UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, DC 20549

FORM 8-K

CURRENT REPORT PURSUANT
TO SECTION 13 OR 15(D) OF THE
SECURITIES EXCHANGE ACT OF 1934

Date of report (Date of earliest event reported): **February 16, 2017**

ABEONA THERAPEUTICS INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-15771**	**83-0221517**
(State or other jurisdiction of incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

3333 Lee Parkway, Suite 600
Dallas, TX 75219
(Address of principal executive offices) (Zip Code)

(214)-665-9495
(Registrant's telephone number, including area code)

N/A
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01. Regulation FD Disclosure

Item 8.01. Other Items

Presentation relating to Abeona Therapeutics Inc. The presentation is attached as Exhibit 99.1 and is incorporated herein.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

99.1 Presentation entitled “Abeona Therapeutics”

99.2 Press release dated February 17, 2017, entitled “Abeona Therapeutics Provides Update from ABO-102 Phase 1/2 MPS IIIA Clinical Trial at the 13th Annual WORLDSymposium™ 2017”

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Abeona Therapeutics Inc.
(Registrant)

By: /s/ Stephen B. Thompson
Stephen B. Thompson
Vice President Finance
Chief Accounting Officer

Dated: February 17, 2017

EXHIBIT INDEX

Exhibit Number

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EXHIBIT 99.1

Abeona
THERAPEUTICS

Working together to find a cure.

NASDAQ: ABEO
www.abeonatherapeutics.com

Safe Harbor Statement

This presentation contains certain statements that may be forward-looking within the meaning of Section 27a of the Securities Act of 1933, as amended, including statements relating to the product portfolio and pipeline and clinical programs of the company, the market opportunities for the all of the company's products and product candidates, and the company's goals and objectives. These statements are subject to numerous risks and uncertainties, including but not limited to the risks detailed in the Company's Annual Report on Form 10-K for the year ended December 31, 2015, and other reports filed by the company with the Securities and Exchange Commission.

This presentation does not constitute an offer or invitation for the sale or purchase of securities or to engage in any other transaction with Abeona Therapeutics or its affiliates. The information in this presentation is not targeted at the residents of any particular country or jurisdiction and is not intended for distribution to, or use by, any person in any jurisdiction or country where such distribution or use would be contrary to local laws or regulations. The Company undertakes no obligations to make any revisions to the forward-looking statements contained in this presentation or to update them to reflect events or circumstances occurring after the date of this presentation, whether as a result of new information, future developments or otherwise.

We have filed a shelf registration statement on Form S-3 (including a prospectus) with the SEC which was declared effective on July 23, 2015. Before you invest in the offering to which this communication relates, you should read the prospectus in that registration statement and the preliminary prospectus supplement related to this offering and the other documents the issuer will file with the SEC for more complete information about the issuer and this offering. You may get these documents for free by visiting EDGAR on the SEC Web site at www.sec.gov. Alternatively, you may obtain a preliminary prospectus by calling 1-877-547-6340.

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Investment Highlights

A World Leader in Rare Disease Therapies

Strategic Focus on Rare Diseases	• Substantial unmet medical need to treat life threatening diseases caused by a defect in a single gene • Most advanced programs in neurological / lysosomal storage diseases where there are no treatment options • Orphan and Rare pediatric disease focus: shorter route to approval, and lower costs • Opportunity to secure Priority Review Voucher (PRV) for rare pediatric diseases
Three Clinical Programs in Phase I/II	• ABO-102: Sanfilippo syndrome Type A – MPS IIIA. Lysosomal storage disease affecting children. • ABO-101: Sanfilippo syndrome Type B – MPS IIIB. Lysosomal storage disease affecting children. • EB-101: Recessive dystrophic epidermolysis bullosa (RDEB). Severely fragile skin disease affecting children.
Robust Pipeline With Multiple Therapies Soon Entering the Clinic	• Based on strong pre-clinical results, two additional programs are expected to begin Phase I/II by the end of 2017 including: – ABO-201: Juvenile Batten disease (CLN3) – ABO-202: Infantile Batten disease (CLN1)
Proprietary Vector Platform For New Product Development	• 60+ proprietary AAV vectors that uniquely enable the delivery of genes across the blood brain barrier making IV administration possible • Next generation chimera vector platform in development with increased gene delivery efficiency, reduced antibody affinity, enhanced ability to create immune responses and target tissue specifications
Several Catalysts will Drive Significant Near Term Value	• Achieve fast track designation for additional programs • Initiate high dose cohort in ABO-102 for MPS IIIA • Initiate and enroll 1st patient in ABO-101 for MPS IIIB Phase 1/2 clinical studies • Dose adolescents in EB-101 trial; secure orphan drug and rare pediatric disease designations

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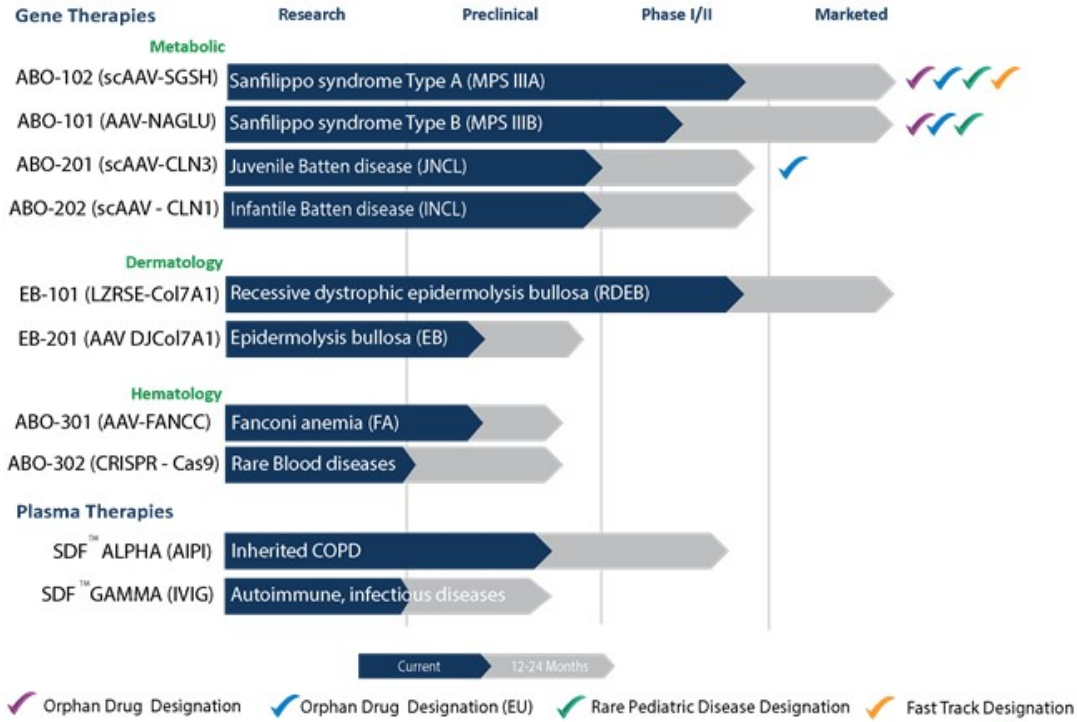
Rare Disease Pipeline



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Focus on Gene Therapy

What, Where and How?

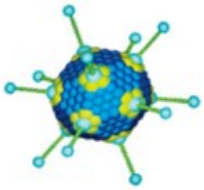
What is Gene Therapy?

- The transplantation of normal genes into cells in place of missing or defective ones in order to correct genetic disorders
- Excitement in gene therapy sector due to recent European approvals of two gene therapies, impressive clinical data and anticipated first approvals of gene therapies in the US

Where?

- Target diseases that occur from single gene defects (mono-genetic defects)

How?



- Delivery, delivery, delivery: Viruses are used as transport vectors to get correct gene sequence into cells; choice of viral vector with affinity to target tissues; constructs which result in robust, uniform expression
- Route of administration: Important to choose route of administration consistent with disease pathology; in Sanfilippo A, Abeona uses a single intravenous injection for whole body and CNS distribution

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AAV Based Gene Therapy Platform

Vector crosses the Blood Brain Barrier

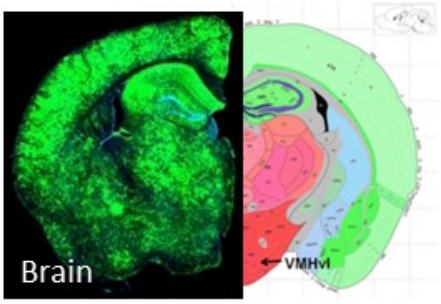
- Expressed in multiple nervous system cell types
- Supra-physiological enzyme expression in brain for over a year observed in animals

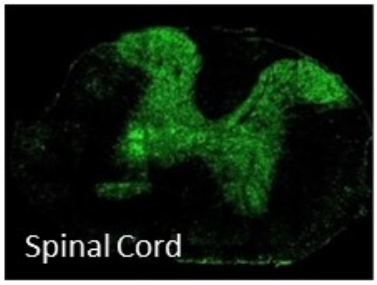
Self-complementary AAV (scAAV) persists as a stable episome in non-dividing cells with transgene expression for years

scAAV vectors are 10-100-fold more efficient than traditional single-stranded (ss) AAV vectors

- Robust, uniform expression in all areas of brain and peripheral tissues

Enables single intravenous injection therapies





Sources: Keilan et al lab.; Eliason SL, Stein CS, Mao Q, et al. A knock-in reporter model of Batten disease. J Neurosci. 2007;27(37):9826-9834.

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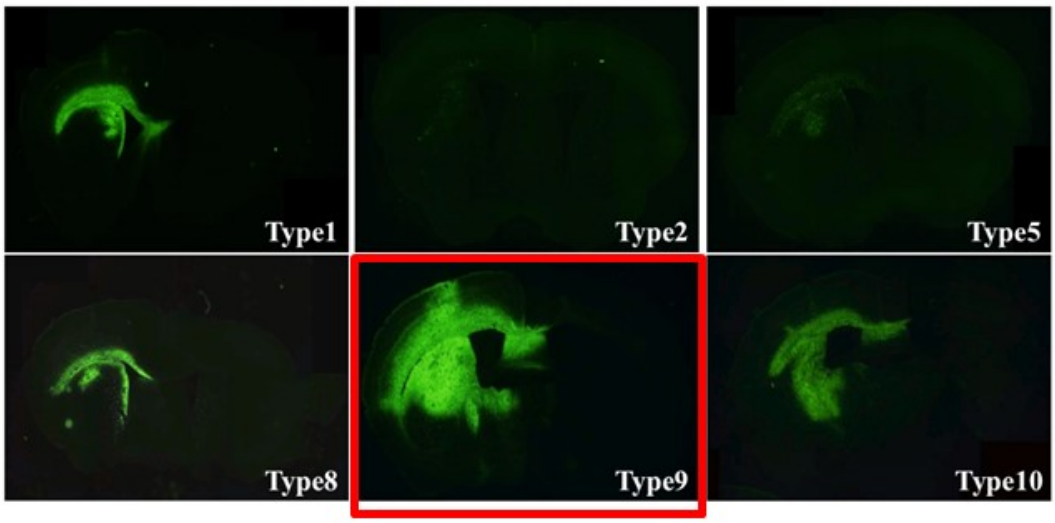
AAV 1, 2, 5, 8, 9, 10 into the CNS: Serotype makes a difference



Brain-directed injection of AAV vectors encoding green fluorescent protein gene (AAV/GFP). Approximately 2.0 x 10^{10} vg of AAV/GFP vectors (serotypes 1, 2, 5, 8, 9, and 10) were injected into the right striatum over a period of 5 min. Expression of GFP was analyzed using fluorescent microscopy at 2 weeks

Source: Miyaki et al., Medicine » Medical Genetics » "Gene Therapy - Principles and Challenges" book edited by Doaa Hashad, ISBN 978-953-51-2221-0, Published: November 26, 2015

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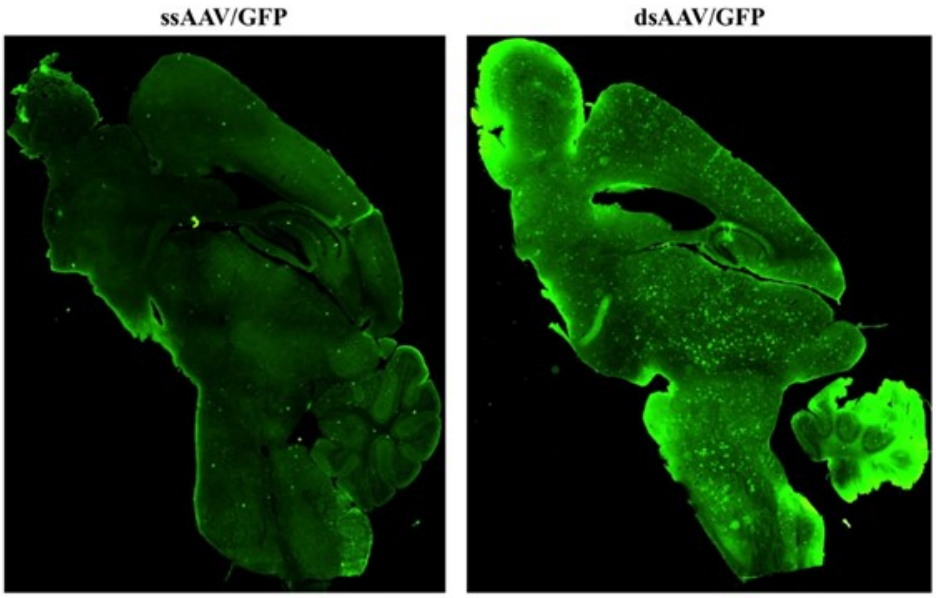
Single Stranded vs. Self-Complementary AAV



After 7.0 x 10^{12} vg of AAV9/GFP vectors were injected via tail veins of adult (7-week-old) mice; GFP images 5 weeks post administration

More Green = More Gene

Source: Miyaki et al., Medicine » Medical Genetics » "Gene Therapy - Principles and Challenges", book edited by Doaa Hashad, ISBN 978-953-51-2221-0, Published: November 26, 2015

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Global Foundation Support

Investors and ongoing supporters of Abeona

★ Original founding foundations

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Clinical-Stage Programs

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Sanfilippo Syndromes (MPS IIIA and IIIB)

Rare lysosomal storage disease affecting children

Sanfilippo syndrome (MPS IIIA & IIIB) is an inherited monogenic disorder that causes lysosomal enzyme deficiency

- Set of four different disease states, each categorized by a missing or deficient enzyme
  - MPS IIIA (SGSH)
  - MPS IIIB (NAGLU)
- Results in abnormal accumulation of glycosaminoglycans (GAGs) (sugars)
- Aggressive behavior, seizures, loss of speech/vision, inability to sleep
- Deterioration is severe, progressive and universally lethal
- Death by end of teens–early 20's

Nationwide Children's
When your child needs a hospital, everything matters.

No treatment options currently available

- Incidence is estimated to be 1 in 70,000 births
- Types A and B more common in North America and Europe

Two ongoing clinical trials

- ABO-102 (scAAV-SGSH) for MPS IIIA – low-dose cohort (n=3) completed, high-dose enrollment has commenced
- ABO-101 (AAV-NAGLU) for MPS IIIB – Recruitment anticipated 2Q17
- Conducted at Nationwide Children's Hospital, Columbus, OH

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Natural History Study

25-patients with Sanfilippo syndrome assessed

Enrollment complete: 25 subjects, 15 MPS IIIA & 10 MPS IIIB

Study visits – assessments at 0, 6, and 12 months:

- Neurocognitive (Leiter) and parental rating assessments (ABAS II)
- Timed functional motor tests
- Standard laboratory assessments
- Serum/leukocyte NAGLU or SGSH activity
- Quality of life (PedsQL)
- Urine and CSF GAG (heparan sulfate) levels

Study visits – assessments at baseline and 12-month assessments

- Brain, liver and spleen MRI (including DTI and 1H spectroscopy)
- CSF for standard chemistries/cell counts and NAGLU or SGSH activity

All subjects through >1 year follow up

Multiple patient cross-over into clinical trials

Truxal, K.V. et al. "A Prospective One-Year Natural History Study Of Mucopolysaccharidosis Types IIIA And IIIB: Implications For Clinical Trial Design". Molecular Genetics and Metabolism 119.3 (2016): 239-248.

Phase 1/2 ABO-102 (scAAV-SGSH) Clinical Trial for MPS IIIA

Global open-label, dose-escalation gene transfer trial

Phase 1/2 open-label, dose-escalation clinical trials

- Low dose: n = 3 patients; high dose: n = 3-6 patients
- Three sites: United States, Spain and Australia

Two dose cohorts of n=6-9 pts via intravenous injection

- Cohort 1 (Low Dose): 5 X 10^{12} vg/kg (n=3 subjects) – ENROLLMENT COMPLETE
- Cohort 2 (High Dose): 1 X 10^{13} vg/kg (n=3-6 subjects) – ENROLLMENT HAS COMMENCED

Primary objective is determination of safety, with secondary objectives

- Reduction in Urine/CSF GAG (heparan sulfate), hepatomegaly and splenomegaly
- Leukocyte SGSH enzyme activity levels
- Leiter International Performance Assessment after 6 months post treatment
- Parent ratings: Vineland Adaptive Behavior Assessment System and Child Behavioral Checklist
- Pediatric Quality of Life Inventory 4.0 (PedsQL)

Dosing and Short-Term Monitoring

- Day -1: subjects receive first dose of oral prednisolone elixir at 1 mg/kg/day
- Day 0: subjects receive gene transfer delivered under sedation within the Procedure Center over 15 minutes
- First 48 hours: Subjects monitored in PICU for 24 hours, then discharged to neurology floor for another 24 hours monitoring prior to discharge
- Discharged on oral prednisolone elixir

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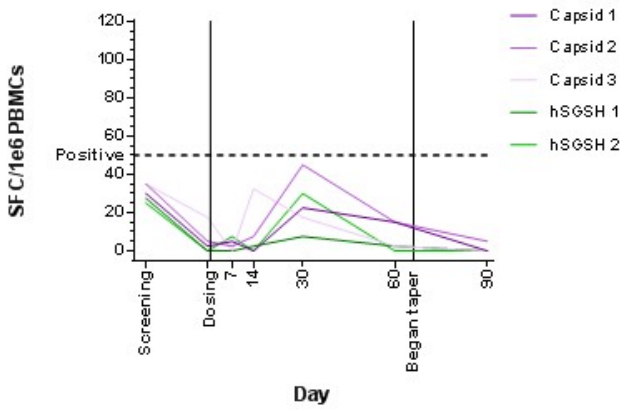
No subjects to date have had a serious adverse event (SAE) through 644 days cumulative post-injection

- Upper respiratory infection most common AE (n=3, judged unrelated)
- Irritability (n=2) attributed to prednisolone dosing
- IFN-γ ELISpot stable through 3 patients



Representative IFN ELISpot

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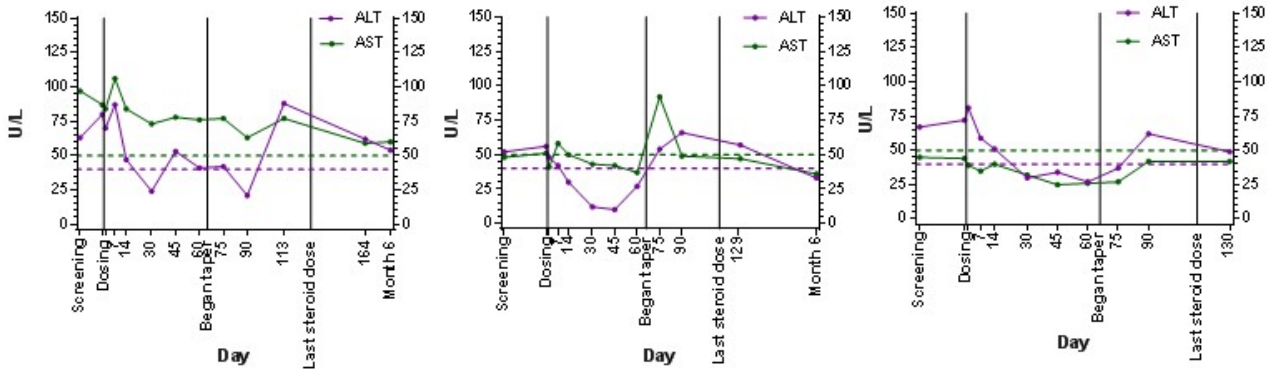
Serum transaminases initially decline, and remain largely stable through 6 months post-injection



Dotted lines represent upper limits of normal for AST (green) and ALT (purple).

From the recent NCH natural history study, elevations in AST levels (up to 1.45 x ULN) and ALT levels (up to 3.2 x ULN) were seen at baseline, and were not correlated with age or degree of hepatomegaly.

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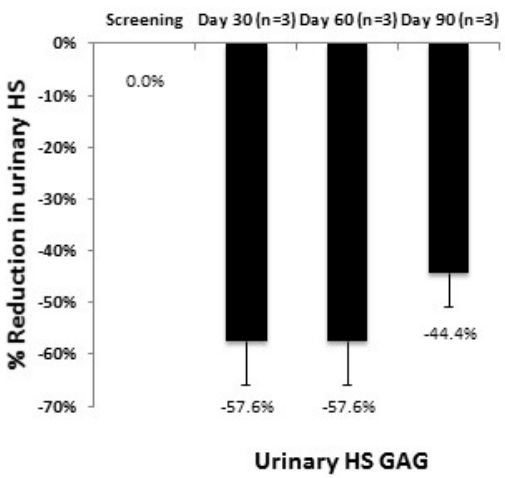
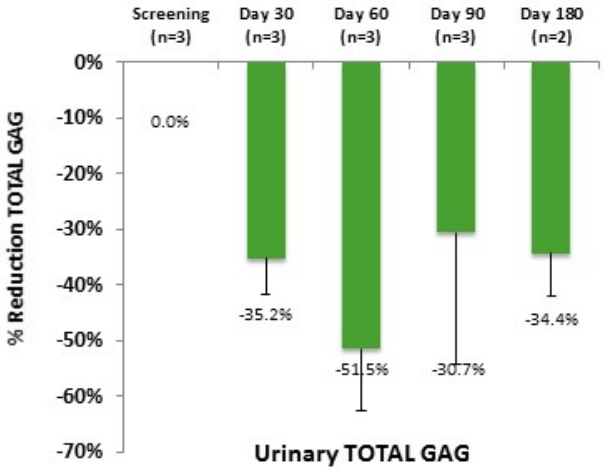
ABO-102 Reduced Heparan Sulfate (HS) and Total Urinary GAG





Urine glycosaminoglycans (GAGs) normally decrease as a function of age among unaffected individuals

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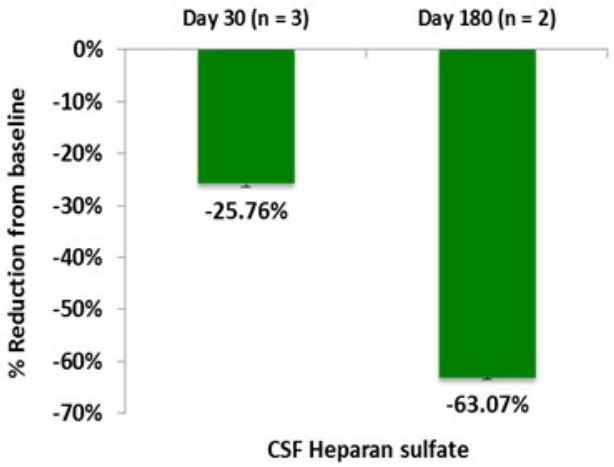
Reduction of CNS HS - Evidence of Biopotency

CSF HS Fragments Decrease After Treatment



- Reduction of CSF HS fragment indicates ABO-102 crosses the blood-brain barrier
- No CSF GAG reduction was reported in a separate MPS IIIA Gene Therapy trial that has been discontinued
- Continuous, sustained reductions (rather than "sine wave -like" fluctuations associated with ERT, when the half-life of the enzyme is short) could enable the "down regulation" of the inflammatory response and allow "repair mechanisms" to begin

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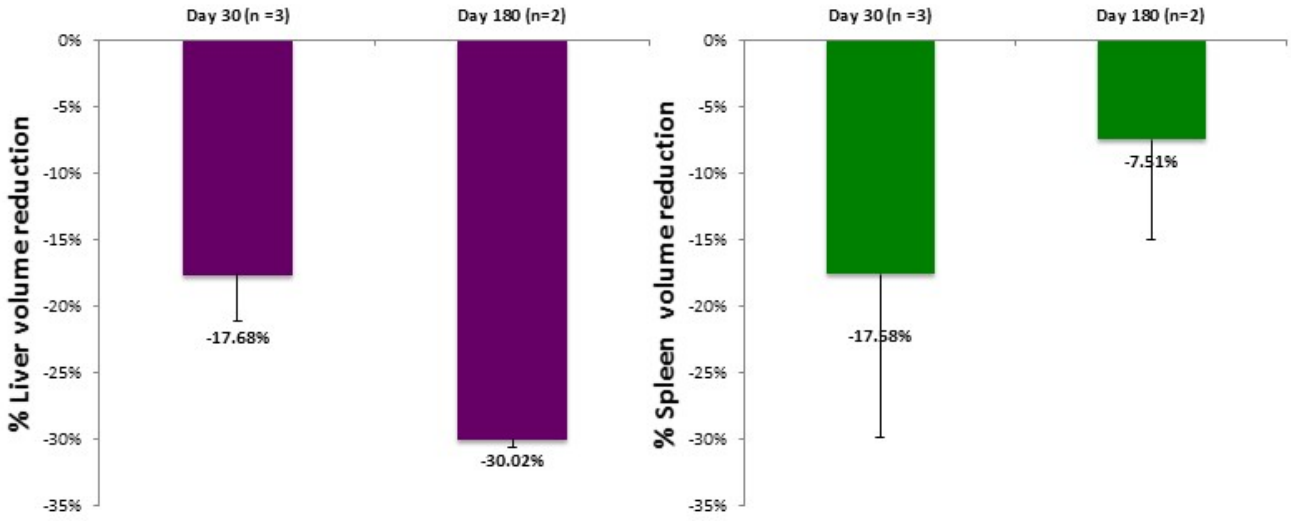
ABO-102 Reduced Liver and Spleen Volumes

30 (n=3) and 180 days (n=2) post-injection assessed by MRI



Source: Wainstein N.et al. Am Med. 2002113:112-119
http://www.fda.gov/downloads/drugs/developmentapprovalprocess/howdrugsaredeveloped andapproved/approvalapplicationstherapeuticbiologicapplications/ucm083695.pdf
http://www.accessdata.fda.gov/drugsatfda_docs/label/2014/021348s010lbl.pdf

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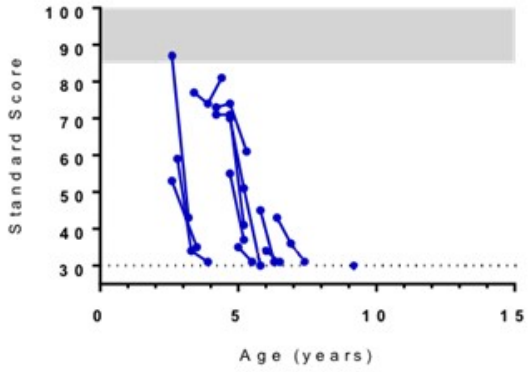
Evidence of Biopotency & Clinical Benefit

Leiter-3 Nonverbal IQ Scale

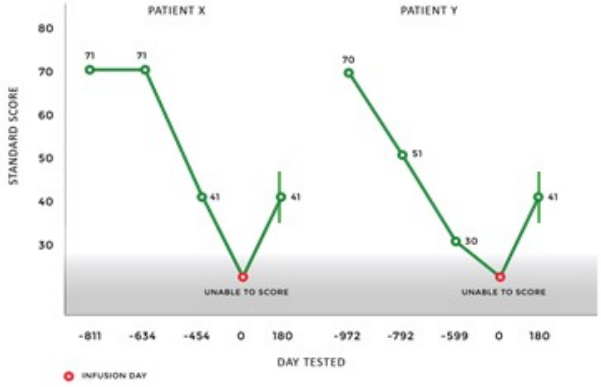
Natural History Study

NHS Leiter–3 (MPS IIIA)



ABO-102 Clinical Study

Leiter-3 with ABO-102



- The Leiter International Performance Scale Third Edition (Leiter-3) is a test of nonverbal intellectual ability for ages 3 and above.
- The Leiter–3 does not require any translations for non English speakers and is suitable for use in children with hearing deficits, given that all directions are provided by gestures and the child responds by placing cards or pointing.

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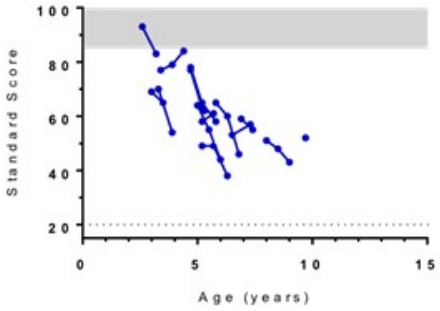
Evidence of Biopotency & Clinical Benefit

Vineland Composite Score

Natural History Study

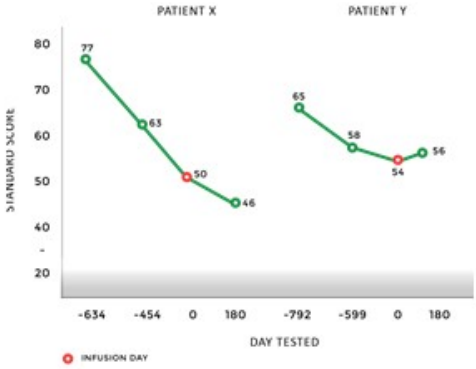
Vineland Composite Score (MPSIIIA)



- **The Vineland Adaptive Behavior Scales, Second Edition (Vineland-II) measures:**
  - Adaptive behaviors
  - Maladaptive behaviors
  - Social and personal skills needed for everyday living

ABO-102 Clinical Study

Vineland Score with ABO-102



Vineland Score with ABO-102

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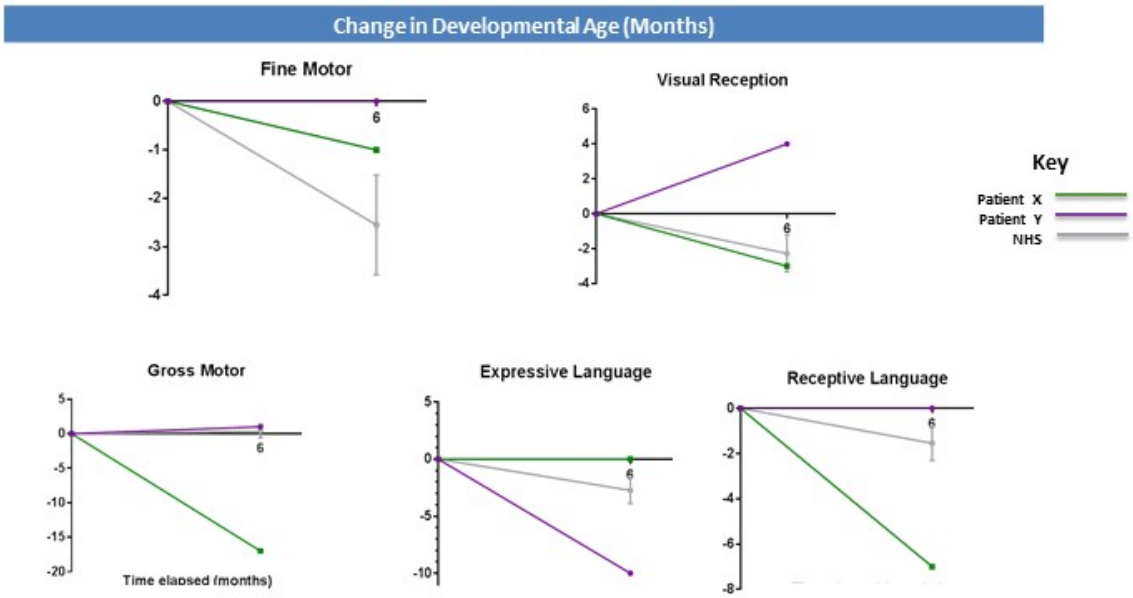
Evidence of Biopotency & Clinical Benefit

Mullen Scale Scores at Six Months Post-Treatment (N=2)



The Mullen Scales of Early Learning, a five domain ability assessment (Gross Motor, Fine Motor, Visual Reception, Receptive Language, Expressive Language) assesses modality performance and identifies learning ability, learning disability, and mental retardation in children between 21 and 63 months of age.

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Summary of ABO-102 Clinical Data

ABO-102 for MPS IIIA is Well Tolerated, and Demonstrates Biopotency and Early Signals of Neurocognitive and Behavioral Improvements

- ***ABO-102 is well tolerated to date, with no serious adverse events (N=4 through 650 days follow up)***
- ***All low dose subjects (N =3) tapered off immunosuppression regimen on or before 90 days (unlike other gene therapy trials by direct intracranial administration where immunosuppression has lasted 12 to 18 months)***
- ***Significant, continuous reduction in HS GAG as measured in the CSF (thought to be the key measure of biopotency/reduction of storage pathology in the CNS) in the low dose cohort (N=2) 6 months post-injection***
- ***Significant reductions in liver and spleen volumes; potential clinical benefit in patient population where hepatosplenomegaly can lead to recurring respiratory and digestive issues***
- ***Early positive signals of neurocognitive assessments– including early signs of improved non-verbal IQ scores, stabilization of previously declining measurements, and possible slowing of disease progression***

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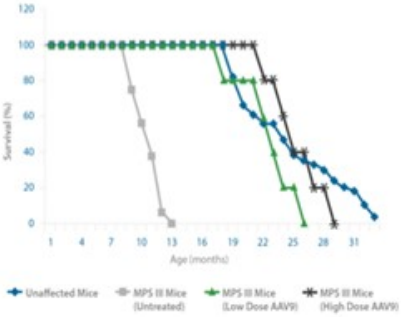
ABO-101 for MPS IIIB

Second FDA allowed IND; clinical trial to commence shortly

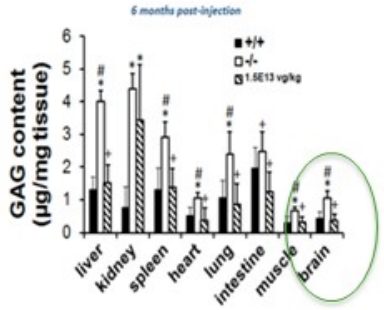
Pre-clinical data

- ABO – 101 Single iv infusion of ABO-101 at 4-6 weeks of age normalized the survival in MPS IIIB mice
- Single IV Delivery of ABO-101 induced clearance of lysosomal GAG storage in the CNS and somatic tissues
- Demonstrates that treated IIIB animals have normalized GAG content, similar to unaffected animals, in multiple tissues compared to untreated IIIB animals (with exception of kidney)



Phase 1/2 open-label, dose-escalation clinical trials

- Two dose cohorts of n=6-9 pts via intravenous injection
- Cohort 1 (Low Dose): 2 X 10^{13} vg/kg (n=3 subjects)
  - ✓ IND ALLOWED; ENROLLMENT TO COMMENCE IN 2Q17
- Cohort 2 (High Dose): 5 X 10^{13} vg/kg (n=3-6 subjects)
- Three sites: United States, Spain and Australia



Source: Fu et al (2011) Correction of Neurological Disease of Mucopolysaccharidosis IIIB in adult mice by rAAv9 Trans-Blood-Brain Barrier Gene Delivery, Mol. Ther 19

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Epidermolysis Bullosa (EB)

Rare and devastating skin disease

Group of devastating inherited connective tissue diseases; characterized by skin blisters and erosions that can also affect internal organs

- There are four forms of EB; EB-101 targets Recessive Dystrophic Epidermolysis Bullosa (RDEB), an extremely rare form of EB, which is caused by the absence of the COL7A1 gene (encodes type 7 collagen fibril that anchors skin to the underlying stroma)
- RDEB is the most severe form and is characterized by severe blistering, open wounds, and scarring in response to minor friction to skin

No FDA approved therapies currently available

- Prevalence of EB is estimated to be 30,000-40,000 of which RDEB is estimated to be 2,500-3,000 in the US alone
- With no FDA approved treatments, any reduction in disease symptoms or improvements in wound healing could be considered clinically meaningful

Ongoing clinical trial

- Conducted at Stanford University School of Medicine
- Phase 1 completed; strong 12-month post treatment data on initial 4 patients
- Phase 2 trial: enrolling patients

STANFORD SCHOOL OF MEDICINE
Stanford University Medical Center

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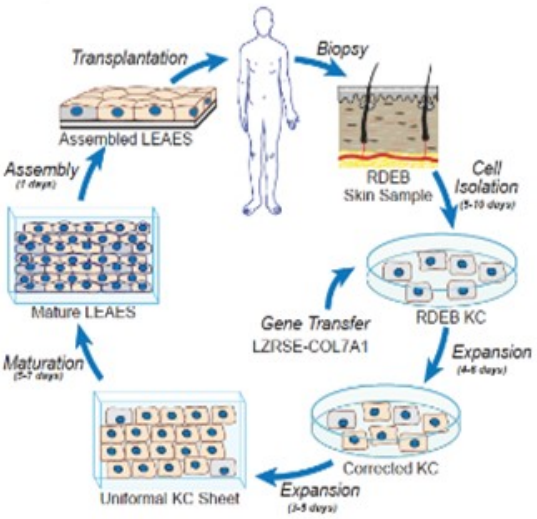
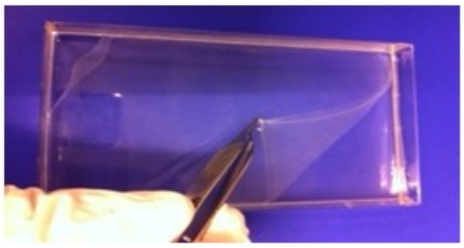
Abeona's EB-101 Approach

Genetically corrected autologous skin grafts

***EB-101: Ex-vivo* gene therapy**

Gene therapy correction of patient cells
To replace COL7A1 cDNA, followed by grafting





EB-101 – Gene therapy corrected skin sheets

EB-101 – Skin graft ready for application

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EB-101 Clinical Program Update

Unprecedented wound healing in this population

STANFORD SCHOOL OF MEDICINE
Stanford University Medical Center

Phase 1 clinical trial in with Recessive Dystrophic Epidermolysis Bullosa (RDEB) - completed using genetically corrected autologous skin grafts

- 5 adult patients treated with six grafts each
- To date, positive 12 month post-treatment data observed; de-risked outlook for pivotal Phase 2
  - All 24 grafts were well tolerated by all subjects, and no serious adverse events were reported
  - At 3 months, 87% (21/24 grafts) showed significant wound healing as defined by 75% healing compared with baseline
  - At 6 months, 67% (16/24) showed significant healing
  - Well tolerated with no SAEs
  - Phase 1 clinical trial data published in the Journal of American Medical Association (JAMA)

Ongoing phase 2 clinical trial – enrolling and treating patients

- Primary endpoints:
  - Expression of C7 and restoration of anchoring fibrils at 3 and 6 months
  - Absence of systemic virus at 3, 6, 12 months post grafting

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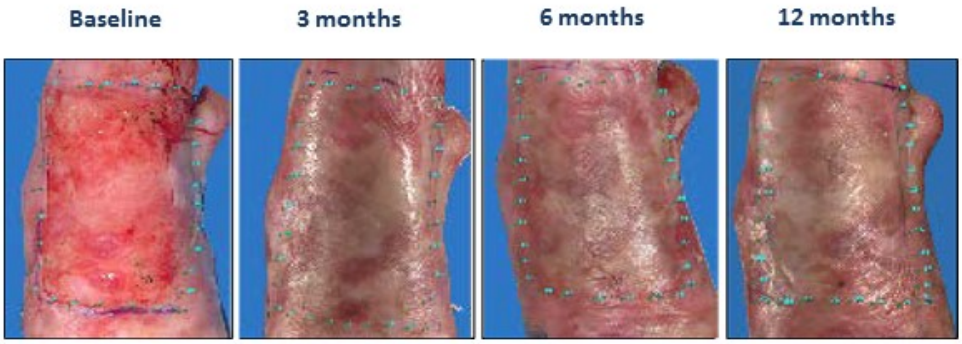
JAMA. 2016; 2016 (17): 1808-1817. doi:10.1001/jama.2016.15588

EB 101 Phase 1 Clinical Trial Results

Subject wounds pre-and post grafting



Site	Location	Description	Estimated duration	3m	6m	12m
A	R lateral hand	Erosion	3-5 yrs			
B	R medial hand	Scar tissue	3-5 yrs			
C	L ventral foot	Erosion and scar	3-5 yrs			
D	L hand	Scar tissue	3-5 yrs			
E	R foot	Erosion and scar	3-5 yrs			
Z	L ventral foot	Induced wound	New			

≥75% healed

50-70% healed

< 50% healed

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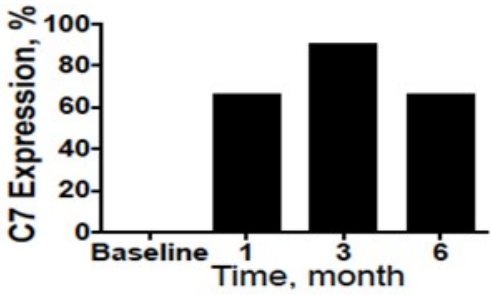
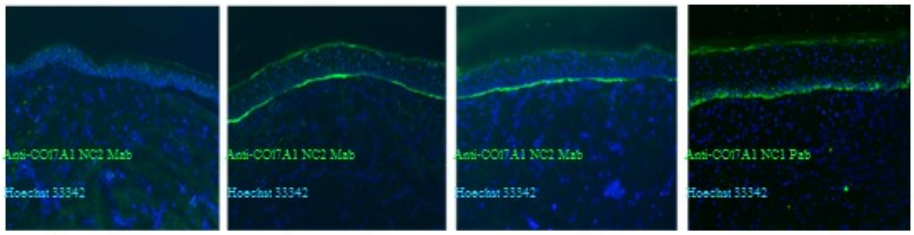
Siprashvili, Zurab et al. "Safety And Wound Outcomes Following Genetically Corrected Autologous Epidermal Grafts In Patients With Recessive Dystrophic Epidermolysis Bullosa". *JAMA* 316.17 (2016): 1808. Web.

EB-101 Phase 1 Clinical Trial Results

Induces sustained C7 levels at follow up





baseline | 3 months | 6 months | 12 months

Biopsies of grafts at follow up visits indicate high levels of C7 expressions

Current as of 10/26/15

Siprashvili, Zurab et al. "Safety And Wound Outcomes Following Genetically Corrected Autologous Epidermal Grafts In Patients With Recessive Dystrophic Epidermolysis Bullosa". *JAMA* 316.17 (2016): 1808. Web.

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Pre-Clinical Programs

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Gene Therapies

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ABO – 201 for Juvenile Batten Disease – CLN3

pre-IND meeting completed, clinical trial to commence in 2017

JBD - Autosomal recessive (inherited) mutation in the CLN3 gene

- Initially presents as blindness, progressing to behavioral issues, sleep disturbances, seizures, cognitive loss, motor abnormalities, premature death (late teens-early 20s)
- Neurodegeneration occurs primarily in thalamus, cortex, and hippocampus, although inclusions are observed throughout the CNS
- Estimated incidence of 1:100,000 births

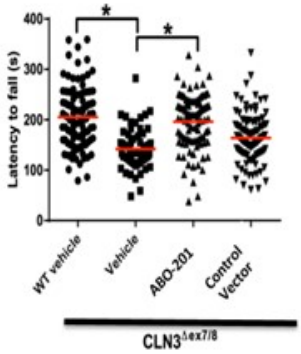
Pre-clinical results mirrored data in MPS IIIA & B; normalization of motor function and survival

- Research conducted at the University of Nebraska Medical Center

ABO-201: Human clinical trials anticipated in 2017 at University of Rochester Medical Center

Ongoing Batten Disease Natural History Study at University of Rochester Medical Center

UNMC
UNIVERSITY OF NEBRASKA MEDICAL CENTER



Source: Moving towards therapies for Juvenile batten disease, Experimental Neurology, 2008

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ABO–202 for Infantile Batten Disease – CLN1

Autosomal recessive (inherited) mutation in the CLN1 gene

Diagnosed between ages 6 months and 2 years – with early death before age 5

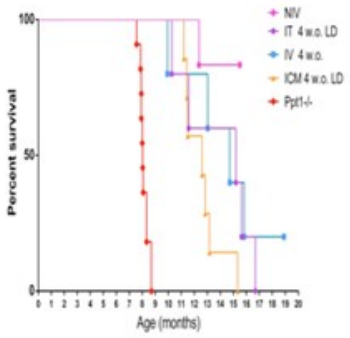
- Affected children fail to thrive and have microcephaly; also typical are short, sharp muscle contradictions called myoclonic jerks

Preclinical work shows promising efficacy in INCL mice after delivery of a functioning copy of the CLN1 gene to cells of the central nervous system

- Extended survival and preserved strength when administered early in the disease course
- Research conducted at the University of North Carolina at Chapel Hill

ABO – 202 Human clinical trials anticipated 2017

THE UNIVERSITY of NORTH CAROLINA at CHAPEL HILL



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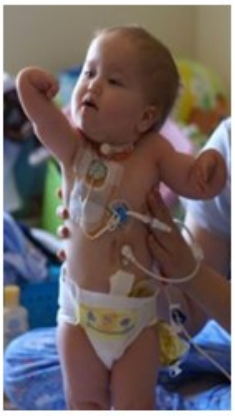
ABO-301 – for Fanconi Anemia

Fanconi anemia (FA) is a rare (1: 160,000) pediatric, autosomal recessive (inherited) disease

Characterized by multiple physical abnormalities, organ defects, bone marrow failure, and a higher than normal risk of cancer—with 20 to 30 year average lifespan



- The major function of bone marrow is to produce new blood cells. In FA, a DNA mutation renders the FANCC gene nonfunctional
- Loss of FANCC causes patient skeletal abnormalities and leads to bone marrow failure
- Higher rates of hematological diseases, such as acute myeloid leukemia (AML) or tumors of the head, neck, skin, gastrointestinal system, or genital tract

ABO-301- demonstrates in vivo efficacy in multiple models, with no off target effects

- Research conducted at University of Minnesota

Next steps: Complete IND enabling pre-clinical studies; and establish safety and preliminary efficacy in human subjects

AIM™- Next Generation AAV Vector Platform For Future Programs

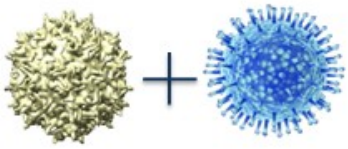
Engineered designer vector

AIM™ Vector Platform

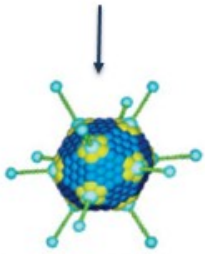
- Abeona has a next generation AAV viral vector platform to target CNS and other tissues with increased efficiency and greater tissue specificity
- The next generation AIM™ Vector is a designer vector engineered by direct evolution of natural AAV Vectors

Key Advantages of AIM™

- ✓ Increased gene delivery efficiency
- ✓ Target tissue specifications
- ✓ Potential of reducing antibody affinity
- ✓ Enhance ability of the virus to create immune responses
- ✓ Engineered to be superior than natural AAV serotypes



Natural AAV Serotypes



Proprietary AIM™ Vector

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Manufacturing Strategy

Parallel paths enable clinical trial and commercial long-term sustainable supply

A combination of third-party and internal manufacturing sources to ensure long-term continuous supply for domestic and international clinical trials as well as commercial demand.

Third Parties	Utilizing established Contract Manufacturing Organizations (CMOs) • Nationwide Children's Hospital, UC Davis & Stanford • Ongoing tech transfer and process development for commercial scale • Leverages existing relationships to develop clinical POC
Internal Manufacturing	Establish company's own manufacturing facility • Manufacturing is a strategic capability – 5 clinical programs in 2H2017 • Secures supply chain control and business continuity - internal QA/QC • Increases supply capacity for key therapies and potentially other products

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Plasma Therapy

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Abeona Proprietary ™ SDF Process for AATD

Inherited autosomal disorder that results in insufficient AAT protein production by liver

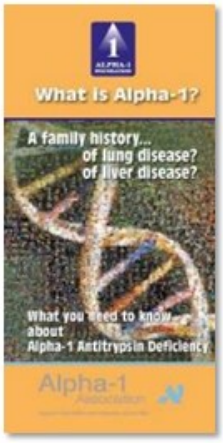
- AATD may lead to liver disease
- Alpha-1 protects lungs from inflammation and damage caused by infection and inhaled irritants

Prevalence – need to diagnose and treat

- AATD has been identified in nearly all populations and ethnic groups
- Roughly 1 in every 2,500 Americans have AATD
- 5 times more prevalent than Cystic Fibrosis
- Up to 3% of all people diagnosed with COPD may have undetected AATD

The global market for plasma proteins is roughly $20 billion

- Currently blood plasma proteins are purified using the Cohn process developed in the 1940's



Abeona's Proprietary ™ SDF Process has demonstrated substantial improvements in yields and margins

Financial Overview

Cash (Q3 2016): $31.2 million*

Cash used in operations (Q1-Q3 2016): $9.6 million

No debt

Primary Common Shares outstanding: 33.5 million**

** Cash, cash equivalents, and short-term investments, does not include cash from public offering of $42M, closed November 1, 2016.*
***End of third quarter 2016, does not include shares from public offering, closed November 1, 2016.*

Investment Highlights

A World Leader in Rare Disease Therapies

Strategic Focus on Rare Diseases	• Substantial unmet medical need to treat life threatening diseases caused by a defect in a single gene • Most advanced programs in neurological / lysosomal storage diseases where there are no treatment options • Orphan and Rare pediatric disease focus: shorter route to approval, and lower costs • Opportunity to secure Priority Review Voucher (PRV) for rare pediatric diseases
Three Clinical Programs in Phase I/II	• ABO-102: Sanfilippo syndrome Type A – MPS IIIA. Lysosomal storage disease affecting children. • ABO-101: Sanfilippo syndrome Type B – MPS IIIB. Lysosomal storage disease affecting children. • EB-101: Recessive dystrophic epidermolysis bullosa (RDEB). Severely fragile skin disease affecting children.
Robust Pipeline With Multiple Therapies Soon Entering the Clinic	• Based on strong pre-clinical results, two additional programs are expected to begin Phase I/II by the end of 2017 including: – ABO-201: Juvenile Batten disease (CLN3) – ABO-202: Infantile Batten disease (CLN1)
Proprietary Vector Platform For New Product Development	• 60+ proprietary AAV vectors that uniquely enable the delivery of genes across the blood brain barrier making IV administration possible • Next generation chimera vector platform in development with increased gene delivery efficiency, reduced antibody affinity, enhanced ability to create immune responses and target tissue specifications
Several Catalysts will Drive Significant Near Term Value	• Achieve fast track designation for additional programs • Initiate high dose cohort in ABO-102 for MPS IIIA • Initiate and enroll 1st patient in ABO-101 for MPS IIIB Phase 1/2 clinical studies • Dose adolescents in EB-101 trial; secure orphan drug and rare pediatric disease designations

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Management & Boards

Management

- Tim Miller, Ph.D. - President and Chief Executive Officer & Director
- Jeffrey B. Davis - Chief Operating Officer & Director
- Harrison G. Wehner - Chief Financial Officer
- David P. Nowotnik, Ph.D. - Senior Vice President, Research & Development
- Kaye Spratt, Ph.D. - Vice President, Regulatory

Board of Directors

- Steven H. Rouhandeh - Executive Chairman
- Mark J. Alvino - Director
- Stephen B. Howell, MD - Director
- Todd Wider, MD - Director

Scientific Advisory Boards: Rare Disease and Plasma focused

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EXHIBIT 99.2

Abeona Therapeutics Provides Update from ABO-102 Phase 1/2 MPS IIIA Clinical Trial at the 13th Annual WORLD*Symposium*™ 2017

New York, NY, and Cleveland, OH – **February 17, 2017 -** Abeona Therapeutics Inc. (Nasdaq: ABEO):

- *ABO-102 gene therapy well-tolerated in 4 subjects (N=3 low dose, N=1 high dose) through 650 days follow up with no Serious Adverse Events*
- *63% +/- 0.5% central nervous system reduction of heparan sulfate GAG 6 months post-injection (N=2)*
- *Continued evidence of biopotency including reduced liver and spleen volumes and decreased urinary GAGs*
- *Two subjects assessed at the 6-month timepoint showed evidence for stabilization or improvement (average 60% over 2 subjects) in several Mullen subdomains*
- *Adaptive behavior ratings on the Vineland stabilized*
- *Subjects showed improved ability to complete individual items on the Leiter-R non-verbal IQ assessment resulting in improved raw scores*

Abeona Therapeutics Inc. (NASDAQ: ABEO), a leading clinical-stage biopharmaceutical company focused on developing therapies for life-threatening rare genetic diseases, announced updated data from the ongoing gene therapy clinical trial for Sanfilippo syndrome Type A (MPS IIIA), at the 13th Annual WORLD*Symposium*™ 2017 lysosomal storage disorders conference in San Diego, CA. The ongoing Phase 1/2 trial for ABO-102 (AAV-SGSH) is a first-in-man clinical trial utilizing a single intravenous injection of AAV gene therapy for subjects with MPS IIIA, a rare autosomal recessive disease affecting every cell and organ in the body, which results in neurocognitive decline, speech loss, loss of mobility, and premature death in children.

“We remain encouraged by continued signs of tolerability and biopotency in the low-dose cohort, and enrollment of the high-dose cohort is underway” stated Kevin M. Flanigan, M.D., principal investigator with the Center for Gene Therapy at Nationwide Children’s Hospital and Professor of Pediatrics and Neurology at The Ohio State University College of Medicine. “Additionally, we are pleased to see further decreases in CSF GAG measurements, as well as preliminary evidence for stabilization or improvement of some cognitive functions, at six months post-dosing.”

Per the design of the clinical trial, subjects received a single, intravenous injection of ABO-102 to deliver the AAV viral vector systematically throughout the body to introduce a corrective copy of the gene that underlies the cause of the MPS IIIA disease. Subjects are evaluated at multiple time points post-injection for safety assessments and initial signals of biopotency and clinical activity, which suggest that ABO-102 successfully reached target tissues throughout the body, including the central nervous system. Observations reported at the WORLD*Symposium*™ conference included:

- *Safety*: ABO-102 is well-tolerated in subjects injected with the low dose of 5E13 vg/kg ABO-102, with no treatment related adverse events or serious adverse events (SAEs) through over 650 days cumulative post-injection. Enrollment in the high dose cohort has commenced with no Serious Adverse Events (SAEs) reported to date.

- *Biopotency*: As reflected in published natural history studies evaluating MPS III subjects, cerebral spinal fluid (CSF) and urine GAG (heparan sulfate or "HS") are significantly elevated in the subject population as a symptom of disease pathology. As announced previously, all subjects in the low-dose cohort experienced reductions from baseline in CSF HS of 25.6% +/- 0.8%, suggesting ABO-102 crossed the blood brain barrier after intravenous administration. At the six-month follow-up (n=2), CSF HS continued to decrease to 63.1% +/- 0.5% of baseline values, suggesting further improvement in the elimination of the storage pathology. Data presented showed reduction in urinary heparan sulfate and urinary total GAG fragments.

- *Hepatosplenomegaly*: The natural history study in 25 subjects with MPS III (*Truxal et. al., 2016, Mol. Genet. Metab*.) demonstrated that subjects had increased liver and spleen volumes averaging 116% and 88%, respectively at baseline that did not change over a year of follow-up. All three subjects demonstrated significant reductions in liver volumes at 30-days post injection (17.1% +/- 1.9%). At the six-month follow-up in low dose subjects (n=2), this effect was sustained, with a liver volume further decreased by 29.7 – 30.3% and spleen volume by 2.2 – 12.9% from baseline.

- *Cognitive Assessments*: The clinical trial utilizes three validated neurocognitive and behavioral assessment tools, including the Leiter International Performance Scale Third Edition (Leiter-3), the Vineland Adaptive Behavior Scale, Second Edition (Vineland-II) and the Mullen Scale of Early Learning. Cognitive assessments are taken at baseline, and have been taken at the six-month (n=2) and will be taken at the twelve-month follow-up visits. These assessments provide the opportunity to measure several sub-domains, such as fine motor, visual acuity, expressive language, receptive language, among others. Assessments at six-month for the first two lose-dose patients provided early evidence of cognitive stabilization. The two subjects assessed at the 6-month timepoint showed evidence for stabilization or improvement of scores (average of 60% across 2 subjects) in several Mullen subdomains. Adaptive behavior ratings on the Vineland also stabilized. Both subjects showed improved ability to complete individual items on the Leiter-R non-verbal IQ assessment resulting in improved raw scores.

"The data demonstrate an early and robust systemic delivery of ABO-102, and the increased reductions in CNS HS GAG support our approach for intravenous ABO-102 delivery for subjects with Sanfilippo syndromes," stated Timothy J. Miller, Ph.D., President and CEO of Abeona Therapeutics. "We are excited about continued biomarker signals in this trial, as well as early positive signals in the neurocognitive assessments. While we are still very early in the trial, we are extremely encouraged by these early results and look forward to expanding enrollment in this clinical trial with enrollments accelerating at two additional international clinical sites."

Abeona's MPS IIIA program, ABO-102, has been granted Orphan Product Designation in the USA and in the European Union, has received the Rare Pediatric Disease Designation in the US, and recently received Fast Track designation by the US FDA.

Sanfilippo syndromes (or mucopolysaccharidosis (MPS) type III): a group of four inherited genetic diseases each caused by a single gene defect, described as type A, B, C or D, which cause enzyme deficiencies that result in the abnormal accumulation of glycosaminoglycans (GAGs, or sugars) in body tissues. MPS III is a lysosomal storage disease, a group of rare inborn errors of metabolism resulting from deficiency in normal lysosomal function. The incidence of MPS III (all four types combined) is estimated to be 1 in 70,000 births. Mucopolysaccharides are long chains of sugar molecule used in the building of connective tissues in the body. There is a continuous process in the body of replacing used materials and breaking them down for disposal. Children with MPS III are missing an enzyme which is essential in breaking down the used mucopolysaccharides called heparan sulfate. The partially broken down mucopolysaccharides remain stored in cells in the body causing progressive damage. In MPS III, the predominant symptoms occur due to accumulation within the central nervous system (CNS), including the brain and spinal cord, resulting in cognitive decline, motor dysfunction, and eventual death. Importantly, there is no cure for MPS III and treatments are largely supportive care.

About Abeona: Abeona Therapeutics Inc. is a clinical-stage biopharmaceutical company developing gene therapies for life-threatening rare genetic diseases. Abeona's lead programs include ABO-102 (AAV-SGSH) and ABO-101 (AAV-NAGLU), adeno-associated virus (AAV) based gene therapies for Sanfilippo syndrome (MPS IIIA and IIIB, respectively). Abeona is also developing EB-101 (gene-corrected skin grafts) for recessive dystrophic epidermolysis bullosa (RDEB), EB-201 for epidermolysis bullosa (EB), ABO-201 (AAV-CLN3) gene therapy for juvenile Batten disease (JNCL), ABO-202 (AAV-CLN1) gene therapy for treatment of infantile Batten disease (INCL), and ABO-301 (AAV-FANCC) for Fanconi anemia (FA) disorder and ABO-302 using a novel CRISPR/Cas9-based gene editing approach to gene therapy for rare blood diseases. In addition, Abeona has a plasma-based protein therapy pipeline, including SDF Alpha™ (alpha-1 protease inhibitor) for inherited COPD, using its proprietary SDF™ (Salt Diafiltration) ethanol-free process. For more information, visit www.abeonatherapeutics.com.

This press release contains certain statements that are forward-looking within the meaning of Section 27a of the Securities Act of 1933, as amended, and that involve risks and uncertainties. These statements are subject to numerous risks and uncertainties, including but not limited to continued interest in our rare disease portfolio, our ability to enroll patients in clinical trials, the impact of competition; the ability to develop our products and technologies; the ability to achieve or obtain necessary regulatory approvals; the impact of changes in the financial markets and global economic conditions; our belief that initial signals of biopotency and clinical activity, which suggest that ABO-102 successfully reached target tissues throughout the body, including the central nervous system; our belief that the data demonstrate an early and robust systemic delivery of ABO-102, and the increased reductions in CNS GAG support our approach for intravenous delivery for subjects with Sanfilippo syndromes, and other risks as may be detailed from time to time in the Company's Annual Reports on Form 10-K and other reports filed by the Company with the Securities and Exchange Commission. The Company undertakes no obligations to make any revisions to the forward-looking statements contained in this release or to update them to reflect events or circumstances occurring after the date of this release, whether as a result of new information, future developments or otherwise.

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