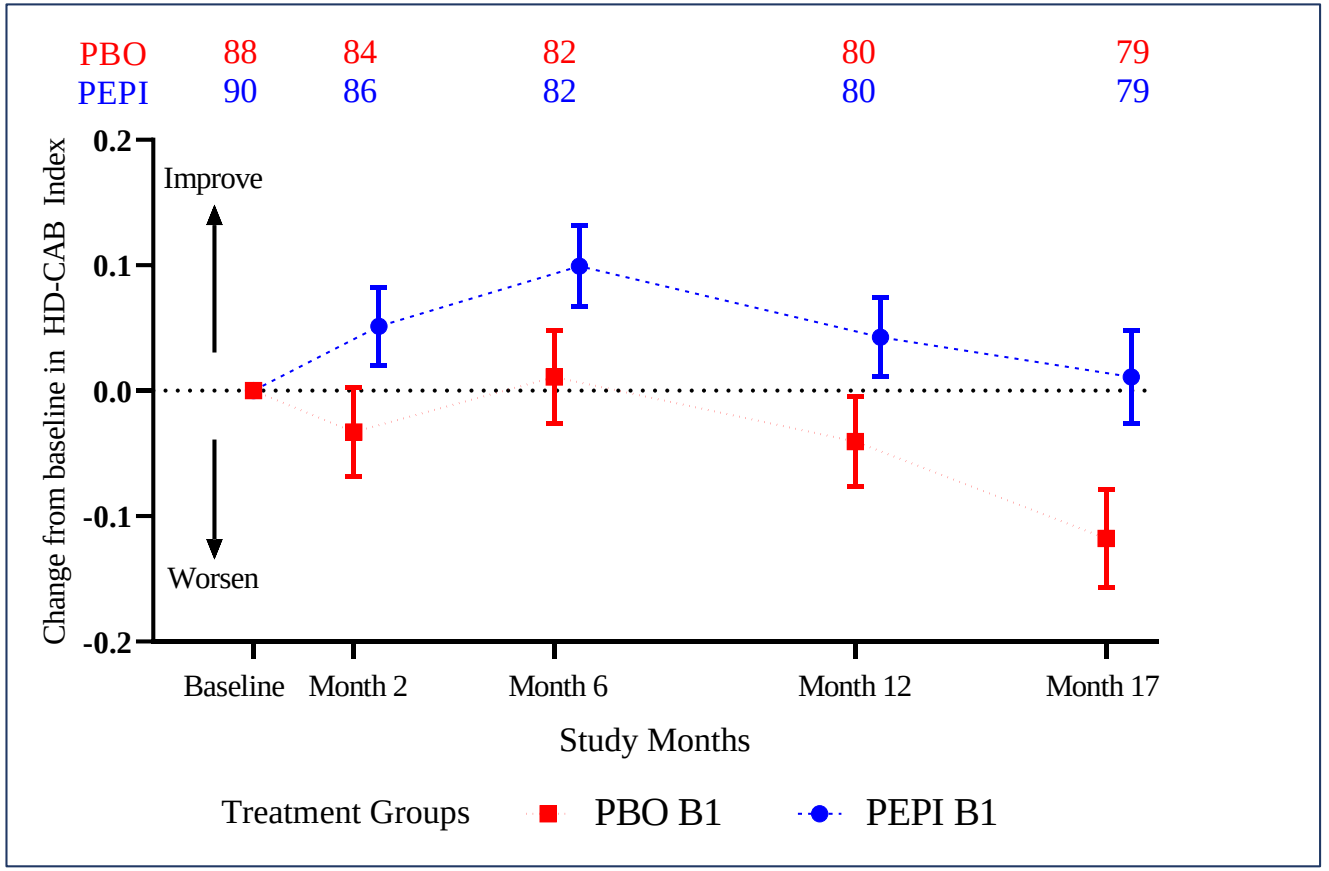
	Cohort B1 (N=179)	
	PBO (N=88) Placebo	**PEPI (N=91) Pepinemab**
Discontinued Treatment Early	**10**	**13**
Had Any SAE (*)	**8**	**4**
Had Any Grade 3+ AE (*)	**14**	**17**
CAG repeat length	44.1 (3.8)	43.5 (3.1)
CAP score (**)	470 (96)	466 (85)
UHDRS-DCL at screening, n(%)		
DCL-4, Unequivocal HD (>99% confident)	88 (100%)	91 (100%)

*pre-COVID era; **CAP score = age × (CAG repeat length – 33.66)

Cognitive Assessment Battery (HD-CAB)
Prespecified exploratory analysis – Early Manifest HD

HD-CAB Composite Index of 6 Cognitive Assessments

Early manifest HD



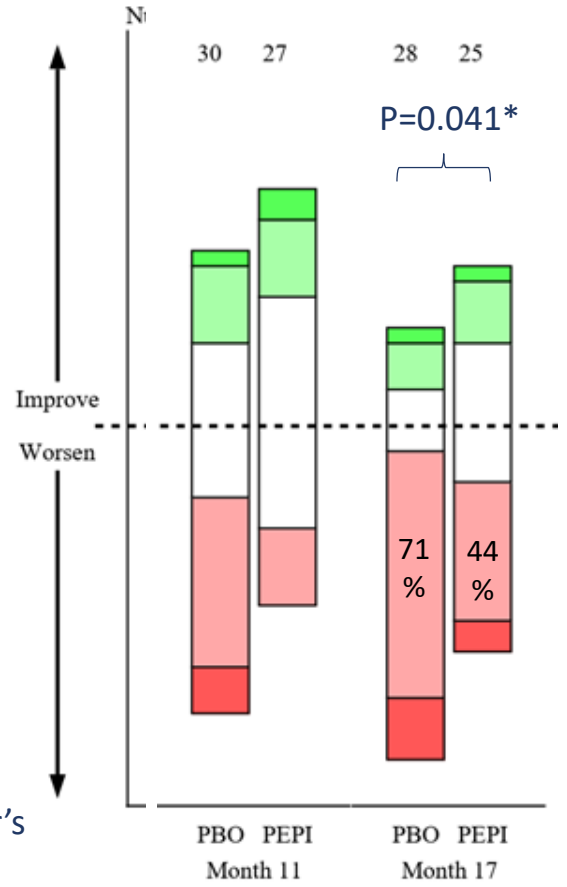
One-sided p-value	Favors PEPI	Critical value
0.007	Yes	Yes [0.025]

Clinical Global Impression of Change (CGIC) Post-hoc Subgroup Analysis – Early Manifest HD

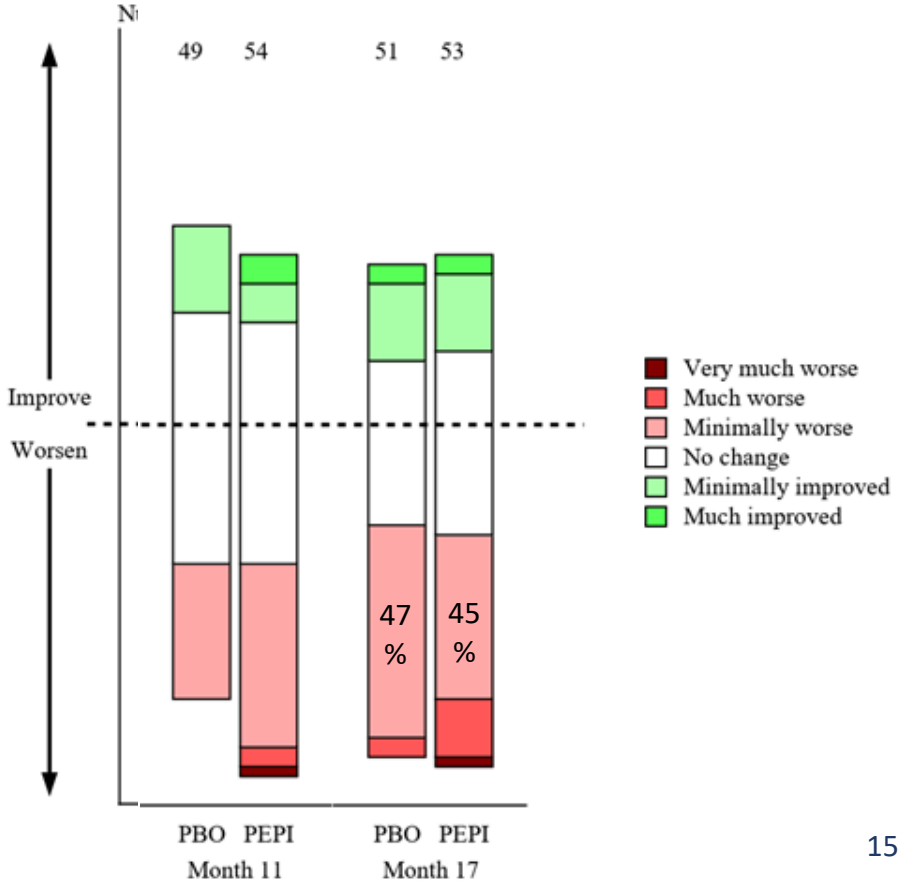
Subjects were less likely to experience decline in CGIC following treatment with pepinemab compared to placebo.

This difference was evident in subjects with somewhat more advanced disease (TFC 11).

CGIC – Subjects with Baseline UHDRS TFC 11

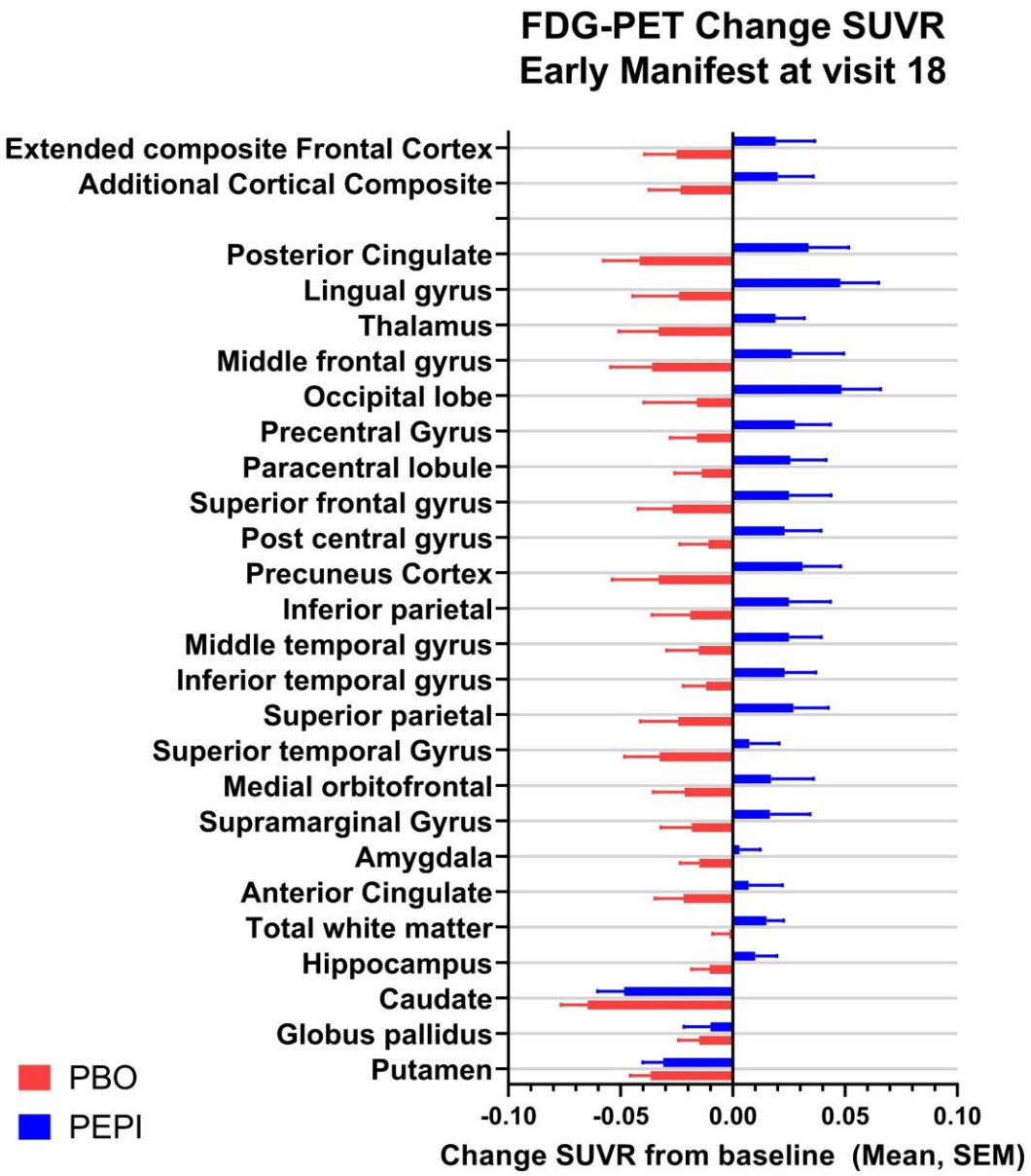


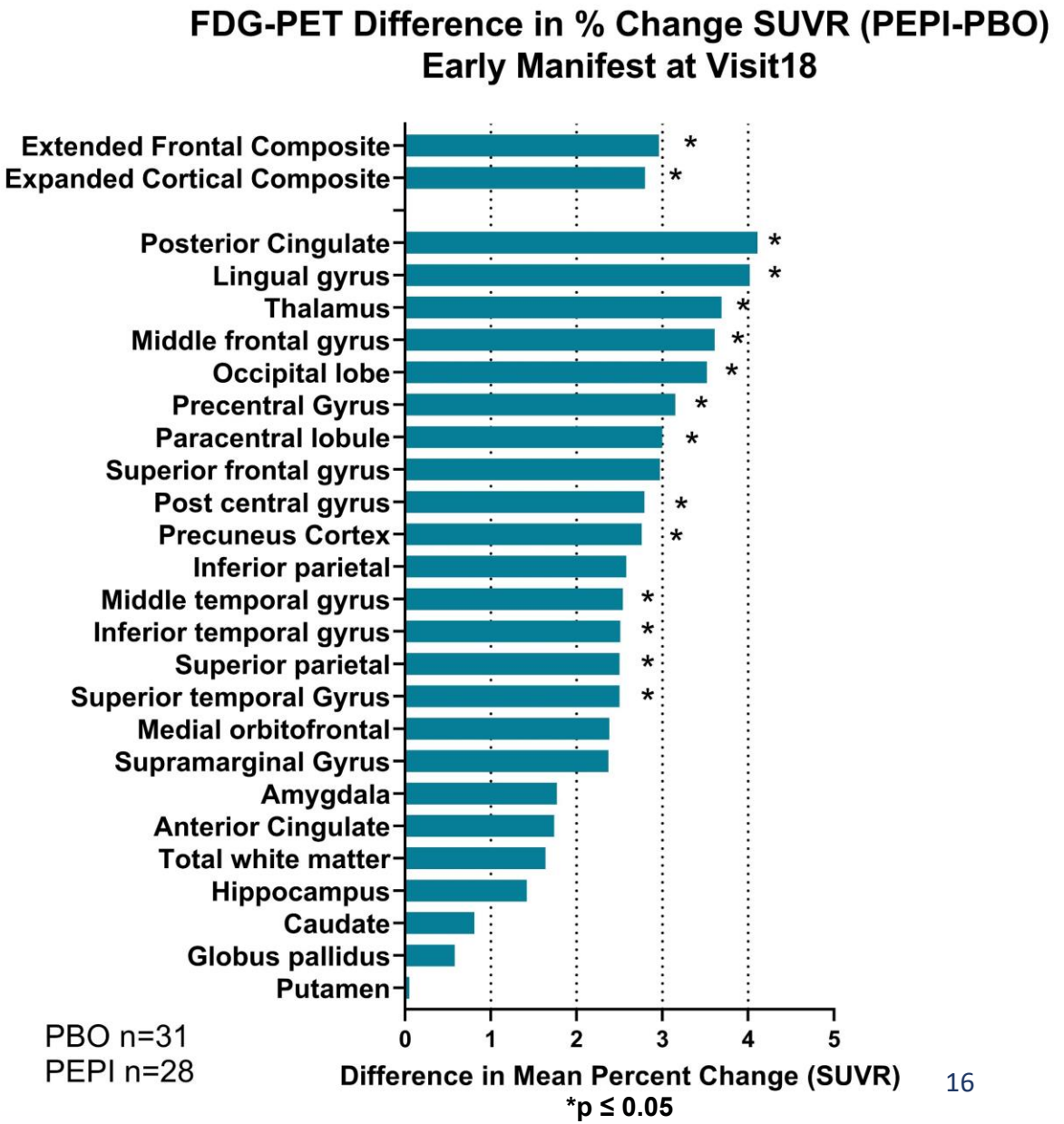
CGIC – Subjects with Baseline UHDRS TFC 12 and 13



*nominal one-sided p-value, Fisher's exact test for worsening score

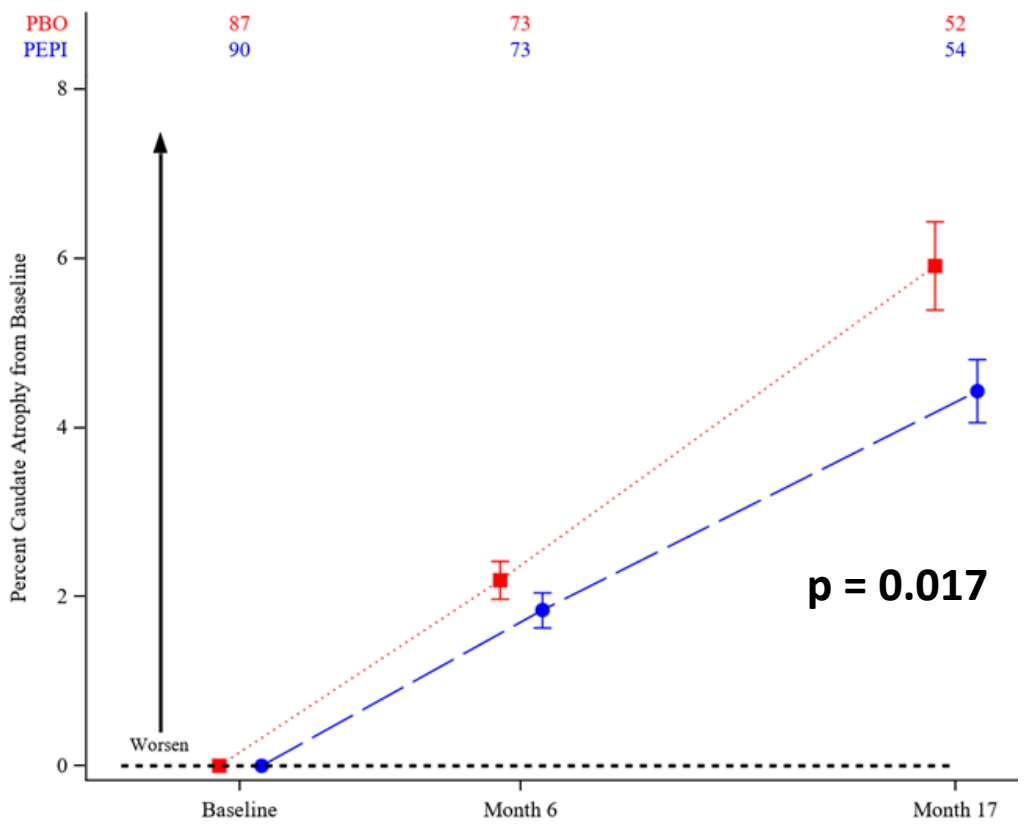
FDG-PET at 18 Months – Early Manifest HD: Pepinemab treatment reverses loss of metabolic activity



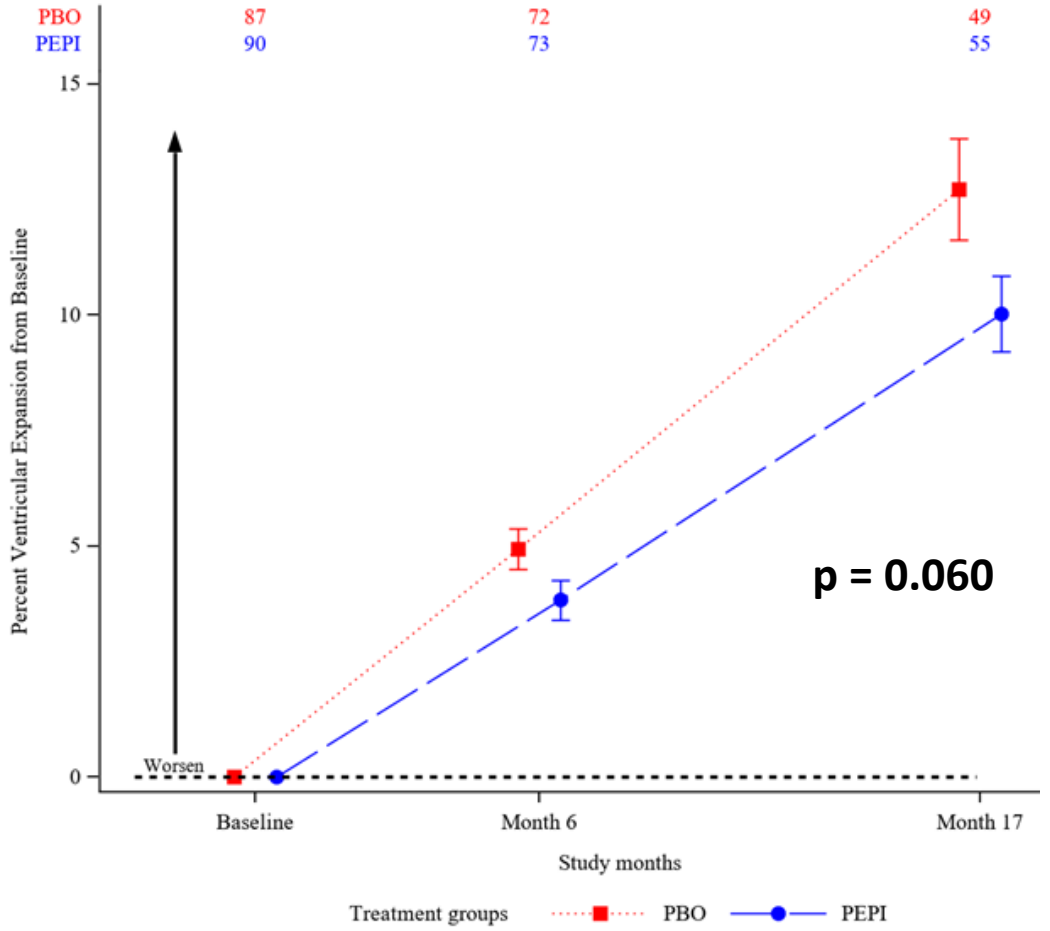


volumetric MRI analysis – Boundary Shift Integral
Pre-specified exploratory endpoint

CBSI (caudate atrophy) Early Manifest (B1)

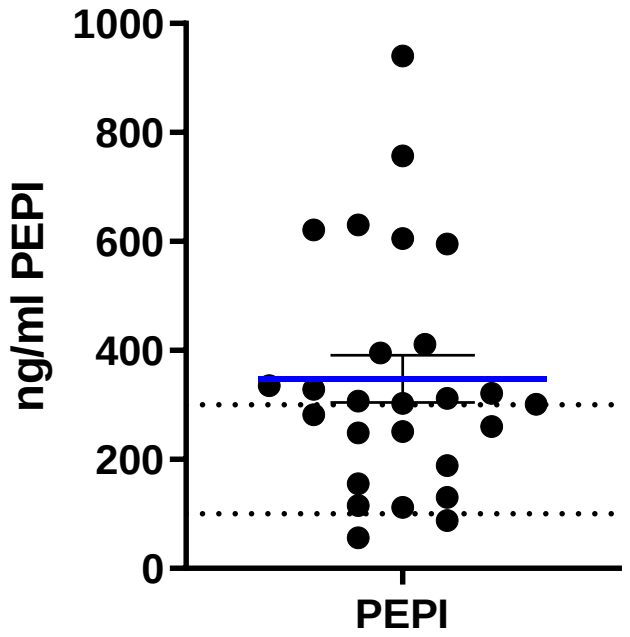


VBSI (ventricular expansion) Early Manifest (B1)

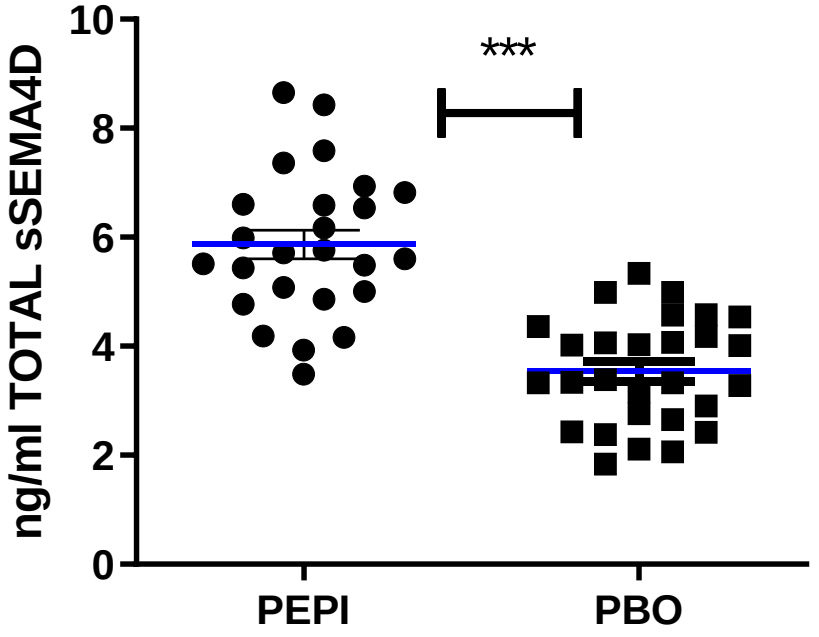


Pepinemab and sSEMA4D levels in cerebrospinal fluid (CSF)

Most subjects dosed with pepinemab have ≥ saturating levels (100-300 ng/ml) in CSF



sSEMA4D increases in subjects dosed with pepinemab – suggesting target engagement



SIGNAL: Early Manifest HD
Results of Phase 2 trial

Orphan Disease and Fast Track designations

Mechanism of Action: Reduce neuroinflammation and restore normal glia function

Safety and tolerability: Well tolerated
Intravenous administration

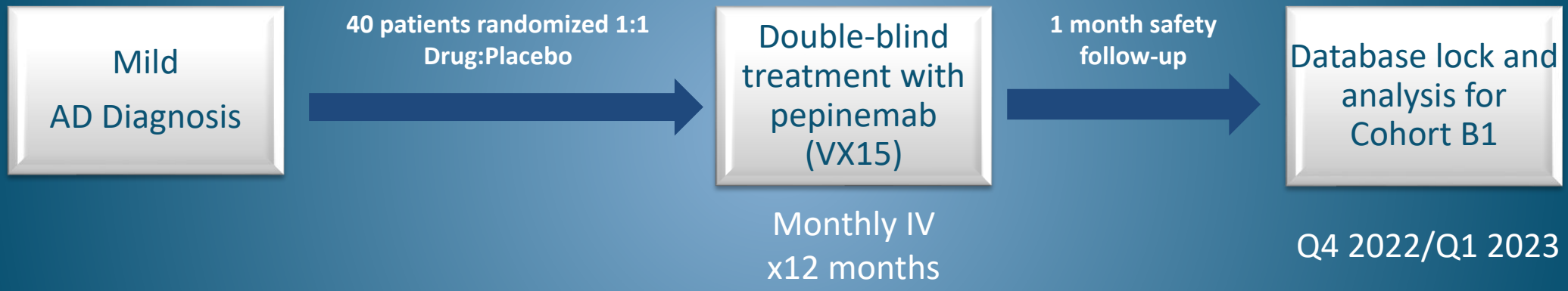
Clinical Efficacy (HD): Improved Cognitive Function and
Clinical global impression of change (TFC 11)
Reduced brain atrophy and increased metabolic activity

Target engagement: Confirmed penetration into CNS at expected level
Antigen-antibody complexes detected

Phase 3-ready asset for HD
Initiated Phase 1b/2a trial for AD
Broad application – targets common pathology in neurodegeneration

Clinical Trial Design
Alzheimer's Disease

SIGNAL-AD



Study Objectives

- Safety and tolerability
- Cognitive Function measures, CDR-SB, ADAS-Cog13
- Brain imaging measures, FDG-PET

Funding by

alzheimer's association

Alzheimer's Drug Discovery Foundation

Broad applications in neurological diseases

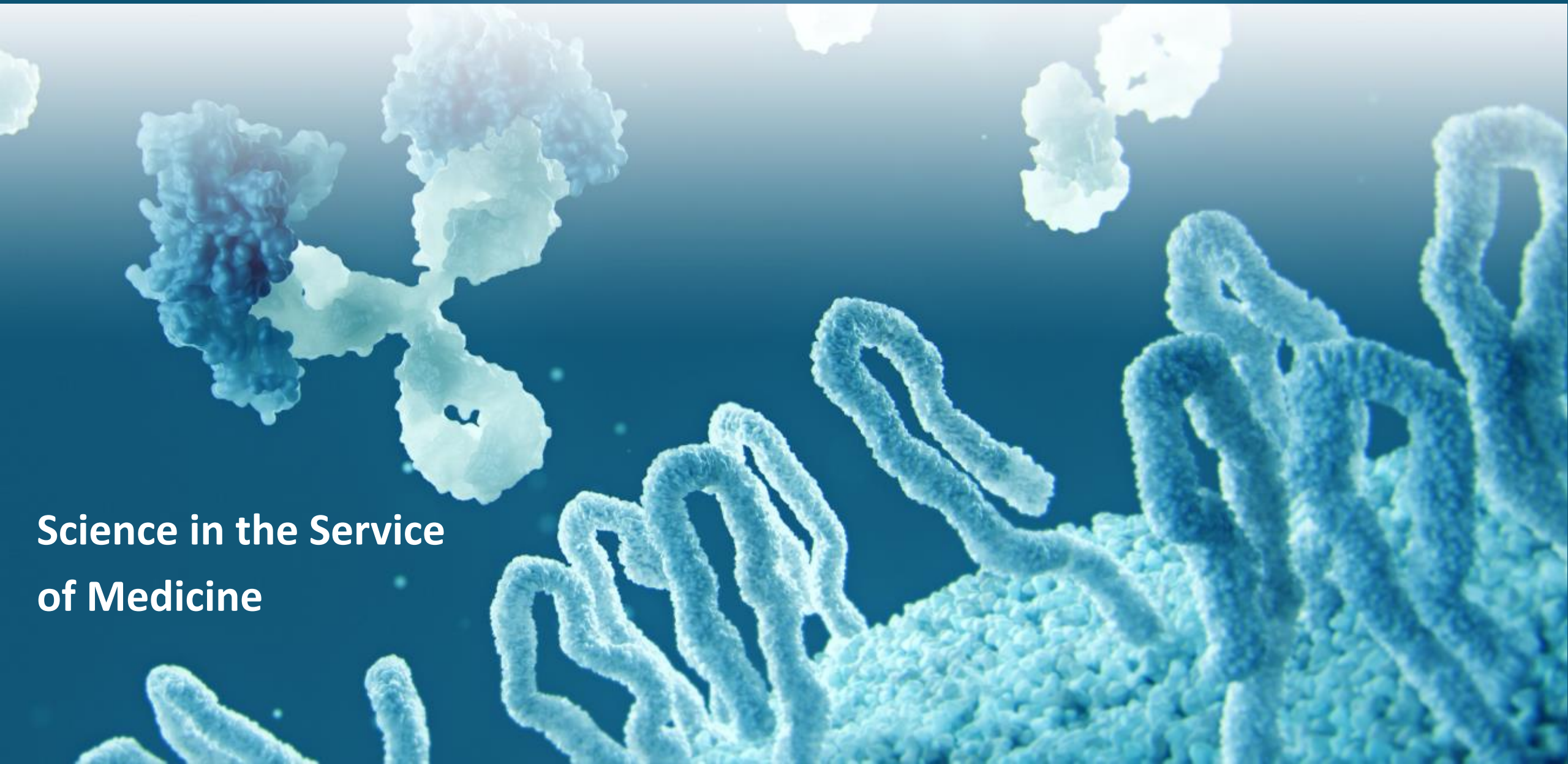
The mechanism of action of pepinemab has broad potential applications in neurological diseases that share common neuroinflammatory pathology, including HD, AD, FTD, PPMS.

Antibody blockade ameliorates neuropathology and symptoms of disease in multiple preclinical models

- Huntington's Disease – YAC128 transgenic model
  - Southwell et al. 2015. Neurobiology of Disease
- Alzheimer's Disease – CVN (APPSwDI/NOS2-/-) transgenic model
  - Available upon request
- Rett Syndrome - Mecp2$^{T158A/y}$ mutant transgenic mice
  - Available upon request
- Multiple Sclerosis – EAE models and lysolecithin-lesion model
  - Smith et al. 2014 Neurobiology of Disease

VACCINEX

Pepinemab Antibody
Cancer Immunotherapy



Science in the Service of Medicine

Pepinemab has a unique mechanism of action

Tumors have developed multiple resistance mechanisms to avoid destruction by immune system

- SEMA4D creates a barrier at tumor boundary to restrict movement of anti-tumor immune cells into the tumor
- SEMA4D also promotes recruitment and suppressive activity of myeloid derived suppressor cells (MDSC)

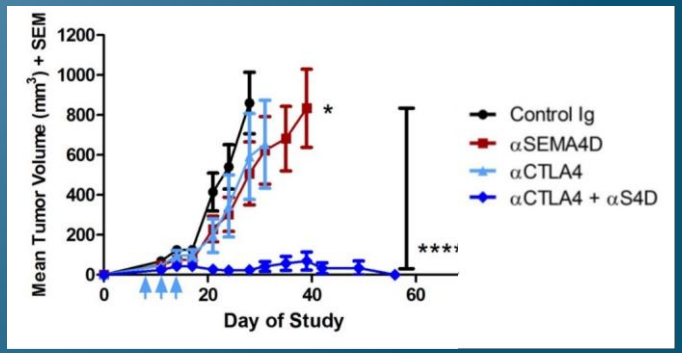
Pepinemab is a SEMA4D blocking monoclonal antibody with a unique mechanism of action to overcome immune resistance:

- Neutralizes SEMA4D barrier at the tumor boundary to facilitate immune infiltration
- Increases cytotoxic T cells
- Inhibits immune suppressor cells

Pepinemab complements other immune-activating therapies

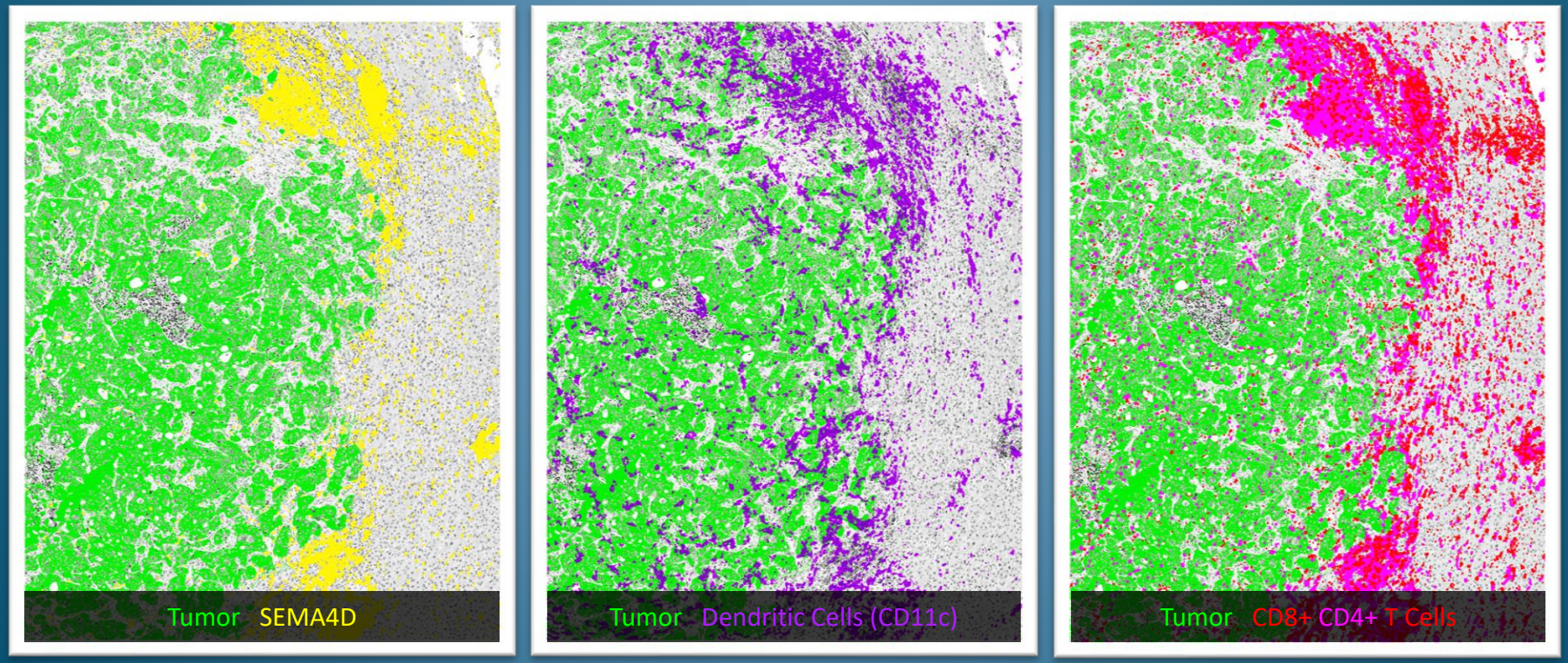
Immune Evasion Mechanisms	Therapy
Exclusion of anti-tumor immune cells	Pepinemab
Activation of suppressor immune cells	Pepinemab
Upregulation of immune checkpoint molecules (PD-1, CTLA-4)	Keytruda, Opdivo, Yervoy, etc

Combination therapy: Preclinical Data



SEMA4D expression and immune exclusion in human biopsies

Dissecting the tumor microenvironment with multiplex IHC



Pro-inflammatory cells are excluded from the metastatic CRC tumor and build up at the invasive edge

CD8 T cells align with Sema4D at the invasive edge of the tumor. Most of these excluded T-cells express Sema4D. Dendritic Cells can be seen within the tumor interior but is heavily excluded at the invasive edge.

Phase 1b/2 CLASSICAL-Lung Highlights

Sponsored by: ACCINEX

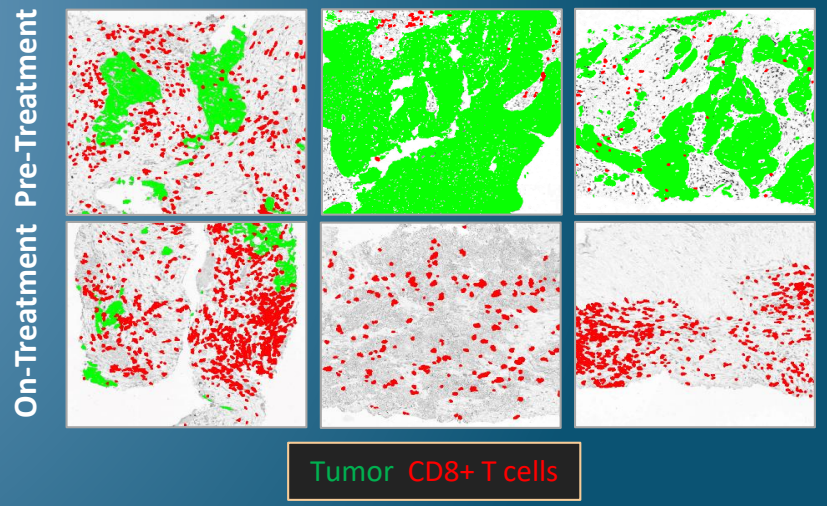
Co-funded by: MERCK

CLINICAL CANCER RESEARCH | CLINICAL TRIALS: IMMUNOTHERAPY

A Phase Ib/2 Study of Pepinemab in Combination with Avelumab in Advanced Non–Small Cell Lung Cancer AC

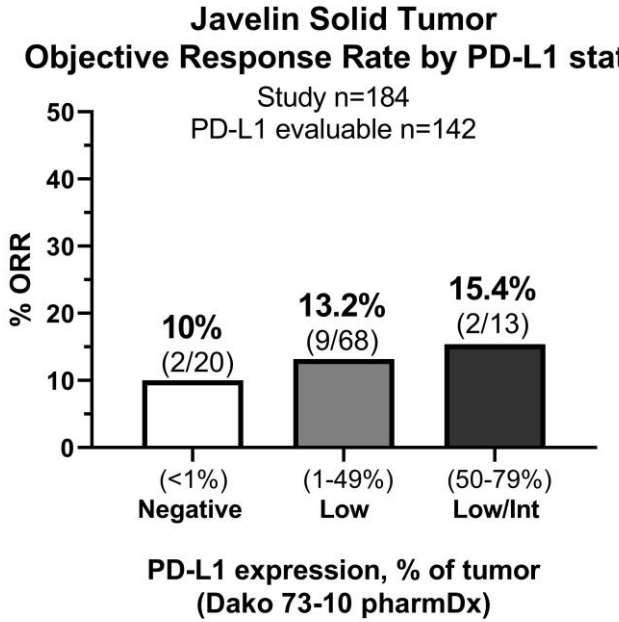
Michael Rahman Shafique[1], Terrence Lee Fisher[2], Elizabeth E. Evans[2], John E. Leonard[2], Desa Rae Electa Pastore[2], Crystal L. Mallow[2], Ernest Smith[2], Vikas Mishra[2], Andreas Schröder[3], Kevin M. Chin[4], Joseph Thaddeus Beck[5], Megan Ann Baumgart[6], Ramaswamy Govindan[7], Nashat Y. Gabrail[8], Alexander I. Spira[9], Nagashree Seetharamu[10], Yanyan Lou[11], Aaron Scott Mansfield[12], Rachel E. Sanborn[13], Jonathan W. Goldman[14], and Maurice Zauderer[2]

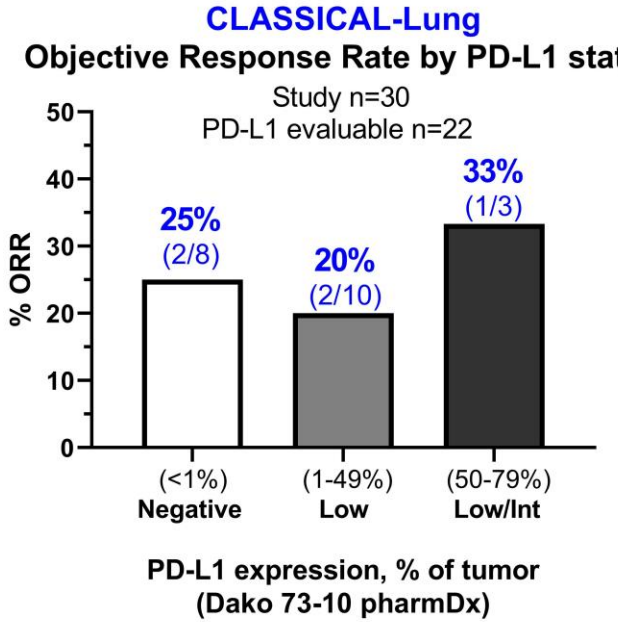
1. Well tolerated
2. Antitumor activity in some patients whose cancer was resistant to prior therapy with single-agent checkpoint inhibitors
   - Disease control rate: 59%, and 7/29 patients with durable responses ≥ 23 weeks
3. Antitumor activity in some patients with challenging PD-L1 negative or low tumors
   - Reported single agent anti-PDx: ORR ~10-15%
   - Combination with pepinemab: ORR 25-33%
4. Increased penetration of cytotoxic T cells following treatment



Phase 1b/2 CLASSICAL-Lung Objective Response Rate by PDL-1 Status (IO Naïve)

Combination therapy achieved a higher overall response rate in difficult to treat PD-L1-low population, compared to historical data in IO naïve patients with avelumab





1. Calculated from data published in:

Gulley JL, Rajan A, Spigel DR, et al. Avelumab for patients with previously treated metastatic or recurrent non-small-cell lung cancer (JAVELIN Solid Tumor): dose-expansion cohort of a multicentre, open-label, phase 1b trial. *Lancet Oncol* 2017; published online March 31. http://dx.doi.org/10.1016/S1470- 2045(17)30240-1.

Gulley JL, Rajan A, Spigel DR, et al. Exposure Response and PD-L1 expression analysis of second-line avelumab in patients with advanced NSCLC: data from the JAVELIN Solid tumor trial: Abstract No. 9086. Presented at 53rd ASCO Annual Meeting; Jun 2-6, 2017

2. 27% (8/30) subjects did not have PD-L1 status reported and 9.5% (2/21) subjects were non-evaluable for post dose scans (included in ORR calculation) due to withdrawal prior to first scan. Note that a score of 80% in Merck KGaA 73-10 assay employed here is equivalent to a score of 50% in The 22C3 assay employed by US Merck.

Pepinemab increases ratio of cytotoxic T cells to myeloid derived suppressor cells

Research Article

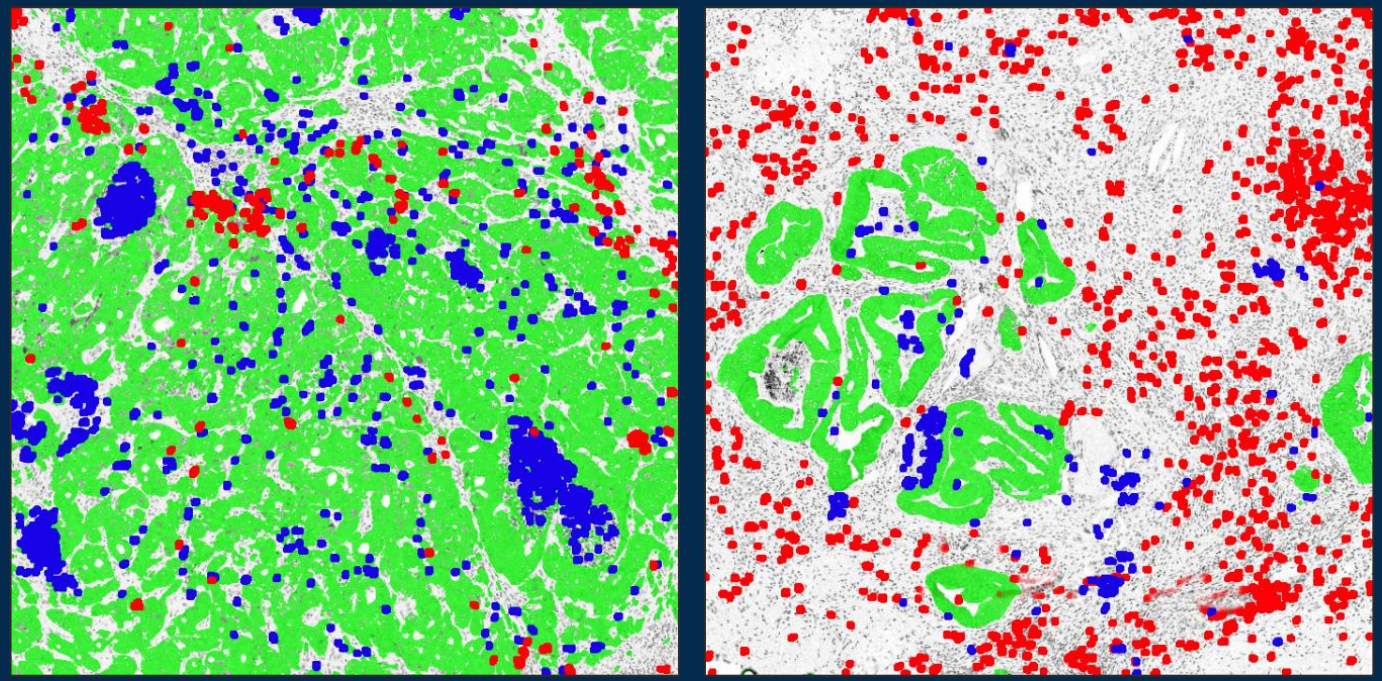
Cancer Immunology Research

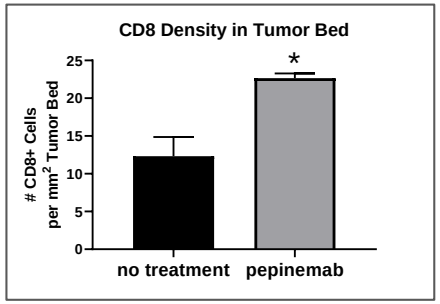
Semaphorin4D Inhibition Improves Response to Immune-Checkpoint Blockade via Attenuation of MDSC Recruitment and Function

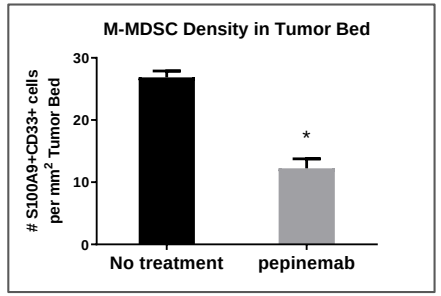
Paul E. Clavijo[1], Jay Friedman[1], Yvette Robbins[1], Ellen C. Moore[1], Ernest Smith[2], Maurice Zauderer[2], Elizabeth E. Evans[2], and Clint T. Allen[1,3]

No treatment
Low CD8+ T cells
High Tumor content and MDSC

Pepinemab
High CD8+ T cells
Low tumor content and MDSC







Density was determined from entire tumor bed (n= 2 sections/patient).

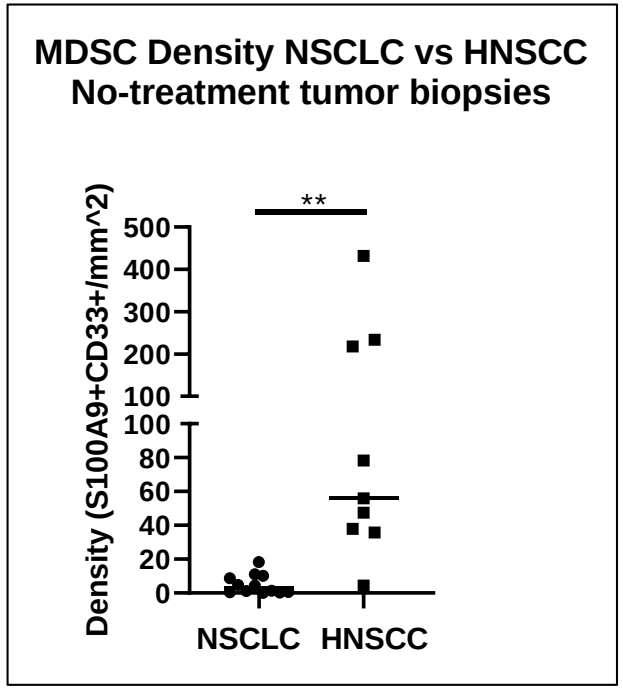
Patients received neoadjuvant chemotherapy before immunotherapy and surgery

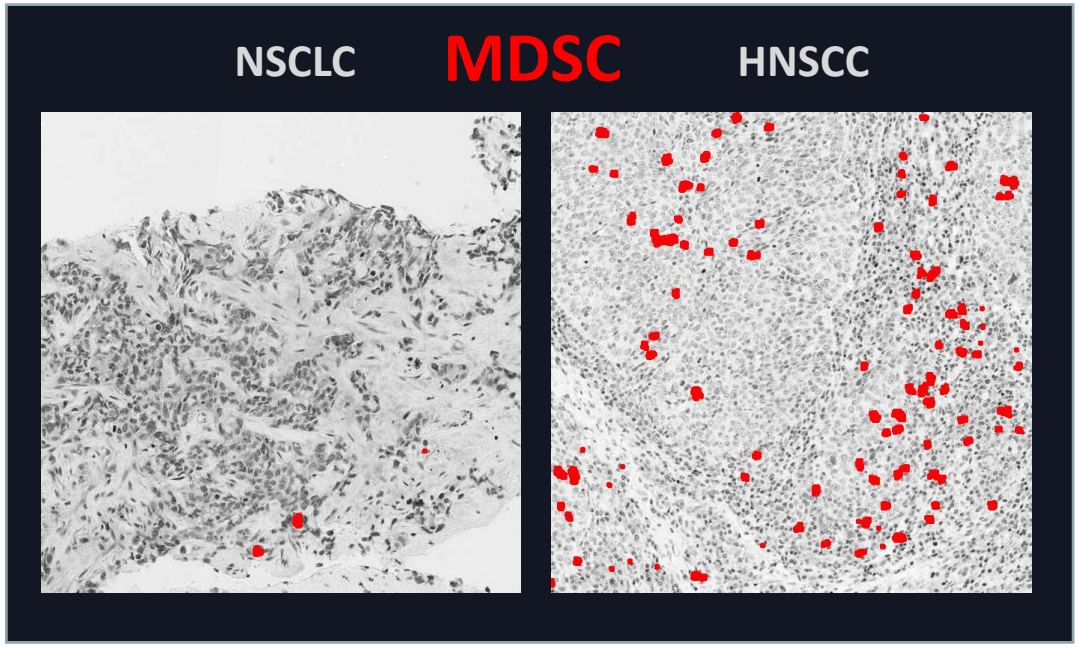
MSS Colorectal cancer metastasis to liver – neoadjuvant/"window of opportunity" study
Winship Cancer Institute, Emory University

M-MDSC (S100A9+CD33+)
CD8+ T cells
Tumors (Cytokeratin+)

NEXT STEPS: Head & Neck Cancer (HNSCC) Combination Immunotherapy with KEYTRUDA®

- We have entered into agreement with MSD to initiate a phase 2 study of pepinemab in combination with pembrolizumab in HNSCC, a tumor indication characterized by high levels of SEMA4D that induce and expand MDSC.
- NSCLC have low MDSC content relative to HNSCC, therefore, benefit from only 1 of 2 major pepinemab mechanisms of action.
- MDSC represent an important mechanism of resistance to immune checkpoint therapy





NSCLC: pre-treatment biopsies from CLASSICAL-Lung
HNSCC: pre-treatment and no treatment biopsies from HNSCC integrated biomarker trial (collaboration at Emory University)

Keynote B84 for front line treatment in patients with recurrent or metastatic Head & Neck Cancer

Phase 1b
Safety Run-in

Phase 2
Efficacy
(up to 65)

20 mg/kg Pepinemab
+200 mg Pembrolizumab
~50% CPS<20
~50% CPS≥ 20

Sponsored by:

Drug provided by:
Merck, MSD

Study Objectives

- Safety, tolerability, RP2D (Phase 1b) and Objective Response Rate (Phase 2)
- Secondary objectives include firther evaluation of activity (PFS, OS, DOR)
- Additional objectives include immunogenicity, PK/PD, and candidate biomarkers of activity
- Data anticipated mid-2022

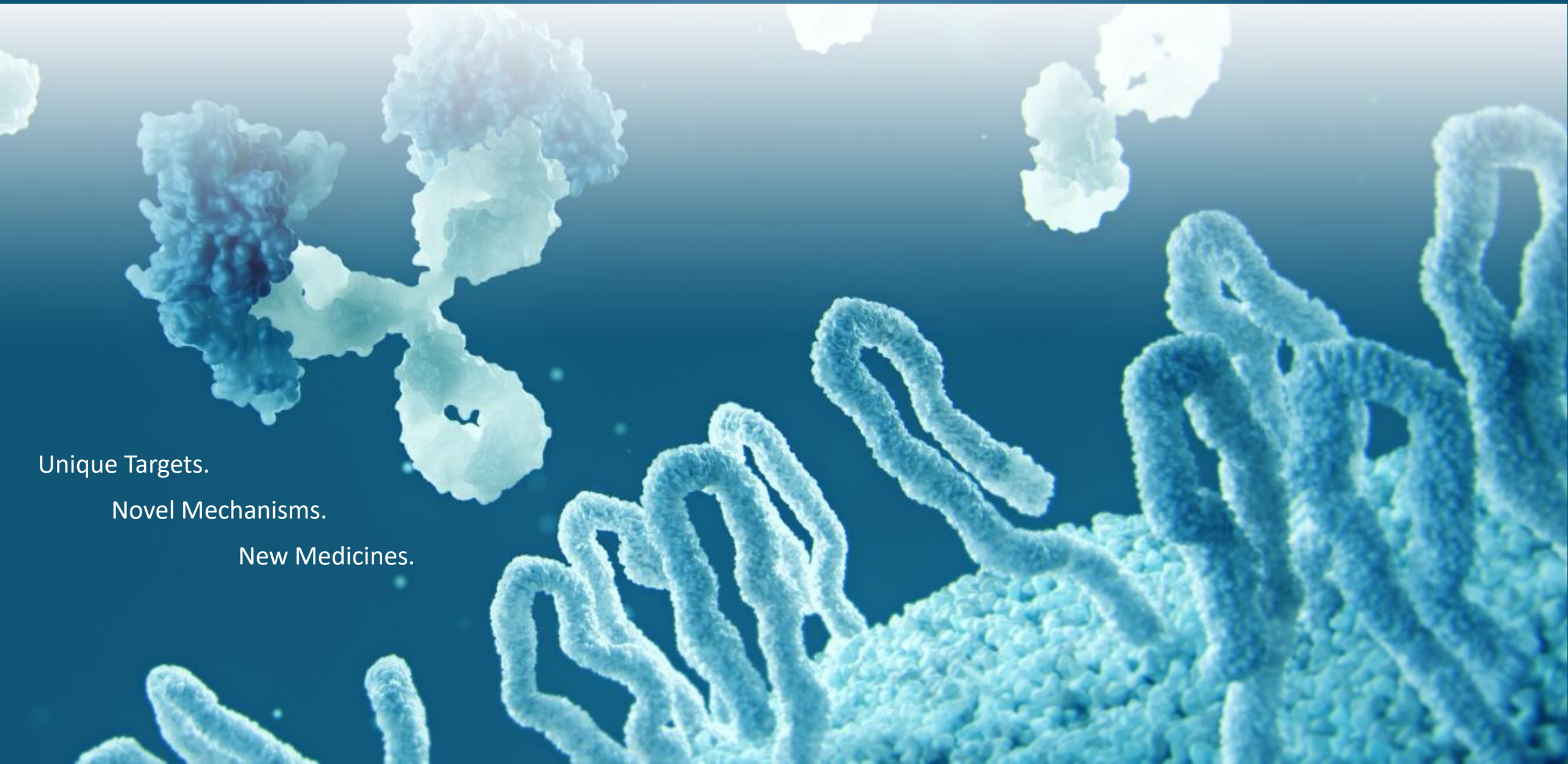
Pepinemab for Immuno-Oncology

Mechanism of Action:	Facilitate infiltration of T cells and dendritic cells Reduce immunosuppression
Safety and tolerability:	Well tolerated Does not enhance immune-related toxicities of partner drug
Clinical Efficacy (POC):	Appears to increases frequency and duration of objective responses Durable responses in some patients with PDx-resistant/refractory disease Apparent 2-3X increase in ORR in patients with PD-L1 negative/low tumors compared to single agent checkpoint inhibitor

Initiated Phase 2 trial in HNSCC

Broad application in solid tumors – enhances activity of immunotherapies

Corporate Summary



Unique Targets.

Novel Mechanisms.

New Medicines.

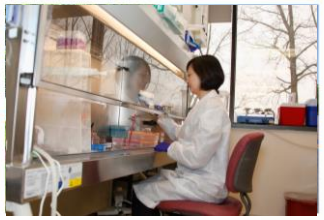
Anticipated Vaccinex 2021 Milestones

Final Clinical Data for SIGNAL Cohort B study in Huntington's Disease	H2 2020
Publish Clinical Data for SIGNAL study in Huntington's Disease	2021
Publish Clinical Data for Pepinemab in Combination with Avelumab in NSCLC Clinical Cancer Research, available online	April 5, 2021
Enrollment of first patient for phase 2 study of Pepinemab in Combination with Keytruda® in front line Head & Neck Cancer **Expect interim data mid-2022**	**Q2 2021** **Mid-2022**
Enrollment of first patient in Alzheimer's disease phase 1b/2a study **Expect data from blinded placebo-control study**	**Q2 2021** **Q4 2022/Q1 2023**

Robust Patent Estate
VX15 (pepinemab) US Patents and Patent Applications

Key Composition of Matter Claims	**US No. 8,496,938 issued 7/30/13)** ***Expected Exclusivity to 2030 (before patent term extension)***
Key Additional Method of Use Claims for Treatment of Cancer and Neurodegenerative Diseases	US No. 9,243,9068 issued 1/26/16 US No. 9,249,227 issued 2/2/16 Filed: 2014 – 2015 Expected Exclusivity to 2035 (before patent term extension)

Total Patent Franchise	US	International
Granted/allowed	26	11
Pending	15	13



Vaccinex, Inc. (Nasdaq: VCNX) is a publicly traded clinical-stage biotechnology company engaged in the discovery and development of targeted biotherapeutics to treat serious diseases with unmet medical needs, including cancer and neurodegenerative diseases.

VCNX (NASDAQ)	
Incorporated	2001
Headquarters	Rochester, NY
Employees	39
IPO	August 2018
February 2021 Capital Raise	$32 M
Cash balance*	$29.4M
Shares Outstanding*	28.4M
Analysts	Oppenheimer (L. Gershell), BTIG (T. Shrader)

* As of 31MAR2021

Vaccinex Board of Directors

Albert D. Friedberg	Chairman, President and CEO of Friedberg Mercantile Group, a Toronto-based commodities and investment management firm he founded in 1971. He served as Chairman of the Toronto Futures Exchange from March 1985 to June 1988.
Chrystyna M. Bedrij	Co-Founder and Principal, Griffin Securities
Jacob B. Frieberg	Principal, The WTF Group.
J. Jeffrey Goater	CEO of Surface Oncology, Formerly, CFO of Voyager Therapeutics.
Bala S. Manian, Ph.D.	Founder (or co-founder) of Quantum Dot Corporation, SurroMed, Biometric Imaging, LumisysInc., Molecular Dynamics and ReaMetrix.
Gerald E. Van Strydonck	Formerly, Managing Partner at PricewaterhouseCoopers.
Barbara Yanni	Formerly, Vice President and Chief Licensing Officer at Merck & Co., Inc.
Maurice Zauderer, Ph.D.	Vaccinex Founder, President and Chief Executive Officer. Formerly, Professor at University of Rochester and at Columbia University.

Contact us

Maurice Zauderer, CEO
mzauderer@vaccinex.com

Elizabeth Evans, COO
eevans@vaccinex.com

www.vaccinex.com

Vaccinex Selected References, Oncology

1. Shafique MR, Fisher TL, Evans EE, Leonard JEE, Pastore DRE, Mallow CL, Smith E, Mishra V, Schroder A, Chin KA, Beck JT, Baumgart MA, Govindan R, Gabrial NY, Spira AI, Seetharamu N, Lou Y, Mansfield AS, Sanborn RE, Goldman JW, Zauderer M. **A Phase Ib/2 Study of Pepinemab in Combination with Avelumab in Advanced Non–Small Cell Lung Cancer**. Clin Cancer Res 2021, doi: 10.1158/1078-0432.CCR-20-4792
2. Clavijo PE, Friedman J, Robbins Y, Moore EC, Smith ES, Zauderer M, Evans EE, Allen CT. **Semaphorin4D inhibition improves response to immune checkpoint blockade via attenuation of MDSC recruitment and function**. Cancer Immunol Res. 2019 Feb;7(2):282-291
3. Evans EE, Jonason AS Jr, Bussler H, Torno S, Veeraraghavan J, Reilly C, Doherty MA, Seils J, Winter LA, Mallow C, Kirk R, Howell A, Giralico S, Scrivens M, Klimatcheva K, Fisher TL, Bowers WJ, Paris M, Smith ES, Zauderer M. **Antibody blockade of semaphorin 4D promotes immune infiltration into tumor and enhances response to other immunomodulatory therapies.** Cancer Immunol Res. 2015 Jun;3(6): 689-701. http://www.ncbi.nlm.nih.gov/pubmed/25614511
4. Amita Patnaik, Glen J. Weiss, John E. Leonard, Drew Warren Rasco, Jasgit C. Sachdev, Terrence L. Fisher, Christine Reilly, Laurie A. Winter, Robert B. Parker, Danielle Mutz, Lisa Blaydorn, Anthony W. Tolcher, Maurice Zauderer and Ramesh K. Ramanathan. **Safety, Tolerability, Pharmacokinetics and Pharmacodynamics of VX15/2503, a Humanized IgG4 anti-SEMA4D Antibody, in a First-In-Human Phase 1 Study of Patients with Advanced Solid Disease.** Clin Cancer Res. 2015 Oct 7. http://clincancerres.aacrjournals.org/content/22/4/827.full.pdf+html
5. Leonard JE, Fisher TL2 Winter LA, Cornelius CA, Reilly C, Smith ES, Zauderer M. **Nonclinical Safety Evaluation of VX15/2503; a Humanized IgG4 Anti-SEMA4D Antibody.** Mol Cancer Ther. 2015 Feb 5 http://www.ncbi.nlm.nih.gov/pubmed/25657333
6. Fisher TL, Reilly CA, Winter LA, Pandina T, Jonason A, Scrivens M, Balch L, Bussler H, Torno S, Seils J, Mueller L, Huang H, Klimatcheva E, Howell A, Kirk R, Evans E, Paris M, Leonard JE, Smith ES, Zauderer M. **Generation and preclinical characterization of an antibody specific for SEMA4D**. Mabs. 2015 Oct 20. http://www.tandfonline.com/doi/abs/10.1080/19420862.2015.1102813
7. Fisher, T. L., J. Seils, C. Reilly, V. Litwin, L. Green, J. Salkowitz-Bokal, R. Walsh, S. Harville, J. E. Leonard, E. Smith, and M. Zauderer. 2016. **Saturation monitoring of VX15/2503, a novel semaphorin 4D-specific antibody, in clinical trials.** *Cytometry B Clin. Cytom.* 90: 199-208. http://onlinelibrary.wiley.com/doi/10.1002/cyto.b.21338/abstract

Schematics created with BioRender.com

Vaccinex Selected References, Neurology

1. Smith ES, Jonason A, Reilly C, Veeraraghavan J, Fisher T, Doherty M, Klimatcheva E, Mallow C, Cornelius C, Leonard JE, Marchi N, Janigro D, Argaw AT, Pham T, Seils J, Bussler H, Torno S, Kirk R, Howell A, Evans EE, Paris M, Bowers WJ, John G, Zauderer M. **SEMA4D compromises blood-brain barrier, activates microglia, and inhibits remyelination in neurodegenerative disease**. Neurobiol Dis. 2014 Oct 18;73C:254-268. doi: 10.1016/j.nbd.2014.10.008. http://www.sciencedirect.com/science/article/pii/S0969996114003015
2. Southwell AL, Franciosi S, Villanueva EB, Xie Y, Winter LA, Veeraraghavan J, Jonason A, Felczak B, Zhang W, Kovalik V, Waltl S, Hall G, Pouladi MA, Smith ES, Bowers WJ, Zauderer M, Hayden MR. **Anti-semaphorin 4D immunotherapy ameliorates neuropathology and some cognitive impairment in the YAC128 mouse model of Huntington disease**. Neurobiol Dis. 2015 Feb 3; 76:46–56. http://www.sciencedirect.com/science/article/pii/S0969996115000145
3. LaGanke, C., L. Samkoff, K. Edwards, L. Jung Henson, P. Repovic, S. Lynch, L. Stone, D. Mattson, A. Galluzzi, T. L. Fisher, C. Reilly, L. A. Winter, J. E. Leonard, and M. Zauderer. 2017. **Safety/tolerability of the anti-semaphorin 4D Antibody VX15/2503 in a randomized phase 1 trial**. Neurol Neuroimmunol Neuroinflamm 4: e367. https://www.ncbi.nlm.nih.gov/pubmed/28642891
4. Leonard JE, Fisher TL2 Winter LA, Cornelius CA, Reilly C, Smith ES, Zauderer M. **Nonclinical Safety Evaluation of VX15/2503; a Humanized IgG4 Anti-SEMA4D Antibody**. Mol Cancer Ther. 2015 Feb 5 http://www.ncbi.nlm.nih.gov/pubmed/25657333
5. Zauderer M, Fisher TL, Mishra V, Leonard JE, Reader A, Mallow C, Balch L, Howell A, Smith ES, and Evans EE. **SEMA4D upregulation signals neuronal stress and triggers reactive transformation of astrocytes**. *In preparation*
6. Mao Y, Evans E, Mishra V, Zauderer M, Gold WA. **Anti-Semaphorin 4D rescues motor, cognitive and respiratory phenotypes in a Rett syndrome mouse model**. *In preparation*
7. Feigin AS, Evans EE , Fisher TL, Leonard JE, Reader A, Wittes J, Oakes D, Smith ES, Zauderer M, and the Huntington Study Group SIGNAL investigators. **Safety and efficacy of pepinemab antibody blockade of SEMA4D in patients with early Huntington's Disease: a randomized, placebo-controlled, multicenter, Phase 2 clinical trial (SIGNAL).** *In preparation*
8. Fisher TL, Reilly CA, Winter LA, Pandina T, Jonason A, Scrivens M, Balch L, Bussler H, Torno S, Seils J, Mueller L, Huang H, Klimatcheva E, Howell A, Kirk R, Evans E, Paris M, Leonard JE, Smith ES, Zauderer M. **Generation and preclinical characterization of an antibody specific for SEMA4D**. Mabs. 2015 Oct 20. http://www.tandfonline.com/doi/abs/10.1080/19420862.2015.1102813

Schematics created with BioRender.com