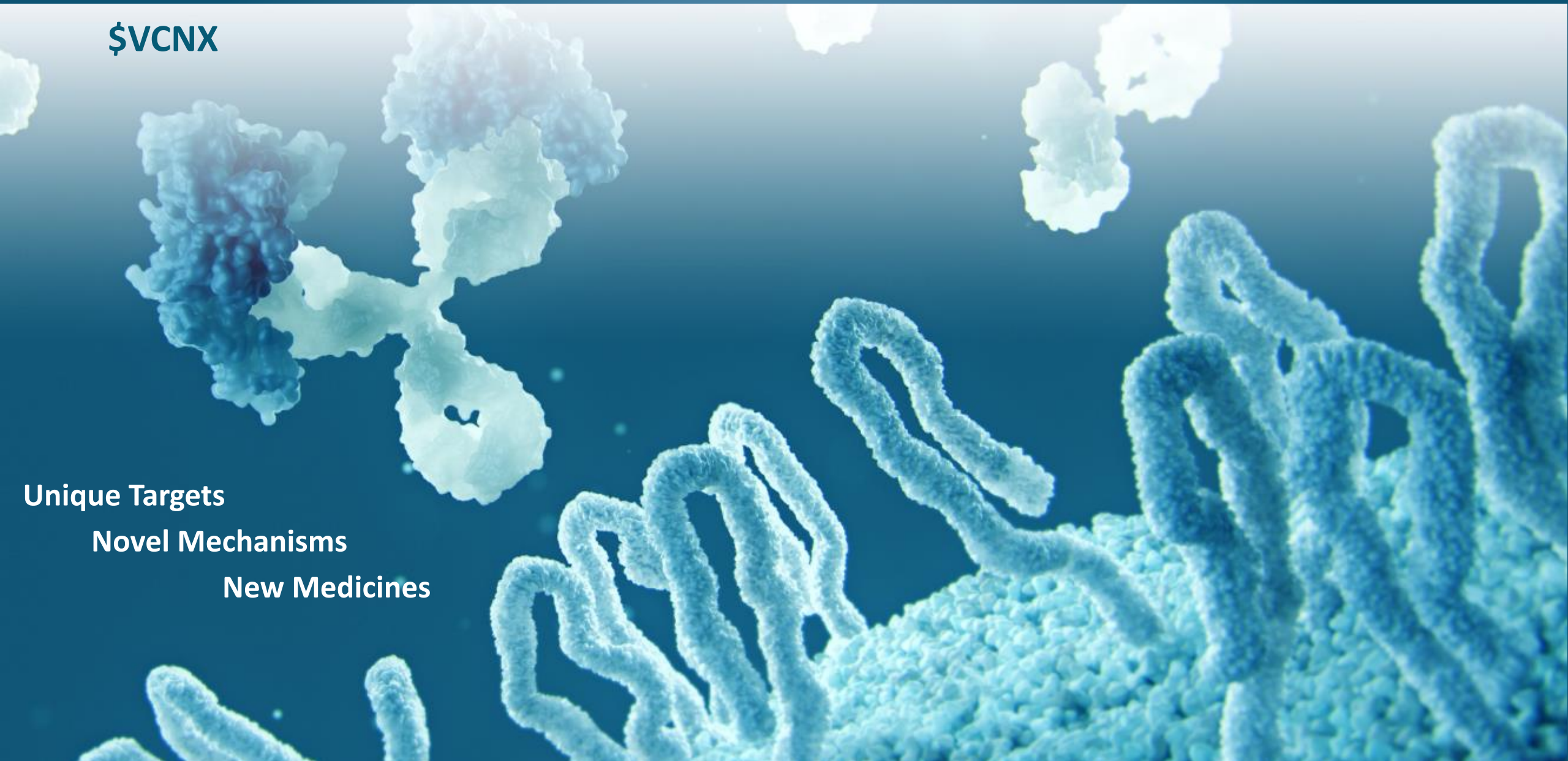
Corporate Presentation
August 2021

$VCNX



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. Such statements include, but are not limited to, statements about the Company's plans, expectations and objectives with respect to the results and timing of clinical trials of pepinemab in various indications, the use and potential benefits of pepinemab in Huntington's and Alzheimer's disease and other indications, and other statements identified by words such as "may," "will," "appears," "expect," "planned," "anticipate," "estimate," "intend," "hypothesis," "potential," "advance," and similar expressions or their negatives (as well as other words and expressions referencing future events, conditions, or circumstances). Forward-looking statements involve substantial risks and uncertainties that could cause the outcome of the Company's 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, uncertainties related to regulatory approval, risks related to the Company's dependence on its lead product candidate pepinemab, the ability to leverage its ActivMAb® platform, the impact of the COVID-19 pandemic, and other matters that could affect its development plans or the commercial potential of its product candidates. Except as required by law, the Company assumes no obligation to update these forward-looking statements. 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 the Company's periodic reports filed with the Securities and Exchange Commission ("SEC") and the other risks and uncertainties described in the Company's Form 10-K for year end December 31, 2021 and subsequent filings with the SEC.

VACCINEX

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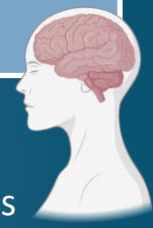
Novel antibody therapeutics target unmet needs

- ✓ **Novel Mechanistic Approach**
  Lead product: Pepinemab
  Humanized IgG4 monoclonal antibody, first in class
  Blocks a unique target, Semaphorin 4D (SEMA4D)
- ✓ **Clinical Proof of Concept**
- ✓ **Advanced clinical programs with near term opportunities for monetization by partnering**
- ✓ **Proprietary Drug Discovery Platform**

ActivMAb Technology

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

Neurodegenerative Disease



- Targets underlying disease pathology, a trigger of neuroinflammation
- Ability to repair and restore normal functions
- Broad application

Cancer Immunotherapy

- Overcomes resistance to existing immunotherapies
- Complements immune checkpoint therapies without added toxicity

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

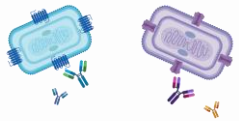
Oncology

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: VACCINEX

Additional Funding &/or Support by:

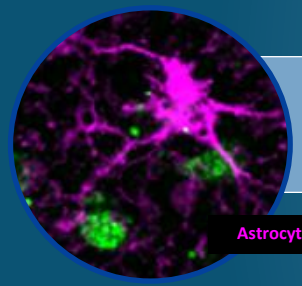
alzheimer's association — Alzheimer's Drug Discovery Foundation

MERCK — Merck KGaA, Darmstadt

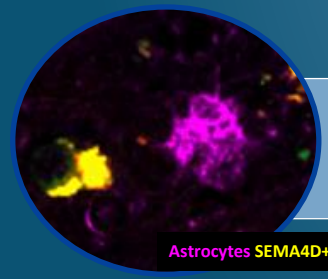
MERCK — Merck, MSD

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

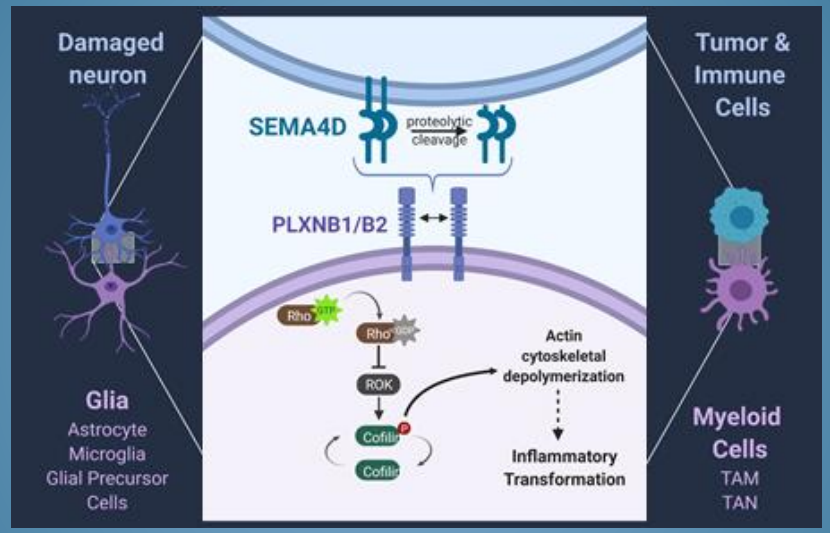
Neurodegenerative Disease



Healthy astrocytes interact with neurons to support normal function



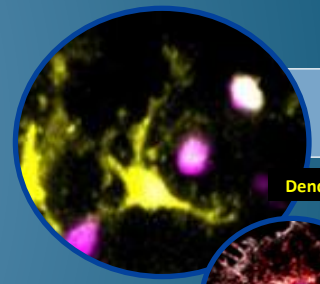
Reactive astrocytes lose interactions and function in Huntington's Disease



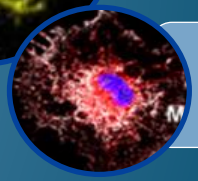
The cell cytoskeleton regulates cellular movement and cell-to-cell interactions

SEMA4D signals through PLXNB1/B2 receptors to trigger dissociation of polymerized F-actin and collapse of cell's cytoskeleton, and reprograms inflammatory responses

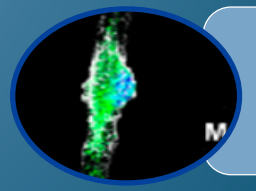
Cancer Immunotherapy



Healthy Dendritic Cells interacting with T cells*



Dendritic arms of M1 macrophage** facilitate movement and antigen presentation



Suppressor cells (MDSC and M2 Tumor Associated Macrophage**) reduce function and recruitment of T cells in tumor.

*Lancaster, et al. *Nat Commun 2019*
** Xiao, et al. *ACS Cent. Sci. 2020*

SEMA4D triggers loss of cellular interactions and normal functions, which can exacerbate neurodegeneration

Tumors utilize SEMA4D to restrict movement and communication of immune cells, leading to immune exclusion and resistance

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 • Initiated June 2021

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

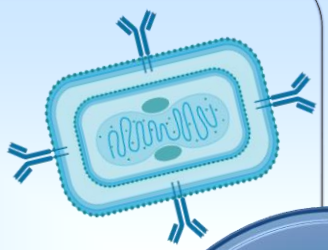
Well tolerated – does not enhance immune-related toxicities of partner drug

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) • Rationale: High levels of myeloid derived suppressor cells (MDSC) are induced by SEMA4D and a source of resistance to immune checkpoint therapy • Endpoints: Safety, ORR, and PFS, OS, DOR, biomarkers within TME • **Initiated June 2021**

ASSET: ActivMab Discovery Solutions

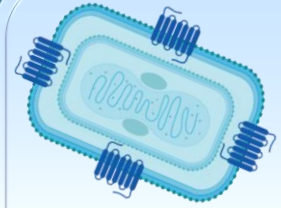
Antibody Library

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



Antigen Virus

Virions expressing Complex Membrane Proteins for Antibody Discovery

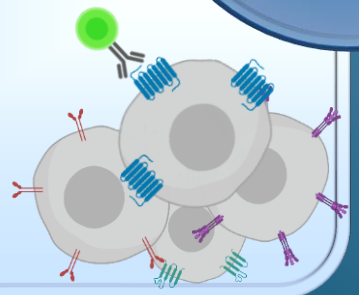


ActivMAb Technology

Discovery Solutions

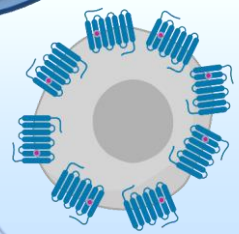
Target Identification

Large cDNA library expressed in Mammalian Cells



Protein Optimization

Mammalian expression of mutant libraries to enhance expression



Poxvirus based antibody and protein engineering solutions

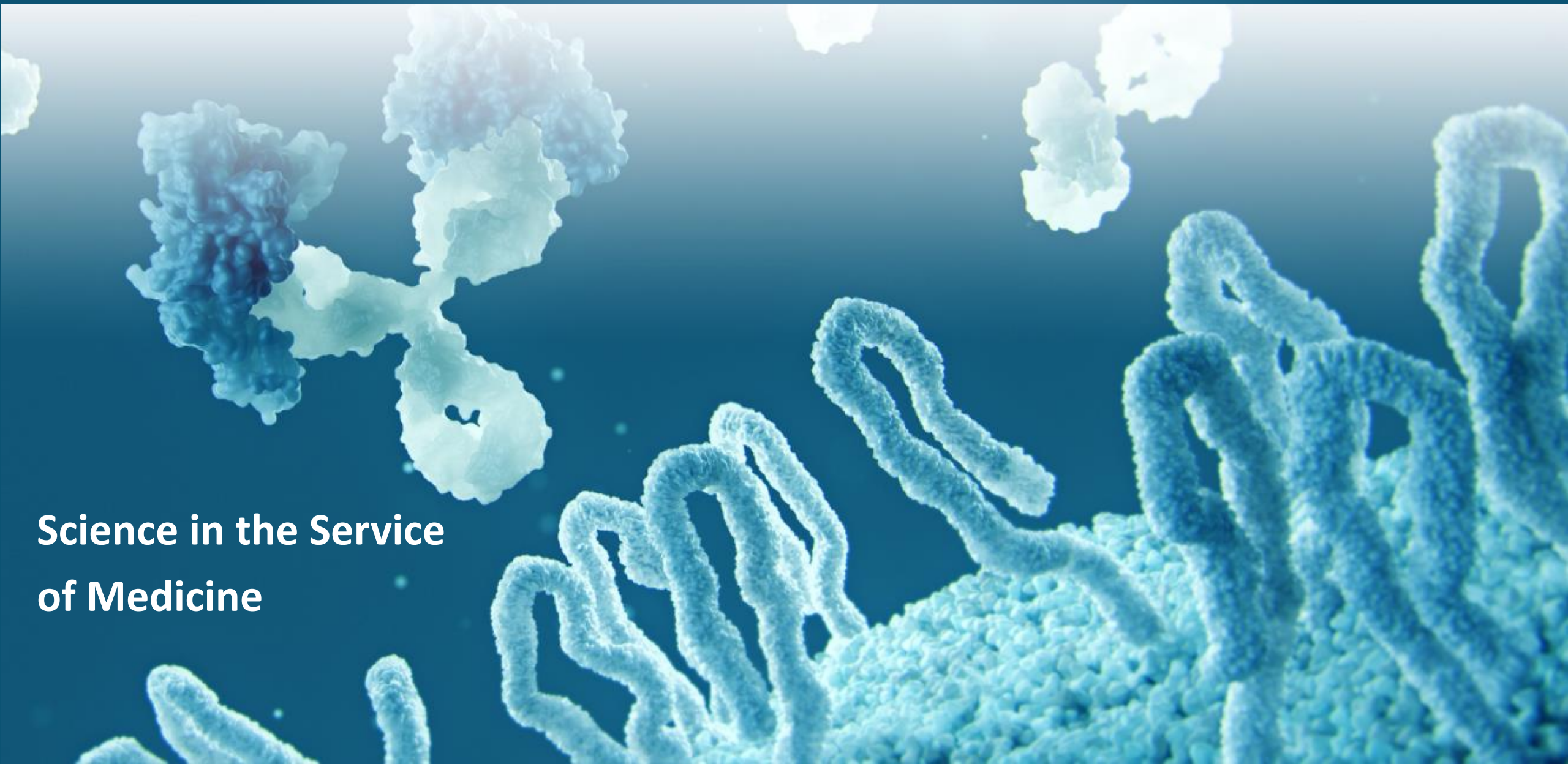
Unique capability for selection of high value antibodies against hard-to-target multi-pass membrane receptors
(i.e. GPCRs, ion channels)

Sustainable engine for value creation through pipeline expansion and strategic collaborations

Active collaborations with two major pharma and multiple biotech partners

VACCINEX

Pepinemab Antibody
Neurodegenerative Disease



Science in the Service
of Medicine

Glial cells respond to damage in the brain

SEMA4D compromises blood–brain barrier, activates microglia, and inhibits remyelination in neurodegenerative disease

Ernest S. Smith [a], Alan Jonason [a], Christine Reilly [a], Janaki Veeraraghavan [a], Terrence Fisher [a], Michael Doherty [a], Ekaterina Klimatcheva [a], Crystal Mallow [a], Chad Cornelius [a], John E. Leonard [a], Nicola Marchi [b], Damir Janigro [b], Azeb Tadesse Argaw [c], Trinh Pham [c], Jennifer Seils [a], Holm Bussler [a], Sebold Torno [a], Renee Kirk [a], Alan Howell [a], Elizabeth E. Evans [a], Mark Paris [a], William J. Bowers [a], Gareth John [c], Maurice Zauderer [a,*]

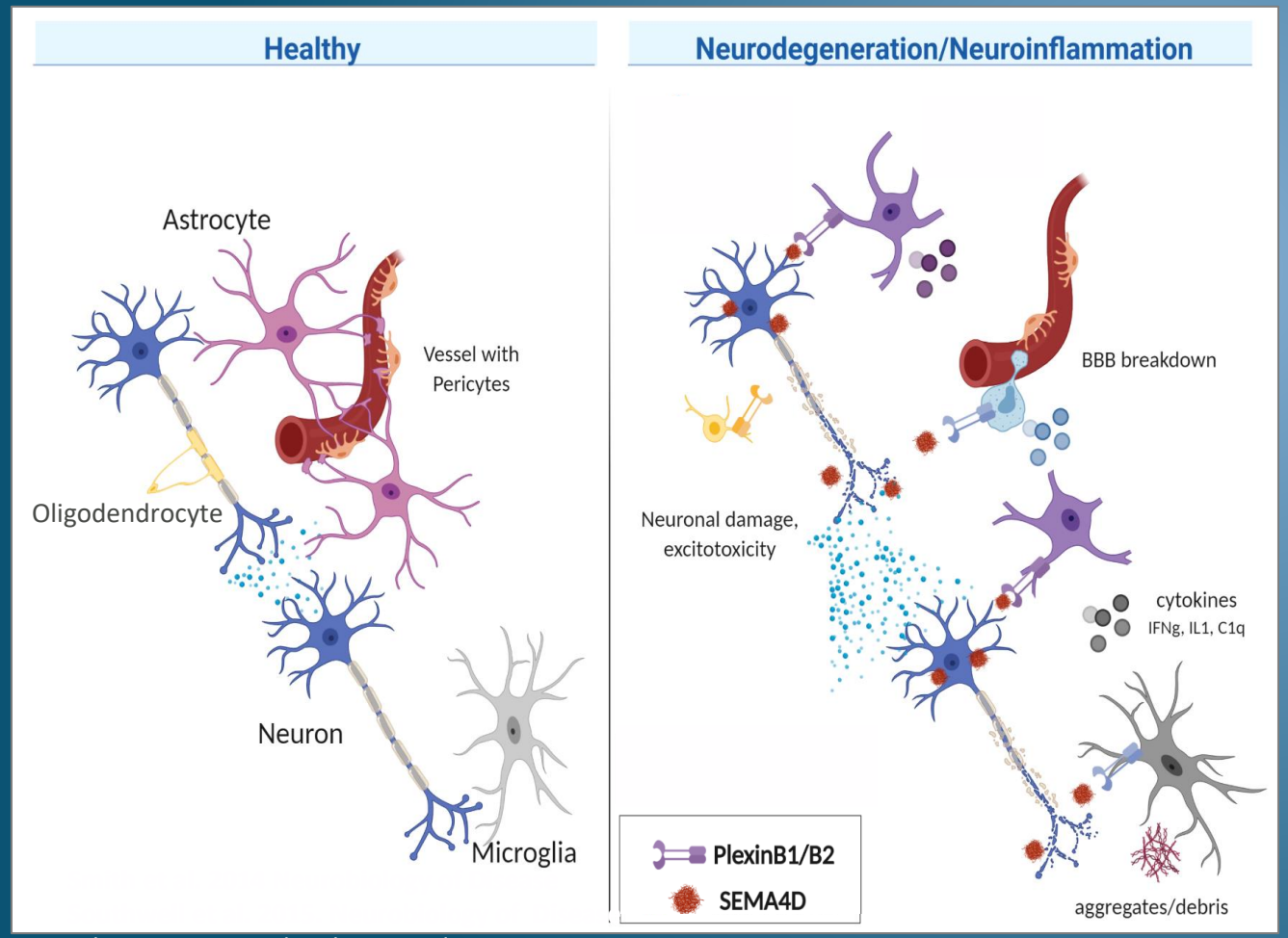
[a] Vaccinex, Inc., Rochester, NY 14620, USA

2014 Neurobiology of Disease

Anti-semaphorin 4D immunotherapy ameliorates neuropathology and some cognitive impairment in the YAC128 mouse model of Huntington disease

Amber L. Southwell [a], Sonia Franciosi [a], Erika B. Villanueva [a], Yuanyun Xie [a], Laurie A. Winter [b], Janaki Veeraraghavan [b], Alan Jonason [b], Boguslaw Felczak [a], Weining Zhang [a], Vlad Kovalik [a], Sabine Waltl [a], George Hall [a], Mahmoud A. Pouladi [c,d], Ernest S. Smith [b], William J. Bowers [b], Maurice Zauderer [b], Michael R. Hayden [a,*]

2015 Neurobiology of Disease



Schematics created with BioRender.com

TARGET: SEMA4D is upregulated on damaged neurons

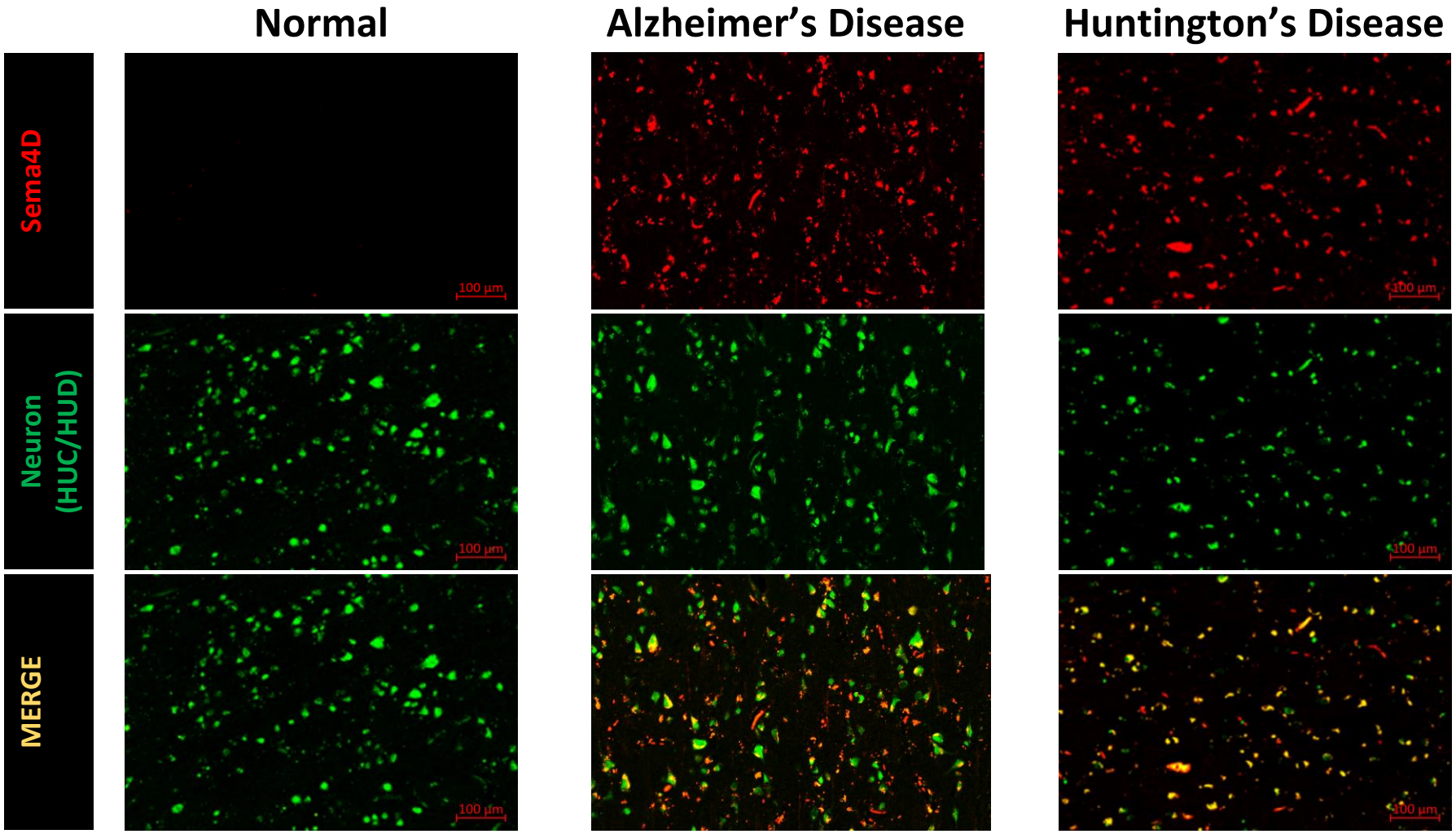
SEMA4D binding to Plexin receptors on glial cells to triggers collapse of cytoskeleton and transformation to reactive inflammatory state

Chronic activation contributes to and exacerbates neurodegeneration

DRUG: Pepinemab is a humanized IgG4 Mab that blocks the binding of SEMA4D to its receptors

- Repair and restore normal glial functions
- Reduce neuroinflammation

SEMA4D is upregulated in neurons during underlying disease progression



Human autopsy sections of frontal lobe

Huntington's Disease - slowly progressive, fatal neurodegenerative disease

Genetic Disease
HD is caused by dominant mutation in a single gene.

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

Orphan Disease
Estimated patient population in major markets is
~80,000 individuals with manifest disease and
>5X more are 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.

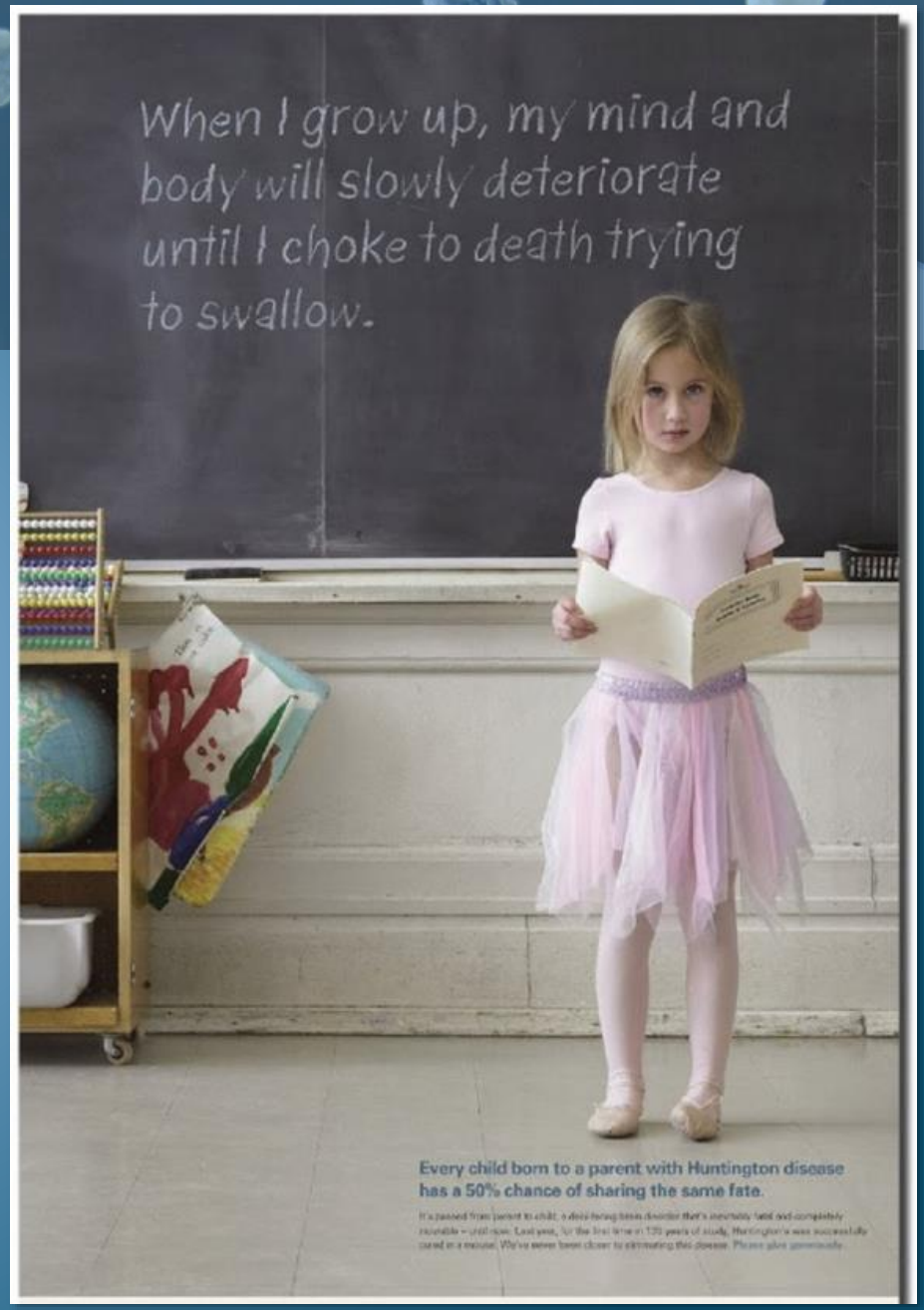
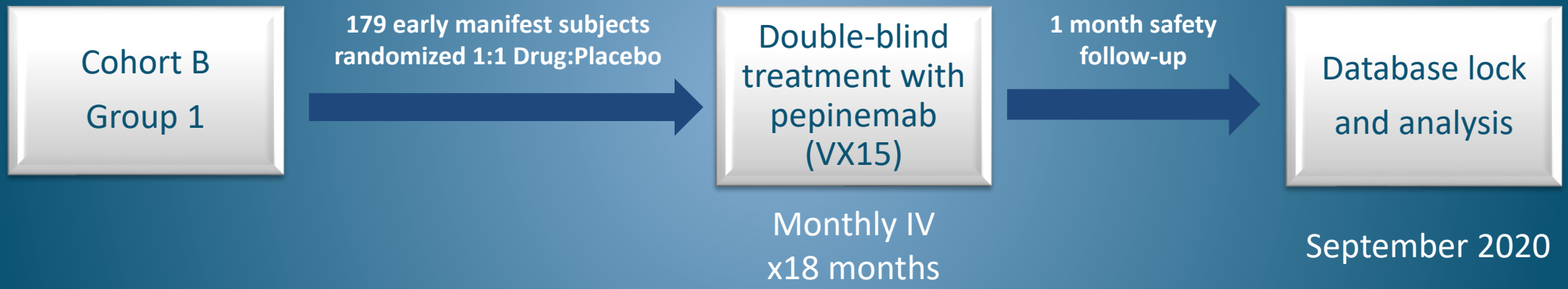


Photo credit: Huntington Society of Canada

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 Safety and Baseline Characteristics ITT population

Pepinemab (PEPI) SEMA4D blocking antibody is well tolerated

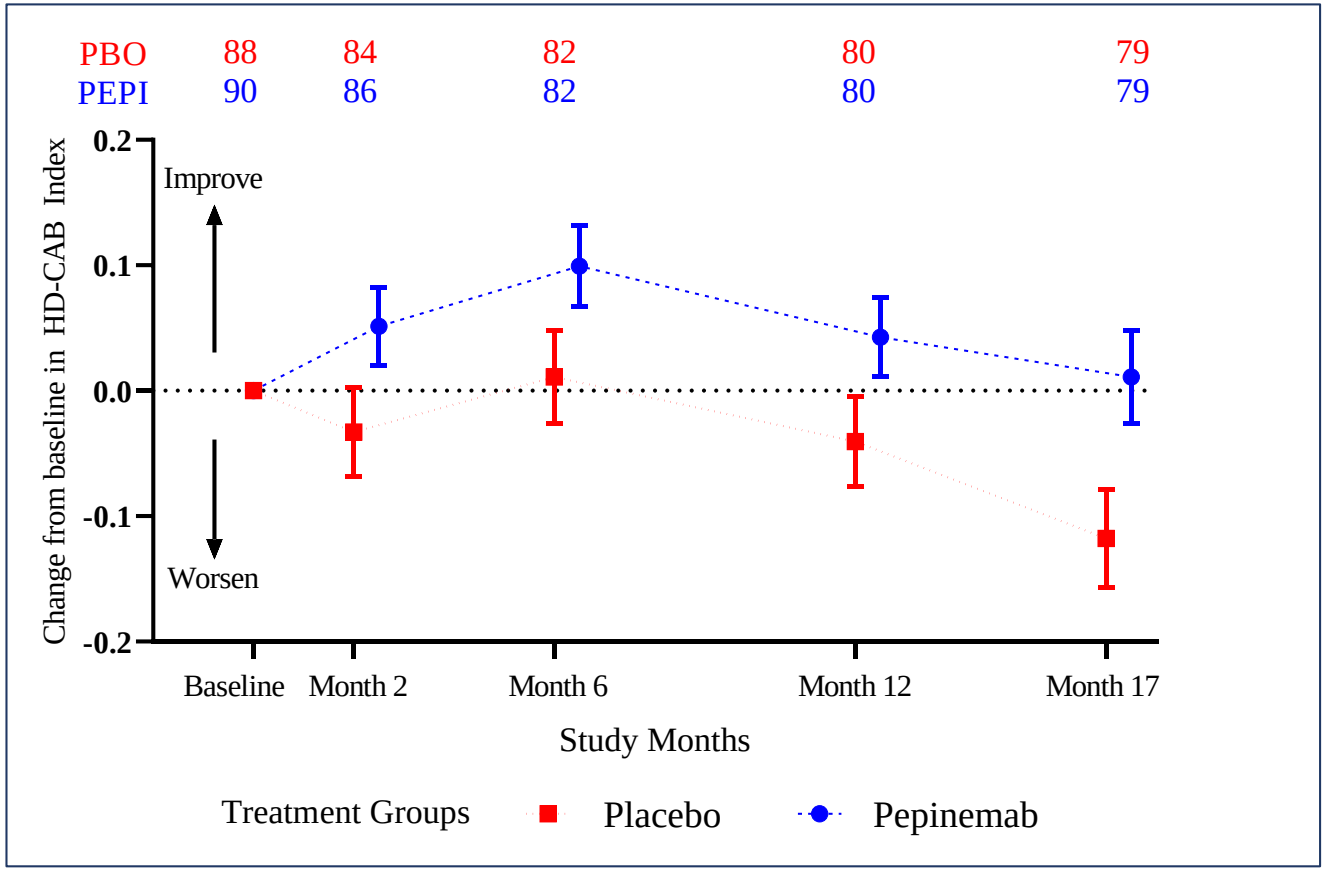
	Early Manifest Cohort B1	
	Placebo (n=88)	Pepinemab (n=91)
Discontinued Treatment Early	10	13
Had any SAE (*)	8	4
Had any Grade 3+ AE (*)	14	17
CAG repeat length	44.1	43.5
CAP score**	470	466
UHDRS-DCL at screening DCL-4, Unequivocal HD (>99% confident)	88 (100%)	91 (100%)
UHDRS-TFC at screening, n (%)		
11 12 13	33 (38%) 18 (20%) 37 (42%)	29 (32%) 37 (41%) 24 (27%)

*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



HD-CAB Composite Index:

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

Co-primary endpoints:

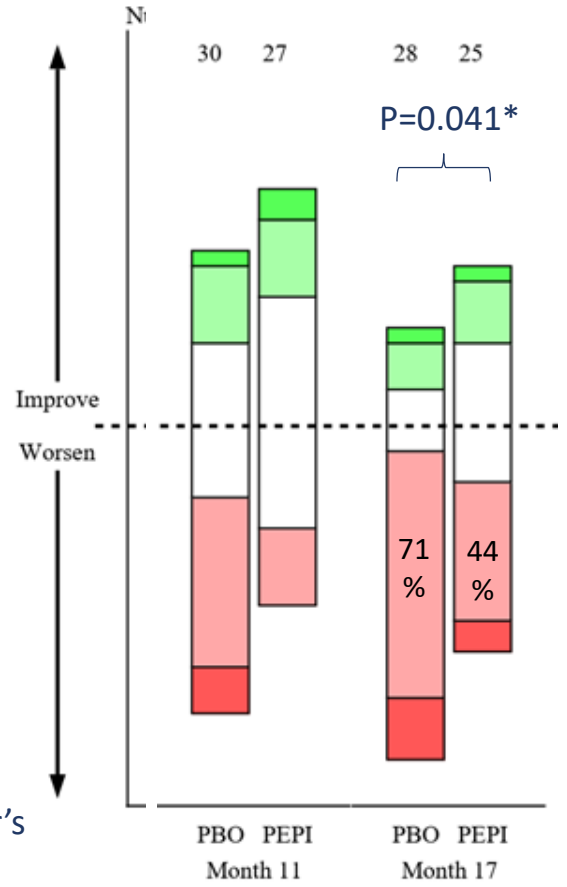
One-sided p-value	Favors PEPI	Success [Critical value]
OTS: 0.028 PTAP: 0.06	Yes	No [0.025] [0.0125]

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

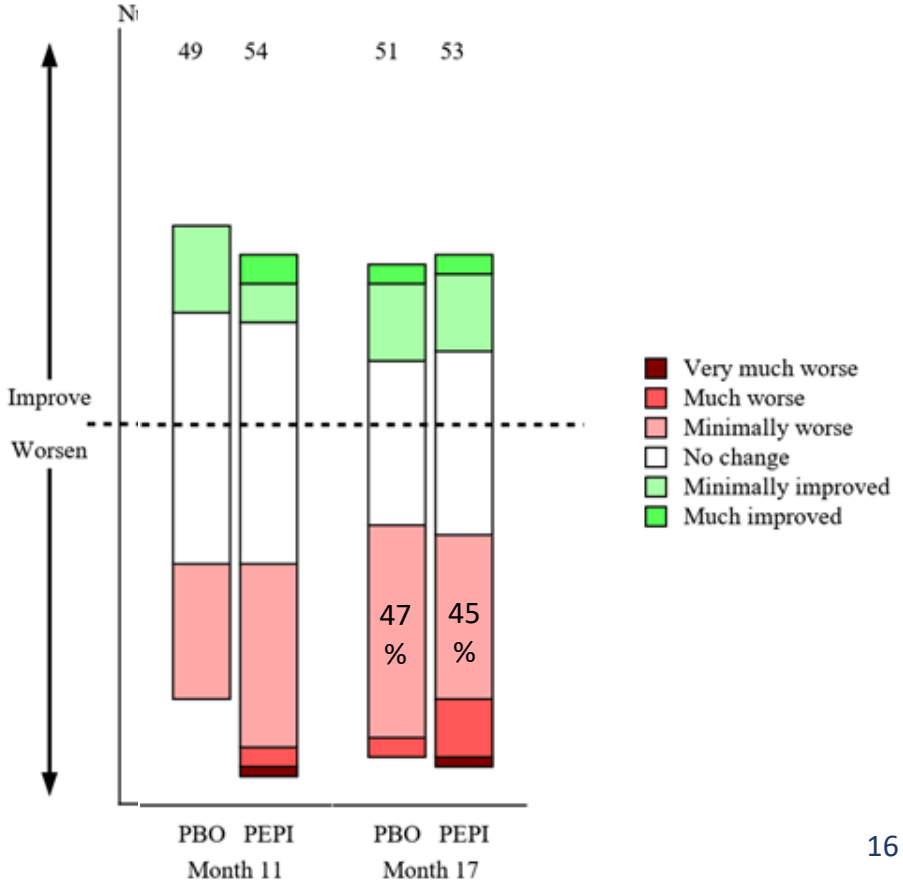
Subjects were less likely to experience decline in CGIC following treatment with pepinemab compared to placebo.

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

CGIC – Subjects with Baseline UHDRS TFC 11

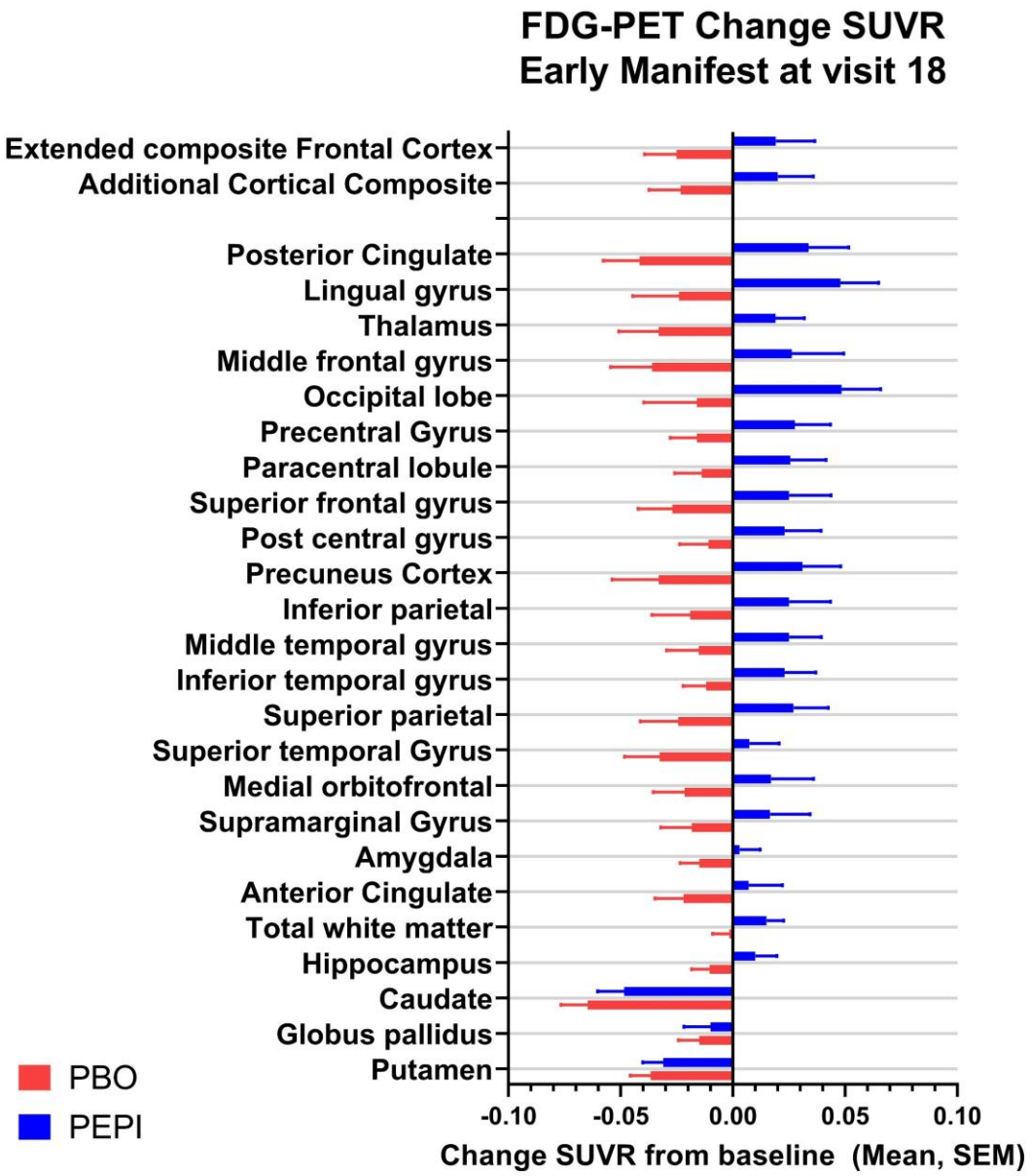


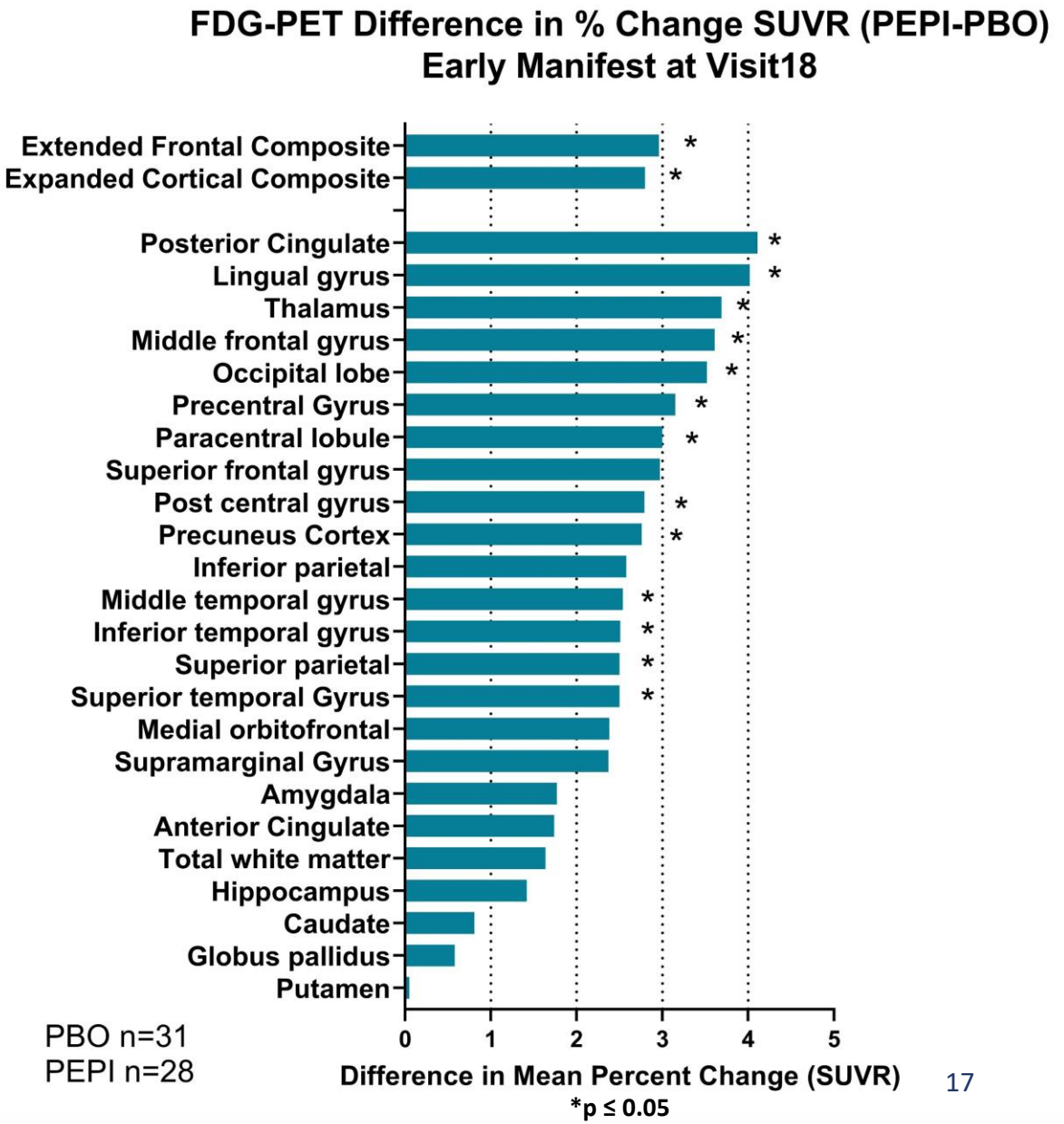
CGIC – Subjects with Baseline UHDRS TFC 12 and 13



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

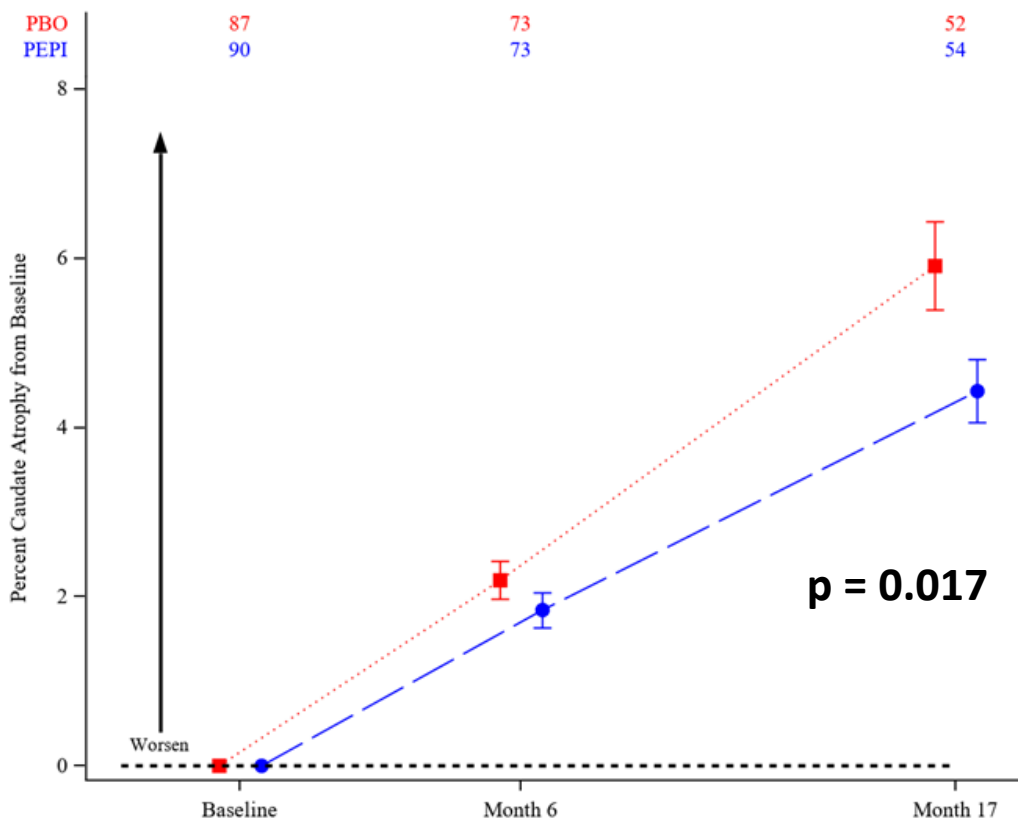
FDG-PET at 18 Months – Early Manifest HD: Pepinemab treatment reverses loss of metabolic activity



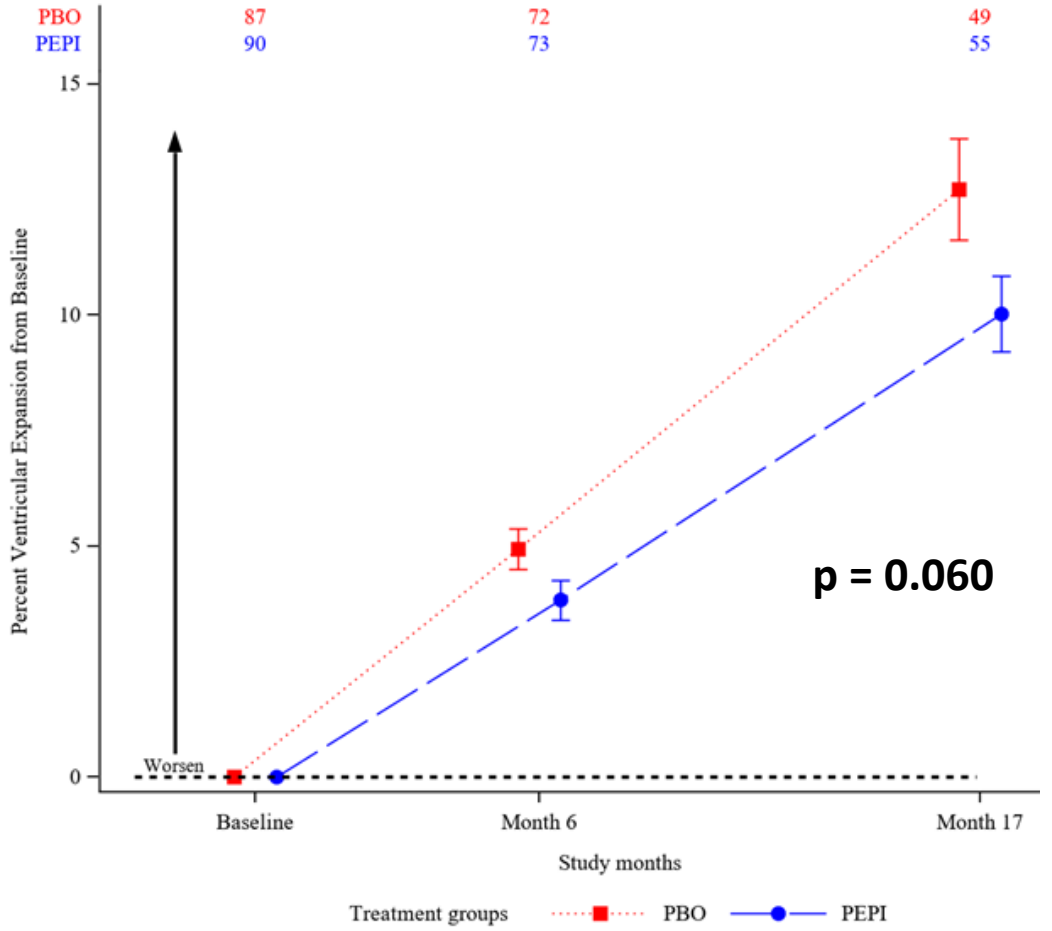


Volumetric MRI analysis – Boundary Shift Integral
Pre-specified exploratory endpoint

CBSI (caudate atrophy) Early Manifest (B1)

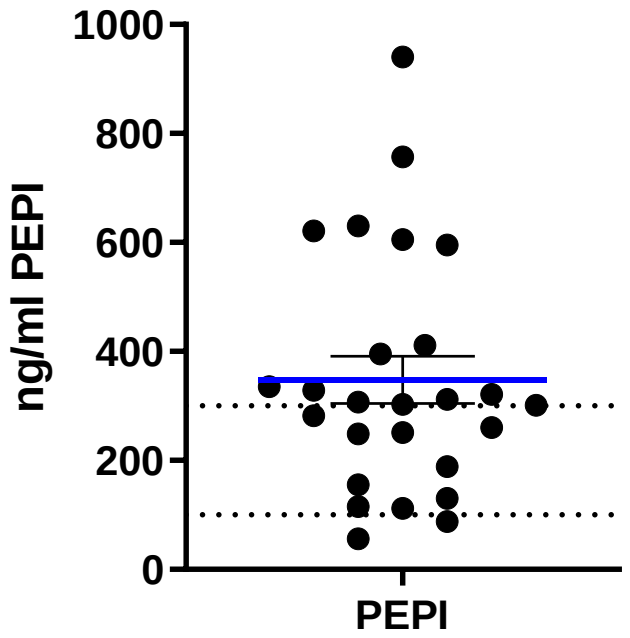


VBSI (ventricular expansion) Early Manifest (B1)

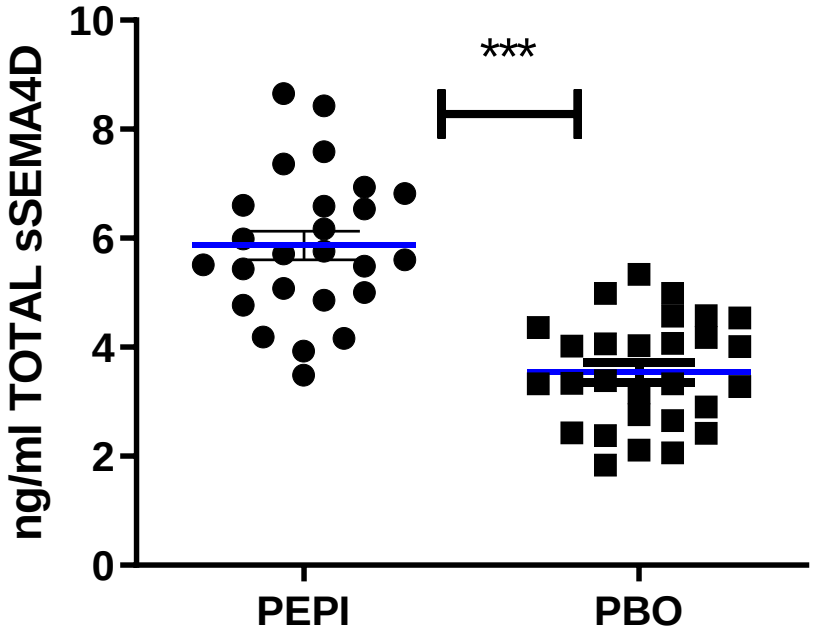


Pepinemab and sSEMA4D levels in cerebrospinal fluid (CSF)

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

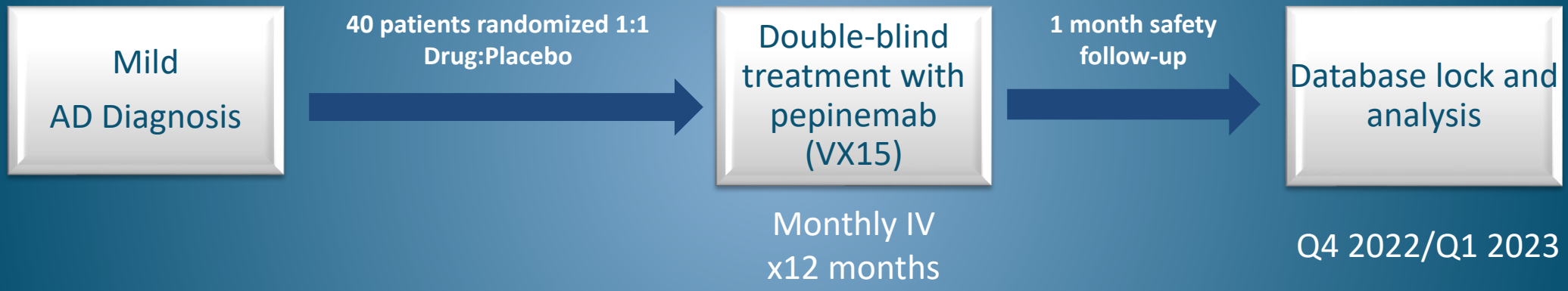
Orphan Disease and Fast Track designations

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

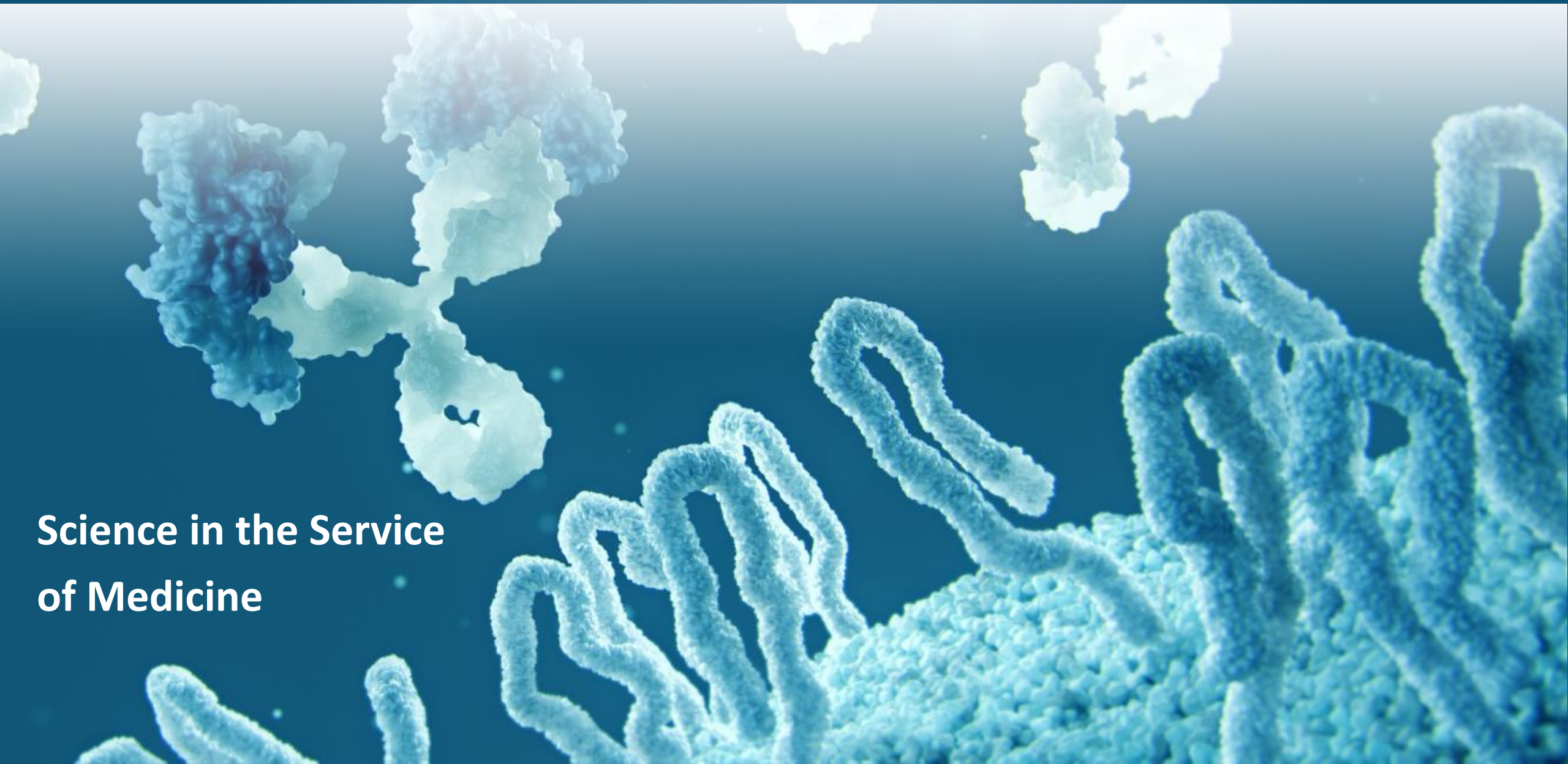
The mechanism of action of pepinemab has broad potential applications in neurological diseases that share common neuroinflammatory pathology, including HD, AD, FTD, PPMS.

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 demyelinating lysolecithin-lesion model
  - Smith et al. 2014 Neurobiology of Disease

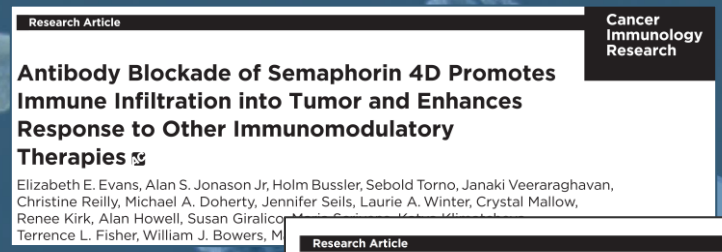
VACCINEX

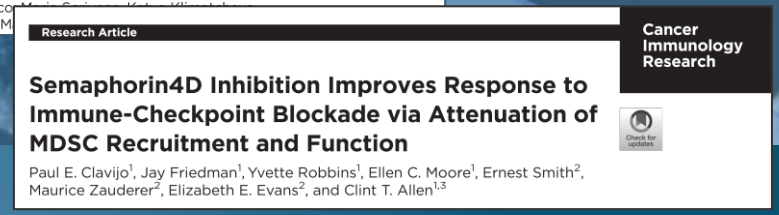
Pepinemab Antibody
Cancer Immunotherapy

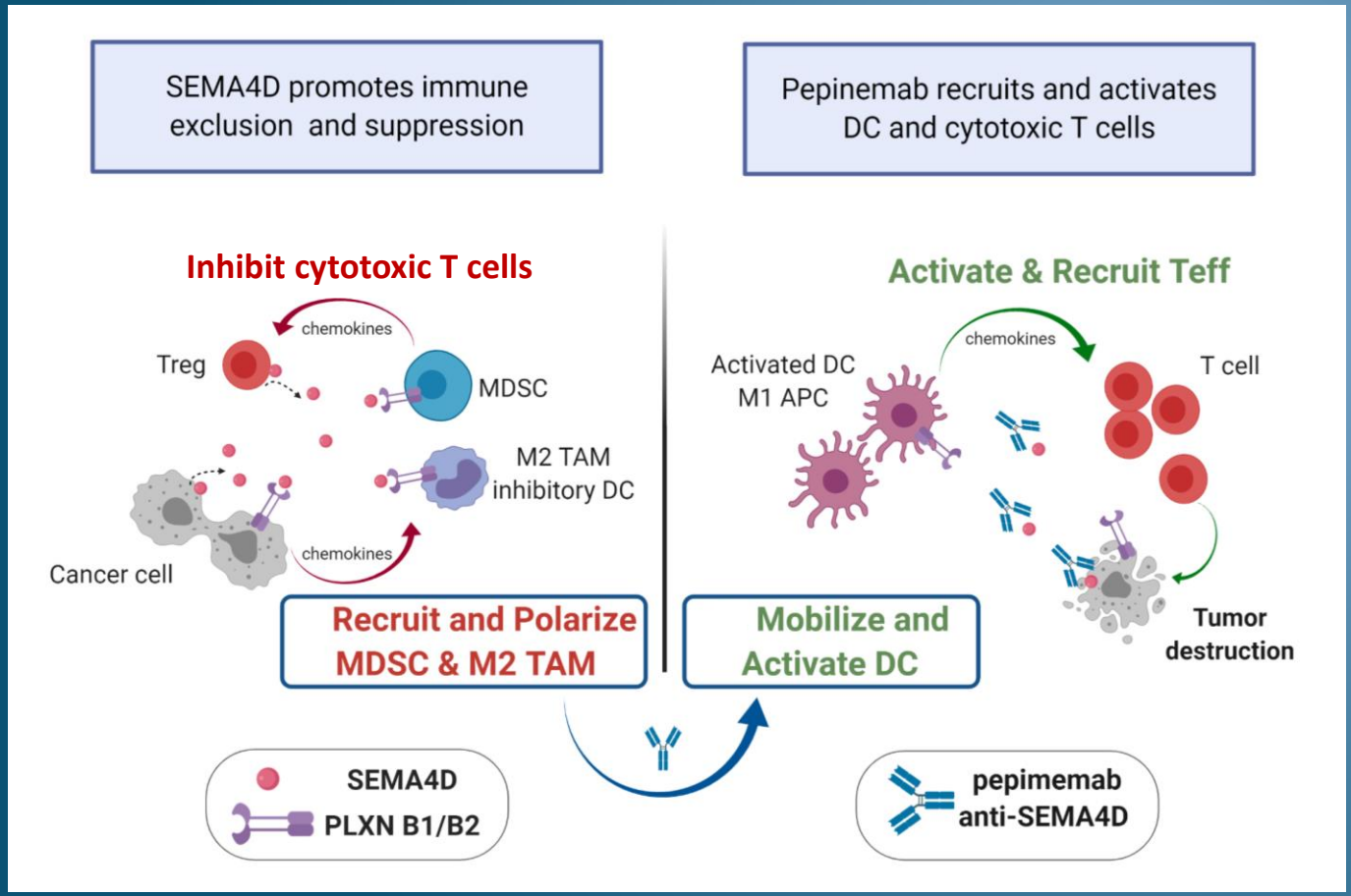


Science in the Service of Medicine

Pepinemab overcomes immune resistance mechanisms







TARGET: SEMA4D in the tumor is a mechanism to avoid destruction by immune system

- Creates barrier to restrict movement of anti-tumor immune cells
- Promotes recruitment and function of inhibitory suppressor cells

DRUG: Pepinemab is a humanized IgG4 Mab that blocks the binding of SEMA4D to its receptors

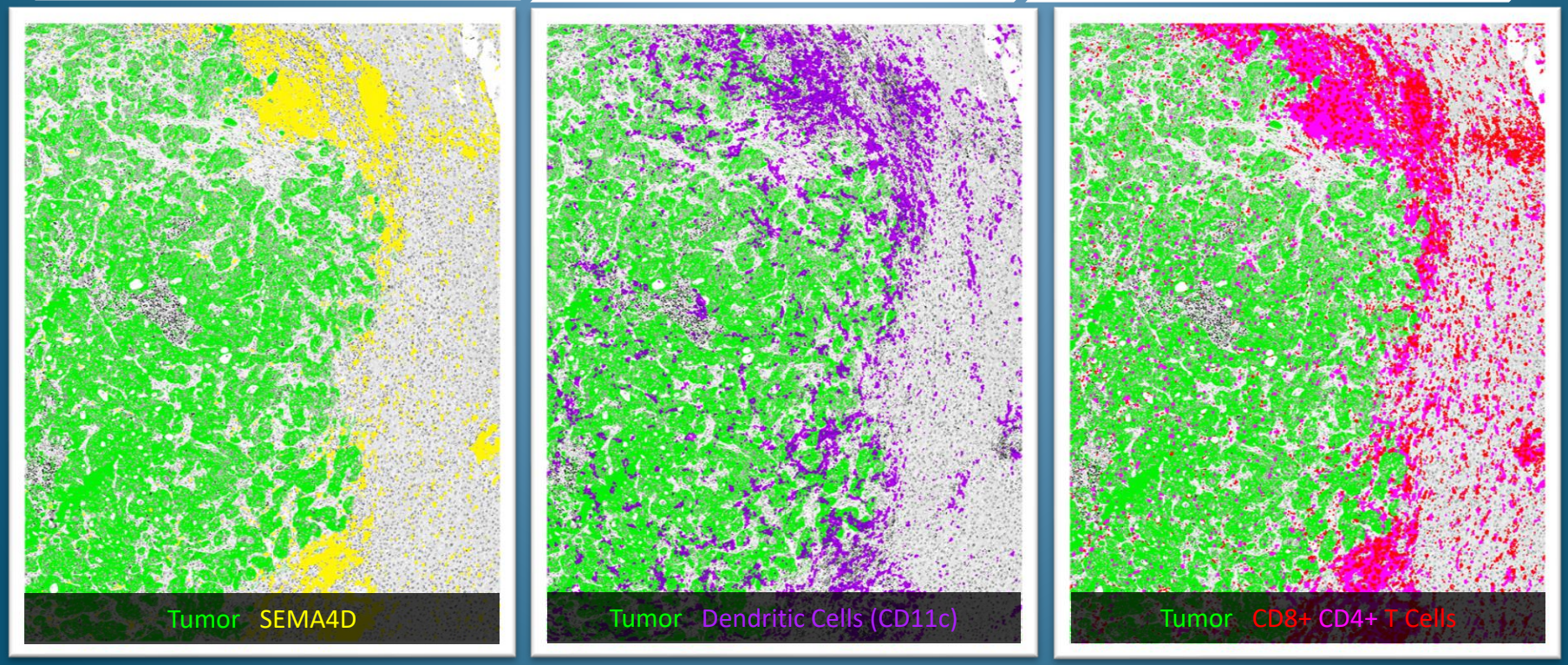
- **Overcomes immune resistance**
- Reduces immune suppression
- Activates and recruits anti-tumor and cytotoxic cells
- Neutralizes SEMA4D barrier at the tumor boundary to facilitate immune infiltration
- Enhances immune checkpoint therapy

SEMA4D creates a barrier of immune exclusion in human biopsies

SEMA4D is expressed at tumor margin

SEMA4D binds to PLXN receptors on DC and restricts penetration

T cells are excluded from tumor



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

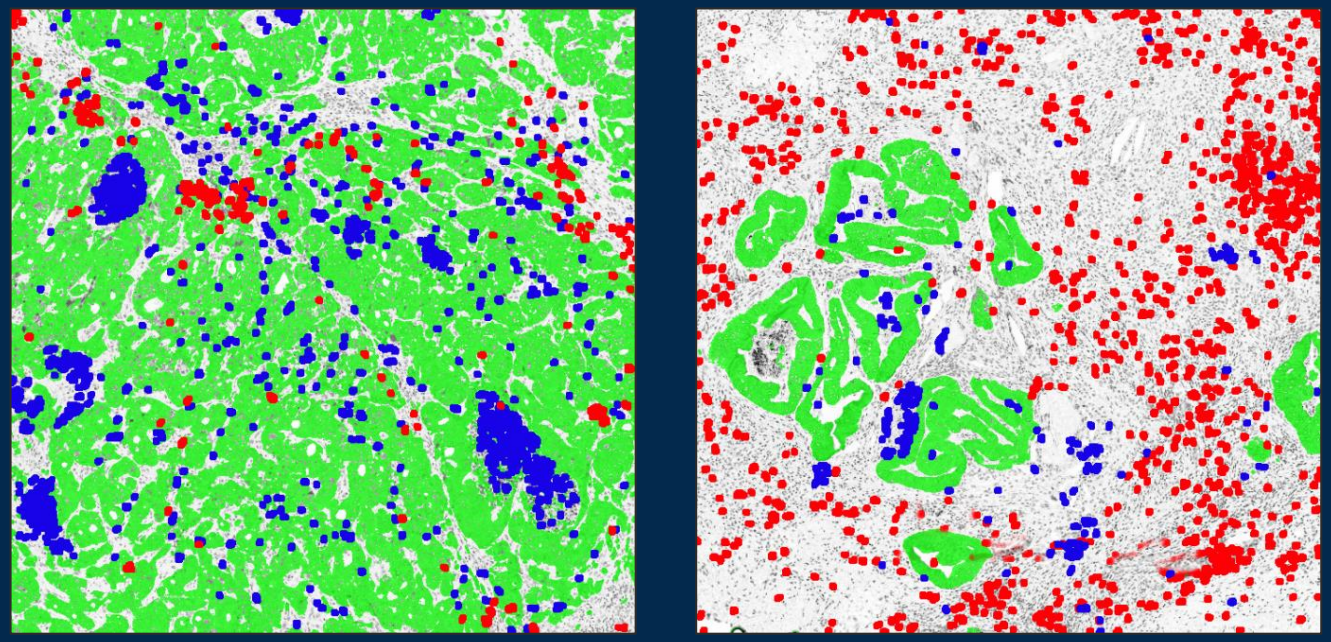
CD8 T cells align with Sema4D at the invasive edge of the tumor. Most of these excluded T-cells express Sema4D. Dendritic Cells express receptors for SEMA4D and are heavily excluded at the invasive edge.

Clinical POC - Pepinemab increases cytotoxic T cells while reducing inhibitory suppressor cells

Biopsies from patients with metastatic MSS Colorectal Cancer

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

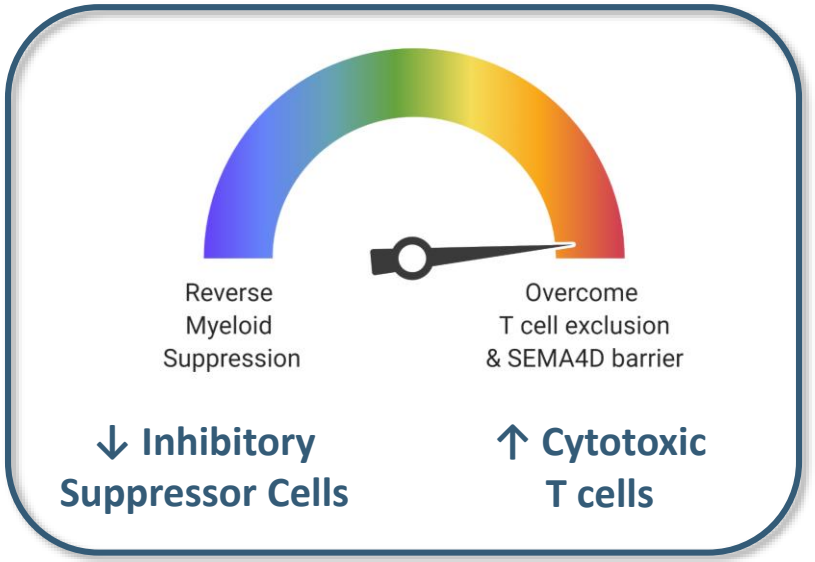
Pepinemab
High CD8+ T cells
Low tumor content and MDSC



Myeloid Derived Suppressor Cells (MDSC) Tumor CD8+ T cells

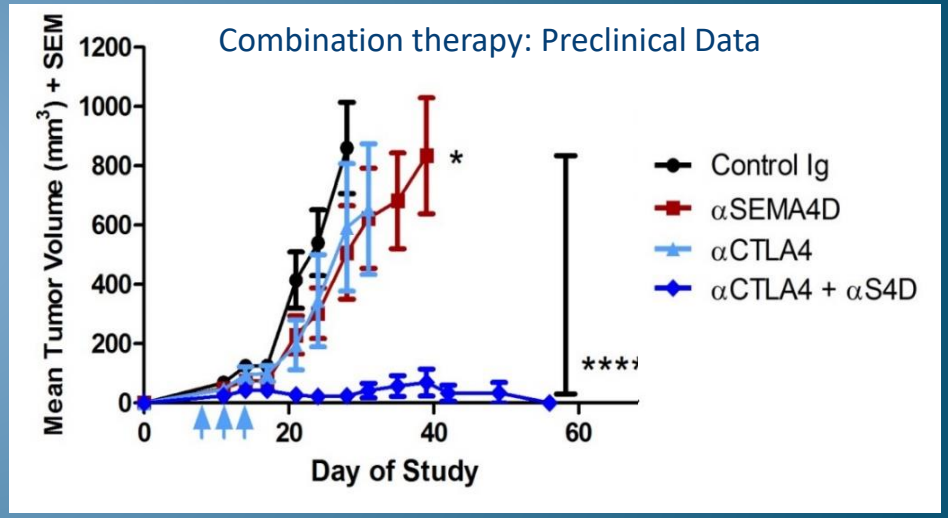
Patients received neoadjuvant chemotherapy before immunotherapy and surgery
Winship Cancer Institute, Emory University – neoadjuvant/"window of opportunity" study

Pepinemab



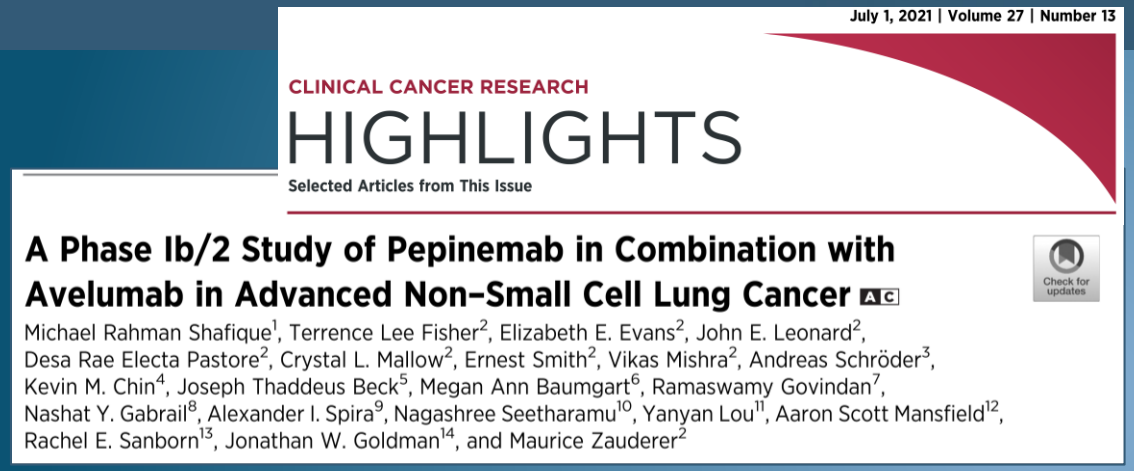
Pepinemab has a unique mechanism of action that enhances other immunotherapies

Immune Resistance 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



Pepinemab complements other immune-activating therapies - anti-PD1/L1, anti-CTLA-4, anti-LAG3, anti-TGF-β, DC vaccine, etc

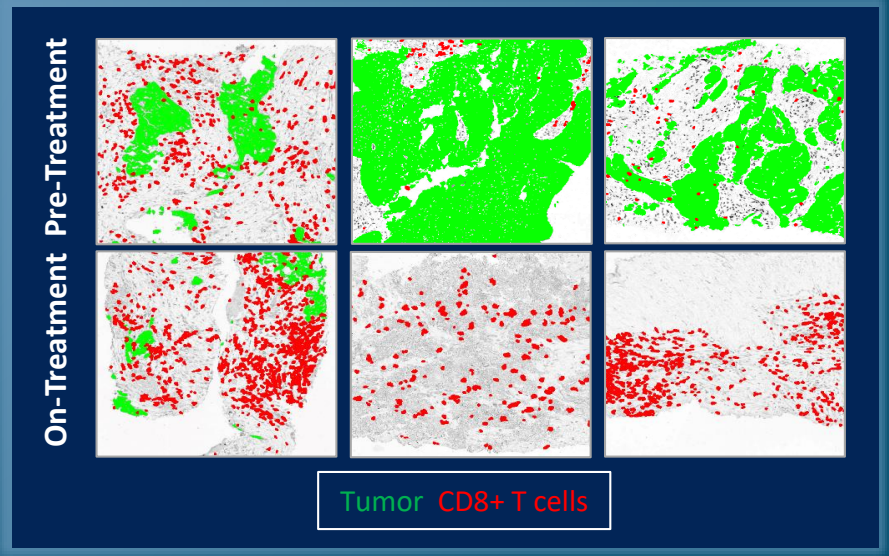
Phase 1b/2 CLASSICAL-Lung Highlights



Sponsored by: VACCINEX

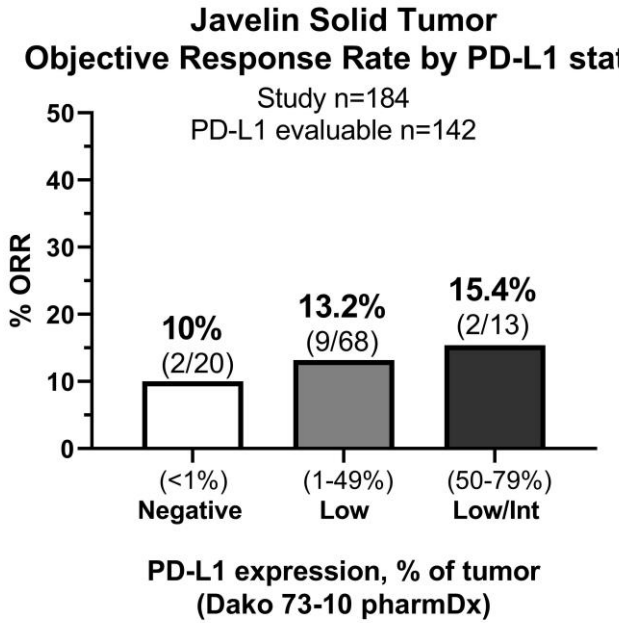
Co-funded by: MERCK

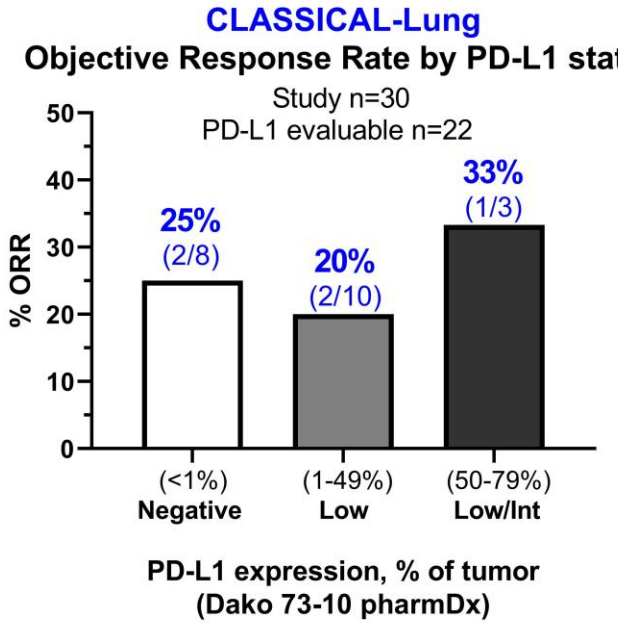
1. Well tolerated. Pepinemab does not enhance immune-related toxicities of partner drug.
2. Antitumor activity in some patients with challenging PD-L1 low or PD-L1 negative tumors
   - **Reported single agent anti-PDx: ORR ~10-15%**
   - **Combination with pepinemab: ORR 25-33%**
3. Antitumor activity 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
4. Increased penetration of cytotoxic T cells following treatment



Phase 1b/2 CLASSICAL-Lung
Objective Response Rate in patients with PD-L1 low/negative tumors

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:

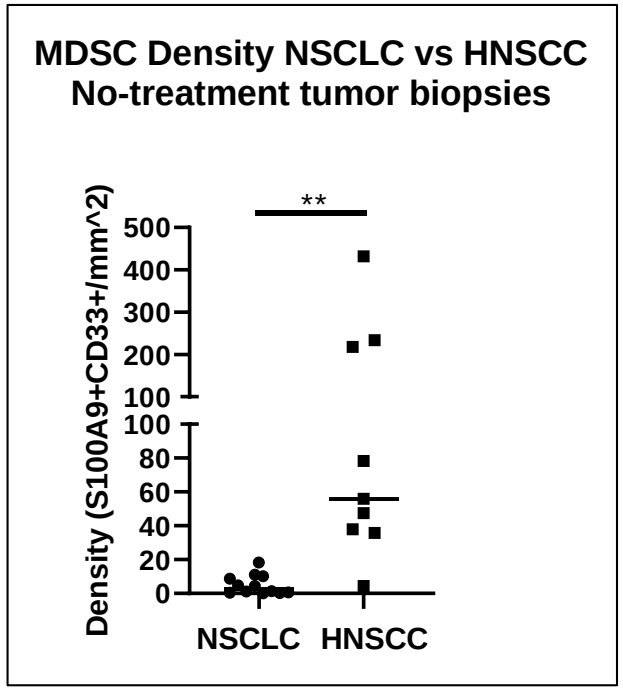
Gulley JL, Rajan A, Spigel DR, et al. Avelumab for patients with previously treated metastatic or recurrent non-small-cell lung cancer (JAVELIN Solid Tumor): dose-expansion cohort of a multicentre, open-label, phase 1b trial. *Lancet Oncol* 2017; published online March 31. http://dx.doi.org/10.1016/S1470-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

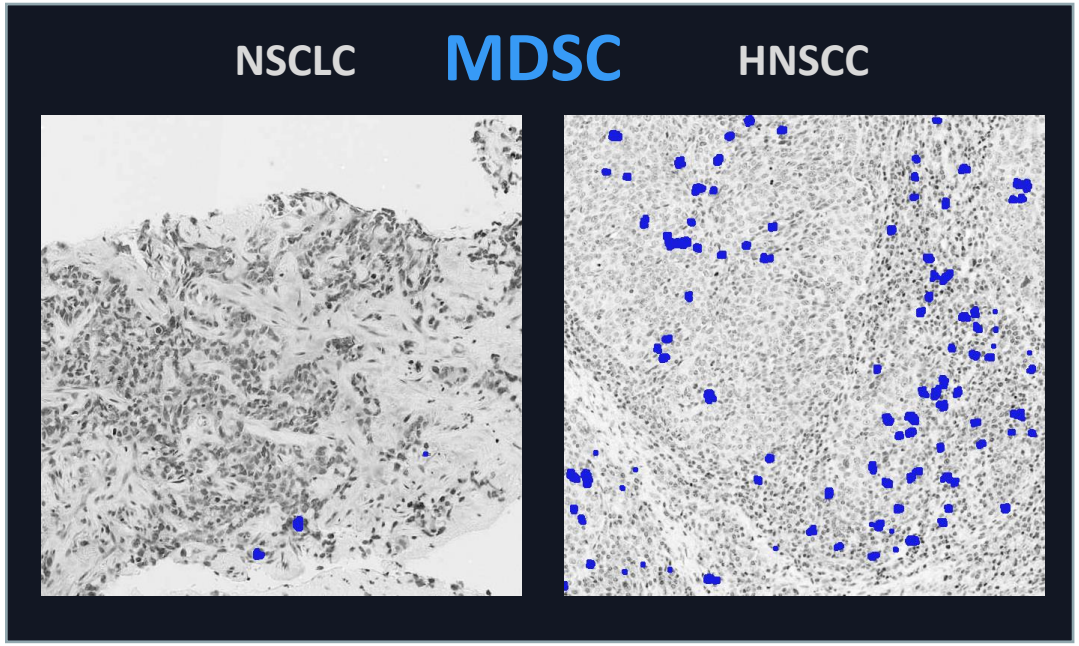
Gulley JL, Rajan A, Spigel DR, et al. Exposure Response and PD-L1 expression analysis of second-line avelumab in patients with advanced NSCLC: data from the JAVELIN Solid tumor trial: Abstract No. 9086. Presented at 53rd ASCO Annual Meeting; Jun 2-6, 2017

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.

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

- HNSCC have high MDSC content relative to NSCLC, therefore, benefit from both major pepinemab mechanisms of action.
- **Rationale**
  - SEMA4D induces high levels of myeloid derived suppressor cells (MDSC) in HNSCC
  - MDSC represent an important mechanism of resistance to immune checkpoint therapy
  - Inhibiting MDSC with pepinemab will enhance response to pembrolizumab





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 further evaluation of activity (PFS, OS, DOR), biomarkers within TME
- Data anticipated mid-2022

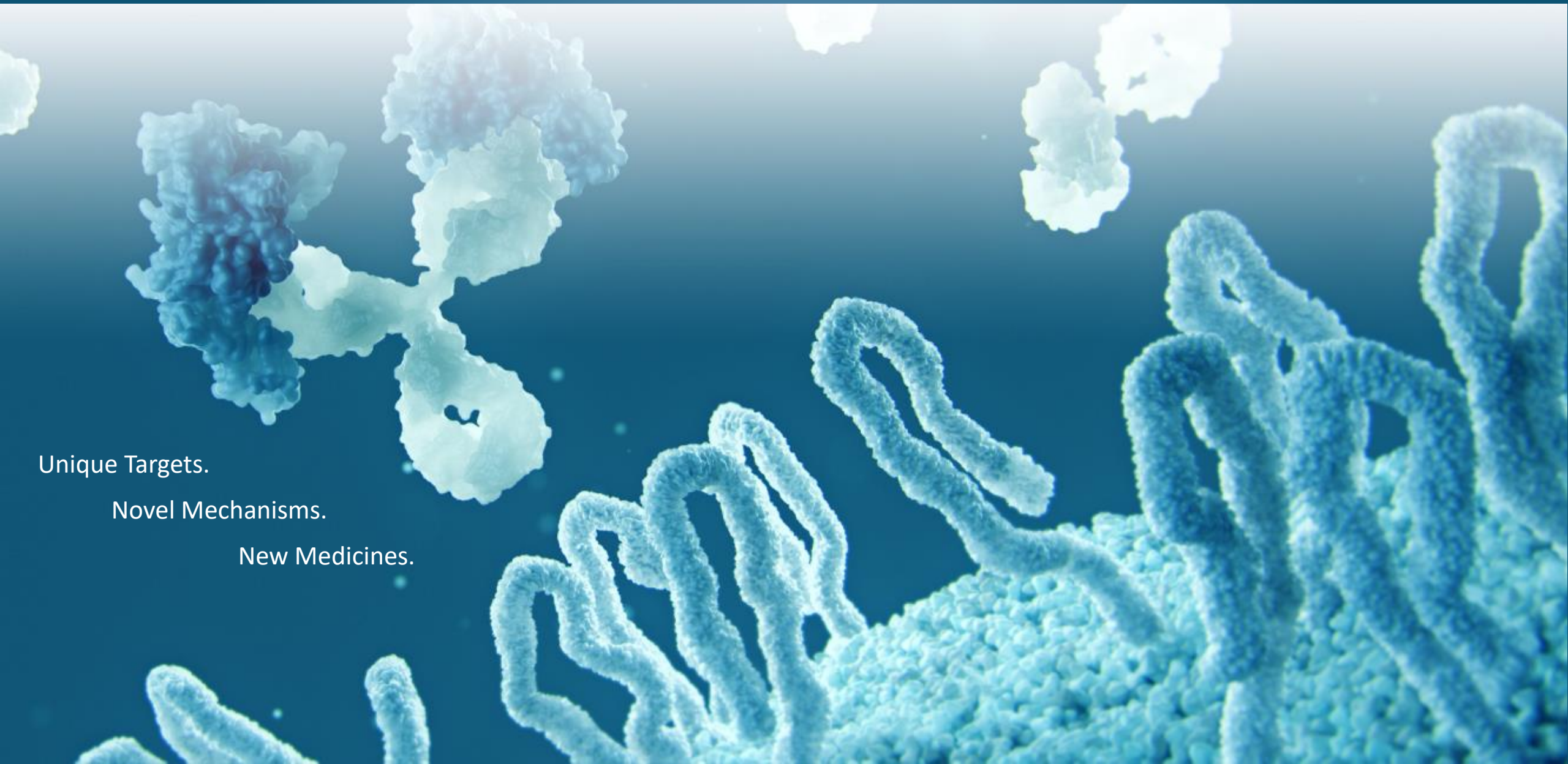
Pepinemab for Immuno-Oncology

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



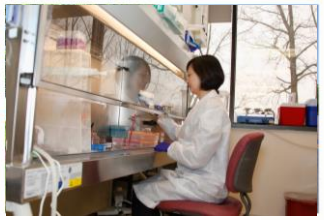
Upcoming Milestones

Final Clinical Data for SIGNAL Cohort B study in Huntington's Disease	Q2 2021
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, https://clincancerres.aacrjournals.org/content/27/13/3505	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.

Vaccinex Scientific Advisors - Neurology

Eric Siemers, MD	President of Siemers Integration LLC. Distinguished medical fellow for Eli Lilly and Company's Alzheimer's Disease Global Development Team, founded and headed the Indiana University Movement Disorder Clinic. Served on the Board of Directors of the American Society of Experimental Neurotherapeutics, as founding member and Chair of the Alzheimer's Association Research Roundtable, and Steering Committee member for the Alzheimer's Disease Neuroimaging Initiative (ADNI).
Karl D. Kieburtz, MD, MPH	President of Clintrex LLC, providing services regarding research and regulatory strategy for therapeutic development of interventions for brain disorders. Chair of the FDA Peripheral and Central Nervous System Drugs Advisory Committee and sits on the American Academy of Neurology (AAN) Clinical Research Subcommittee, the International Executive Committee of the Movement Disorders Society (MDS), the Board of Directors for the American Society for Experimental NeuroTherapeutics (ASENT), and the Council of the American Neurological Association (ANA), chair of the FDA Peripheral and Central Nervous System Drugs Advisory Committee.
Ira Shoulson, MD	Dr. Shoulson is a long time leader in Huntington's disease research. From 2011 to July 2018, Dr Shoulson was Professor of Neurology, Pharmacology and Human Science and Director of the Program for Regulatory Science and Medicine (PRSM) at Georgetown University where he was principal investigator of the FDA-Georgetown University Collaborating Center of Excellence in Regulatory Science and Innovation. From 1990 to 2011, Dr Shoulson was the Louis C. Lasagna Professor of Experimental Therapeutics and Professor of Neurology, Pharmacology and Medicine at the University of Rochester School of Medicine & Dentistry in Rochester, New York. Dr. Shoulson is an elected member of the National Academy of Medicine of the National Academy of Sciences.
Ralf Reilmann, MD	Founding Director and C.E.O. of the George-Huntington-Institute, Dept. of Radiology at the University of Muenster and the Dept. of Neurodegeneration and Hertie Institute for Clinical Brain Research at the University of Tuebingen.

Vaccinex Scientific Advisors - HNSCC Clinical Advisory Board

Barbara Burtness, MD	Professor of Medicine (Medical Oncology) at Yale, leader of the Disease Aligned Research Team for the Head and Neck Cancers Program and Co-Leader of the Developmental Therapeutics Research Program at Yale Cancer Center. Chair of ECOG-ACRIN Head and Neck Therapeutics Committee, served on the NCCN and SITC Head and Neck Guidelines Committee, and the NCI Head and Neck Cancer Steering Committee. Co-chair of the NCI Clinical Trials Planning Meeting on TP53-Mutated Head and Neck Cancer and FDA Project 2025 for Head and Neck. Founding Director of the Yale Head and Neck Cancer SPORE and has led numerous clinical trials, including the international phase III trial which led to regulatory approval of immunotherapy in first-line treatment of head and neck cancer.
Robert Haddad, MD	Chief, Division of Head and Neck Oncology. McGraw Chair, Head and Neck Oncology, Dana-Farber Cancer Institute. Professor, Medicine, Harvard Medical School.
Douglas Adkins, MD	Professor, Department of Medicine, Oncology Division, Medical Oncology, Washington University School of Medicine in St. Louis. NCI Head and Neck Steering Committee and Metastatic and Recurrent Head & Neck Cancer Task Force
Nabil Saba, MD	Director of the Head and Neck Cancer Medical Oncology Program at Winship Cancer Institute of Emory University, Professor and Vice Chair for Quality and Safety in the Department of Hematology and Medical Oncology and holds a joint appointment as Professor in the Department of Otolaryngology at Emory University School of Medicine. Chair of the National Cancer Institute's task force for recurrent metastatic head and neck cancer and Chair of the Rare Tumors Task Force of the National Cancer Institute's Head and Neck Cancer Steering Committee. Member of the NRG Oncology and Eastern Cooperative Oncology Group (ECOG) Head and Neck Cancer Core Committees, the ASCO clinical guidelines committee, and the ASCO Head and Neck Guideline Advisory Group.

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