UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 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): September 12, 2016

REGENXBIO INC.
(Exact Name of Registrant as Specified in its Charter)

Delaware	**001-37553**	**47-1851754**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

9712 Medical Center Drive, Suite 100
Rockville, Maryland **20850**
(Address of principal executive offices) **(Zip Code)**

(240) 552-8181
(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.

REGENXBIO Inc. (the "Company") is furnishing a corporate presentation which it may use from time to time in conversations with investors and analysts beginning September 12, 2016. A copy of the presentation will be available on the Company's website. A copy of the presentation is attached hereto as Exhibit 99.1 and is incorporated by reference herein.

The Company expressly disclaims any obligation to update the corporate presentation or any other information available on or through its website, and cautions that the information set forth therein is only accurate as of the date indicated on such materials. The inclusion of any data or statements in the corporate presentation (or available on or through the Company's website) does not signify that such information is considered material.

The Company faces many challenges and risks in the industry in which the Company operates. The corporate presentation contains forward-looking statements that involve risks and uncertainties many of which are beyond the Company's control. The Company's actual results may differ materially from those discussed in any forward-looking statement because of various factors, including those described under "Forward-Looking Statements" in the corporate presentation. Please see "Forward-Looking Statements" in the corporate presentation for additional information about the known material risks that the Company faces.

The information in Item 7.01 of this Current Report on Form 8-K, including the exhibit attached hereto, is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	REGENXBIO Inc. Corporate Presentation.

SIGNATURES

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.

REGENXBIO INC.

Date: September 12, 2016

By: /s/ Kenneth T. Mills
Kenneth T. Mills
President and Chief Executive Officer

EXHIBIT INDEX

Exhibit No.	Description
99.1	REGENXBIO Inc. Corporate Presentation.

Exhibit 99.1

Corporate Presentation

September 12, 2016

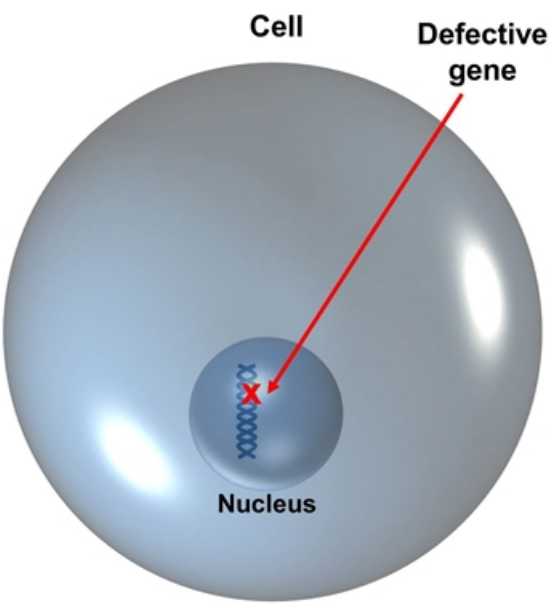
Forward-looking statements

This presentation contains "forward-looking statements," within the meaning of the Private Securities Litigation Reform Act of 1995, regarding, among other things, the timing and success of preclinical studies and clinical trials conducted by REGENXBIO and its development partners, estimates regarding the prevalence of diseases REGENXBIO's product candidates may be approved to treat in the future and REGENXBIO's expected cash burn. Such forward-looking statements are based on current expectations and involve inherent risks and uncertainties, including factors that could cause actual results to differ materially from those projected by such forward-looking statements. All of REGENXBIO's development timelines could be subject to adjustment depending on recruitment rate, regulatory agency review, and other factors that could delay the initiation and completion of clinical trials. Meaningful factors which could cause actual results to differ include, but are not limited to, the timing of enrollment, commencement and completion of REGENXBIO's clinical trials; the timing and success of preclinical studies and clinical trials conducted by REGENXBIO, its development partners and its NAV Technology Licensees; the ability to obtain and maintain regulatory approval to conduct clinical trials and to commercialize REGENXBIO's product candidates, and the labeling for any approved products; the scope, progress, expansion, and costs of developing and commercializing REGENXBIO's product candidates; REGENXBIO's ability to obtain and maintain intellectual property protection for our product candidates and technology; trends and challenges in REGENXBIO's business and the markets in which REGENXBIO operates; REGENXBIO's ability to attract or retain key personnel; the size and growth of the potential markets for REGENXBIO's product candidates and the ability to serve those markets; the rate and degree of market acceptance of any of REGENXBIO's product candidates; REGENXBIO's ability to establish and maintain development partnerships, including those with NAV Technology Licensees; REGENXBIO's expenses and revenue, the sufficiency of REGENXBIO's cash resources and needs for additional financing, regulatory developments in the United States and foreign countries, as well as other factors discussed in the "Risk Factors" and "Management's Discussion and Analysis of Financial Condition and Results of Operations" sections of REGENXBIO's Annual Report or Form 10-K for the year ended December 31, 2015 and Quarterly Report on Form 10-Q for the quarter ended June 30, 2016, which are on file with the SEC and available on the SEC's website at www.sec.gov. Additional factors may be set forth in those sections of REGENXBIO's Quarterly Report on Form 10-Q for the quarter ending September 30, 2016, to be filed in the fourth quarter of 2016. In addition to the risks described above and in REGENXBIO's filings with the SEC, other unknown or unpredictable factors also could affect REGENXBIO's results. There can be no assurance that the actual results or developments anticipated by REGENXBIO will be realized or, even if substantially realized, that they will have the expected consequences to, or effects on, REGENXBIO. Therefore, no assurance can be given that the outcomes stated in such forward-looking statements and estimates will be achieved.

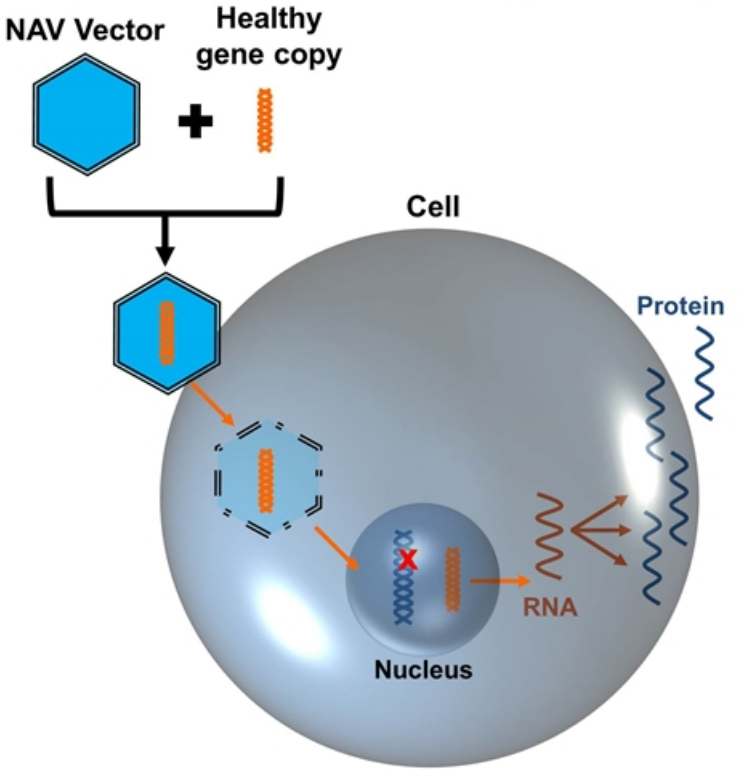
All forward-looking statements contained in this presentation are expressly qualified by the cautionary statements contained or referred to herein. REGENXBIO cautions investors not to rely too heavily on the forward-looking statements REGENXBIO makes or that are made on its behalf. These forward-looking statements speak only as of September 12, 2016 (unless another date is indicated). REGENXBIO undertakes no obligation, and specifically declines any obligation, to publicly update or revise any such forward-looking statements, whether as a result of new information, future events or otherwise.

NAV Gene Therapy – using a genetically modified virus as a delivery vehicle for healthy genes

Problem: Patient cannot make a protein the body needs because of a defective gene



NAV Gene Therapy delivers a healthy gene copy to cells to address the underlying defect



REGENXBIO: adeno-associated virus (AAV) gene therapy company developing life-altering treatments for patients with severe diseases

Proprietary NAV® Technology Platform includes exclusive worldwide rights to over 100 AAV vectors, including AAV7, AAV8, AAV9 and AAVrh10

NAV Technology Platform proof-of-concept and durability supported by data from multiple clinical trials

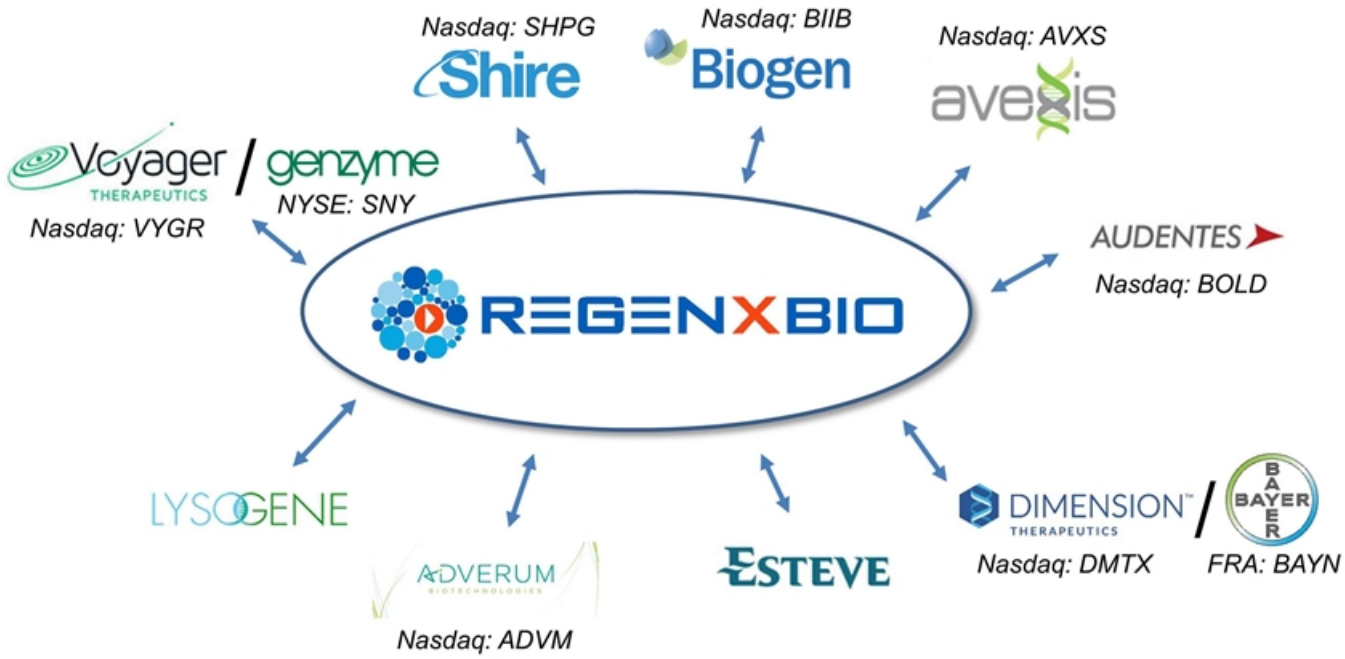
Lead program clinical trial initiation anticipated in 2H 2016 and expect to have active INDs for our four lead programs in 2017

24 partnered product candidates being developed by third-party licensees, with four in clinical trials[1]

Management team and scientific advisors are leaders in gene therapy

[1] As of June 30, 2016

REGENXBIO's NAV Technology Platform has been widely adopted



REGENXBIO's lead programs

Internally developed product candidates

Indication	Development Stage: Research	Development Stage: Preclinical	Development Stage: Clinical	Regulatory / Clinical Status
Metabolic Diseases				
▲ Homozygous familial hypercholesterolemia (HoFH)		RGX–501		IND active, Phase I/II trial initiation anticipated 2H 2016
Neurodegenerative Diseases				
▲ ★ Mucopolysaccharidosis Type I (MPS I)		RGX–111		IND submission anticipated 1H 2017
▲ ★ Mucopolysaccharidosis Type II (MPS II)		RGX–121		IND submission anticipated 1H 2017
Retinal Diseases				
Wet age-related macular degeneration (wet AMD)		RGX–314		IND submission anticipated Q1 2017
X-linked retinitis pigmentosa (XLRP)		RGX–321		

▲ Orphan Drug Designation

★ Rare Pediatric Disease Designation

REGENXBIO®

Internal Development Programs

RGX–501 for treatment of homozygous familial hypercholesterolemia (HoFH)

THE DISEASE

- Defective LDLR gene leads to limited / no LDL cholesterol receptors, allowing LDL to build up in bloodstream
- Total cholesterol levels >500 mg/dL
- Coronary artery disease at young age
- Even with existing standard of care therapies, the mean age of death is 32 years

PATIENTS

Approx. 11,000 globally

RGX–501 PRODUCT CANDIDATE

Vector

AAV8

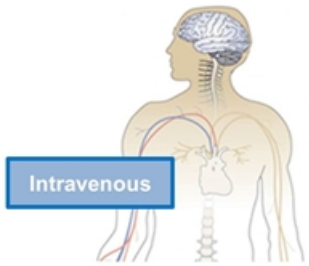
Gene

LDLR

Mechanism of action

Correction of defective LDLR, reducing circulating LDL cholesterol

Route of administration

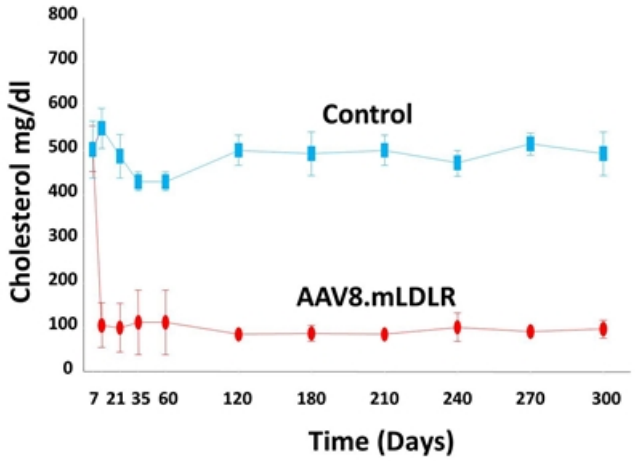


Special Regulatory Status

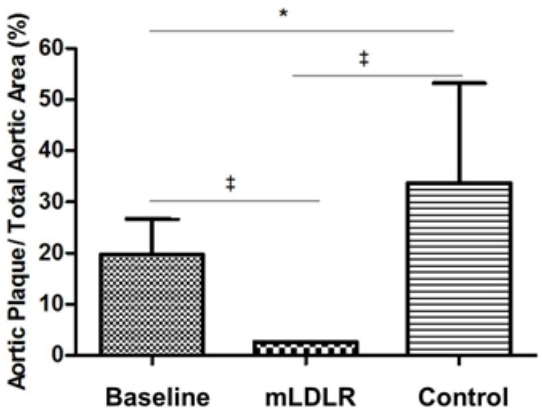
Orphan Drug Designation

Preclinical studies using NAV Gene Therapy have shown a significant improvement in familial hypercholesterolemia mice

- Familial hypercholesterolemia mice injected with AAV8
  - Mouse LDLR gene or control



Stable correction of cholesterol levels seen for nearly one year



Correction of LDLR defect prevents and induces regression of atherosclerotic lesions

Source: Kassim et al. 2010 *PLOS One*

RGX–501 Phase I/II clinical trial in HoFH

Objectives

- **Primary**
  - To determine the safety of RGX-501 administration in patients with HoFH
- **Secondary**
  - Percent change in LDL-C levels at 12 weeks
  - Other lipid outcome measures

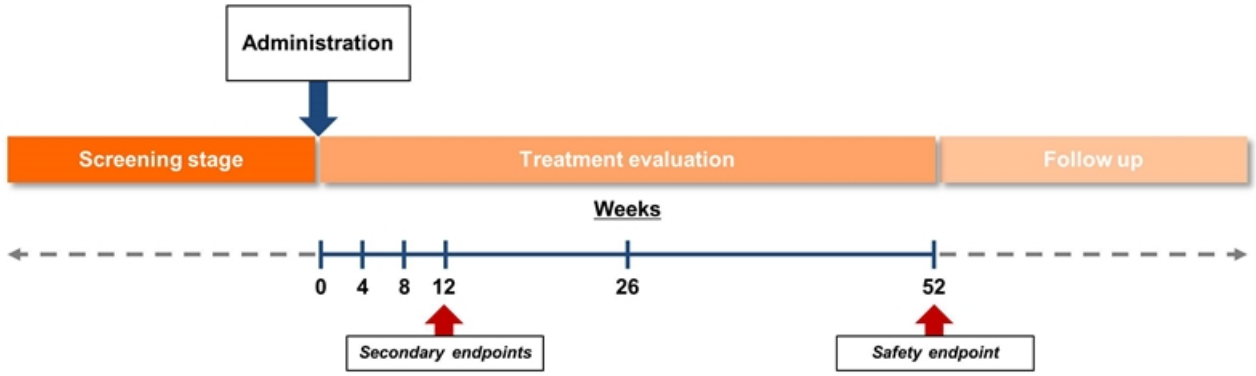
Key inclusion criteria

- Male or female >= 18 years of age
- Untreated and/or treated LDL-C levels and clinical presentation consistent with the diagnosis of HoFH
- Molecularly defined LDLR mutations at both LDLR alleles
- NAbs titer <=1:5
- Stable concurrent allowed lipid lowering medications
  - Statins, ezetimibe, bile acid sequestrants, **PCSK9i**

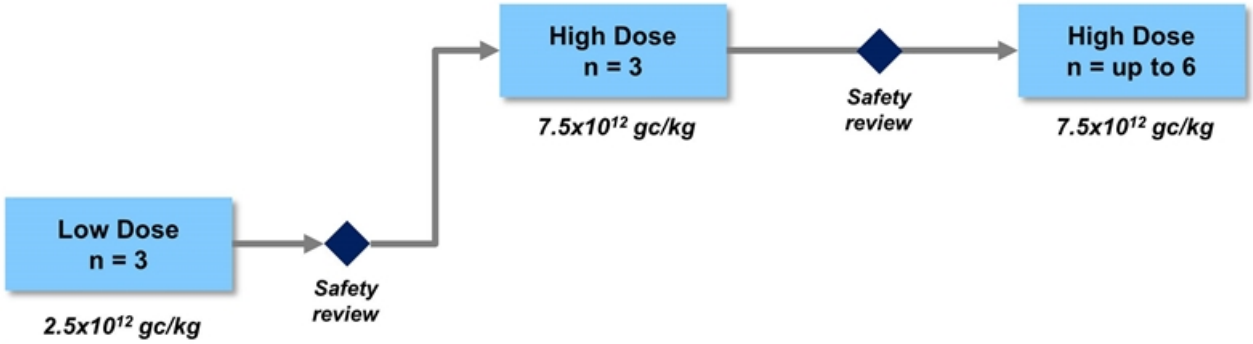
Total subjects: up to 12

RGX–501 Phase I/II clinical trial – administration and dose escalation

Administration and follow-up timeline



Expected dose escalation pathway



RGX–314 for treatment of wet age-related macular degeneration (wet AMD)

THE DISEASE

- **Blurring of central vision and progressive vision loss due to formation of leaky blood vessels in the eye**
- **VEGF inhibitors can prevent / reverse new blood vessel formation**
- **Frequency / uncomfortable administration of current anti-VEGF therapies affects compliance and ultimately efficacy**

PATIENTS

More than 3 million estimated globally

RGX–314 PRODUCT CANDIDATE

Vector

AAV8

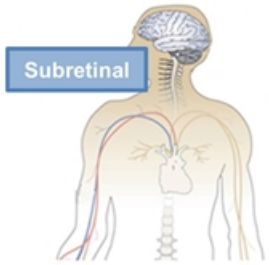
Gene

Anti-VEGF

Mechanism of action

Reducing leaky blood vessel formation by giving ocular cells the ability to produce anti-VEGF

Route of administration



RGX–314 – potential for long-term therapeutic anti-VEGF expression

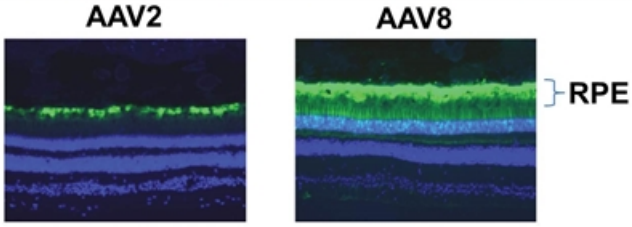
Improved AAV vector technology

+

Leveraging current standard of care in transgene

=

RGX-314: AAV8 encoding anti-VEGF fab



More efficient gene delivery to the RPE[1]

- Current standard of care includes FDA-approved mAbs and mAb fragments that inhibit VEGF
- **RGX-314 gene encodes an anti-VEGF mAb fragment (fab)**

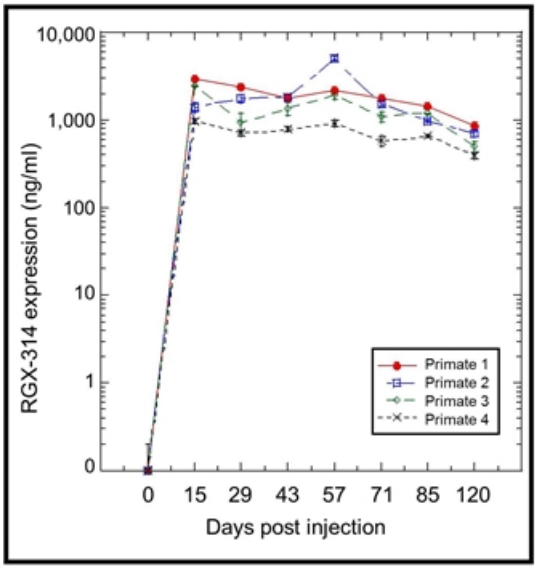
Potentially eliminate frequent, uncomfortable administration of current anti-VEGF therapies that affect compliance, and ultimately efficacy

[1] Vandenberghe et al. 2011 *Science Translational Medicine*

RGX–314 has significant potential advantages over earlier generation candidates for wet AMD gene therapy

Sponsor	Vector	ROA	Transgene	Dose (GC/eye)	Max. expression (ng/ml)[2]
REGENXBIO	AAV8	Subretinal	anti-VEGF fab	1.0e11	4,992
AVALANCHE BIOTECH[3]	AAV2	Subretinal	sFlt	8.0e11	0.217
genzyme[4]	AAV2	Intravitreal	sFLT01	2.4e10	528

RGX-314 expression in non-human primates[1]



[1] Wielechowski et al. 2016 Poster session presented at the meeting of the American Society of Gene & Cell Therapy, Washington, DC
[2] Maximum expression in the anterior chamber of non-human primate eyes
[3] Lai et al. 2012 *Gene Therapy*
[4] MacLachlan et al. 2011 *Molecular Therapy*

RGX–111 for treatment of mucopolysaccharidosis Type I (MPS I)

THE DISEASE

- Defective IDUA gene leads to reduced ability to process glycosaminoglycans (GAGs), a cellular waste product
- Severe GAG buildup causes neurodegeneration and early death
- Available treatment is inadequate to treat neurodegeneration; bone marrow transplant partially effective

PATIENTS

Approx. 500 – 1,000 born annually

RGX–111 PRODUCT CANDIDATE

Vector

AAV9

Gene

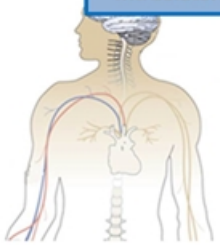
IDUA

Mechanism of action

Replacement of IDUA, reducing GAG accumulation

Route of administration

Intracisternal

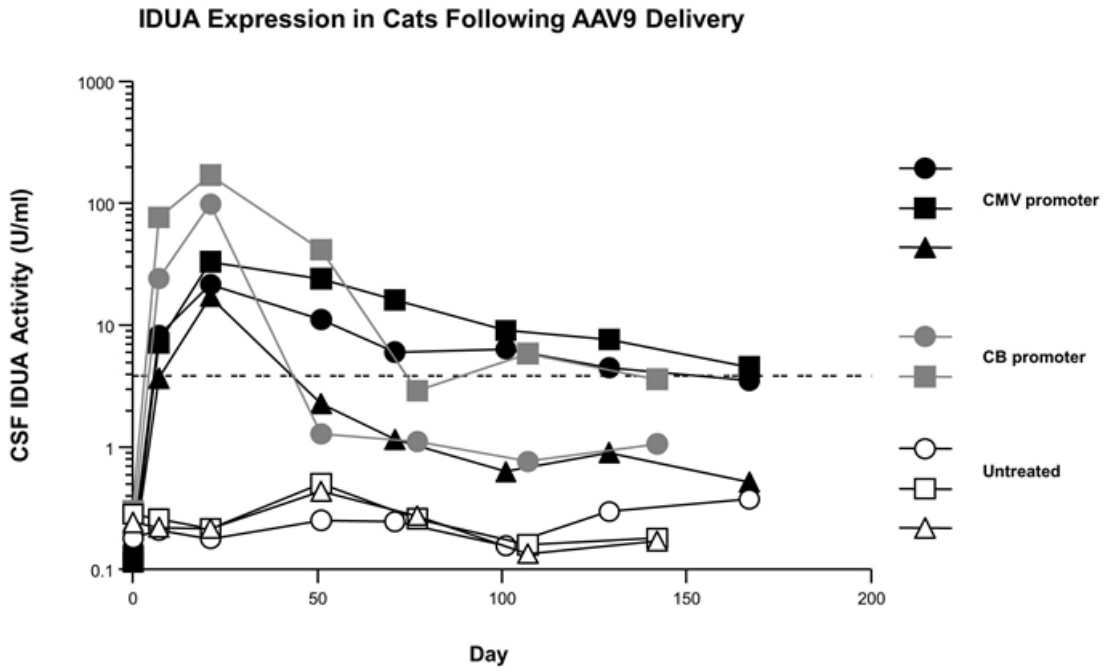


Special Regulatory Status

Orphan Drug Designation

Rare Pediatric Disease Designation

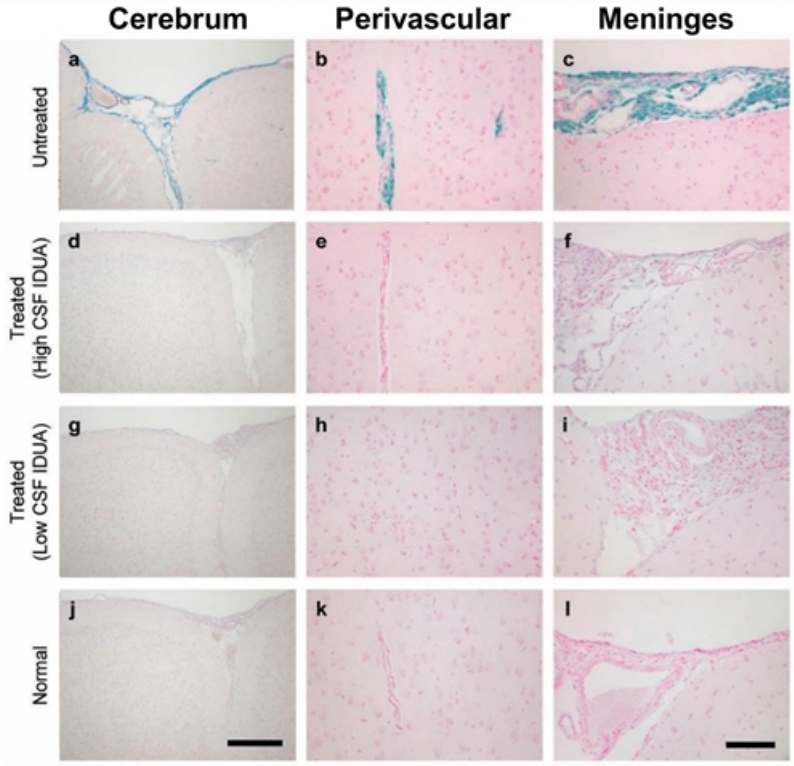
Preclinical studies using NAV Gene Therapy have shown a significant improvement in MPS I cats



CNS correction observed was substantially greater than that observed in a previous study of MPS I cats treated with BMT at similar ages

Source: Bell et al. 2014 *Molecular Therapy*

In preclinical studies, MPS I cats benefitted from cross-correction



Correction observed throughout the CNS

Source: Bell et al. 2014 *Molecular Therapy*

RGX–121 for treatment of mucopolysaccharidosis Type II (MPS II)

THE DISEASE

- Defective IDS gene leads to reduced ability to process GAGs
- Severe GAG buildup causes neurodegeneration and early death (similar phenotype to severe MPS I)
- X-linked recessive disease (predominantly occurs in males)
- Available treatment is inadequate to treat neurodegeneration

PATIENTS

Approx. 500 – 1,000 born annually

RGX–121 PRODUCT CANDIDATE

Vector

AAV9

Gene

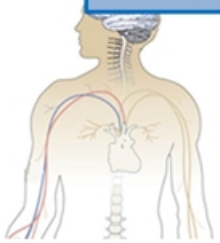
IDS

Mechanism of action

Replacement of IDS, reducing GAG accumulation

Route of administration

Intracisternal



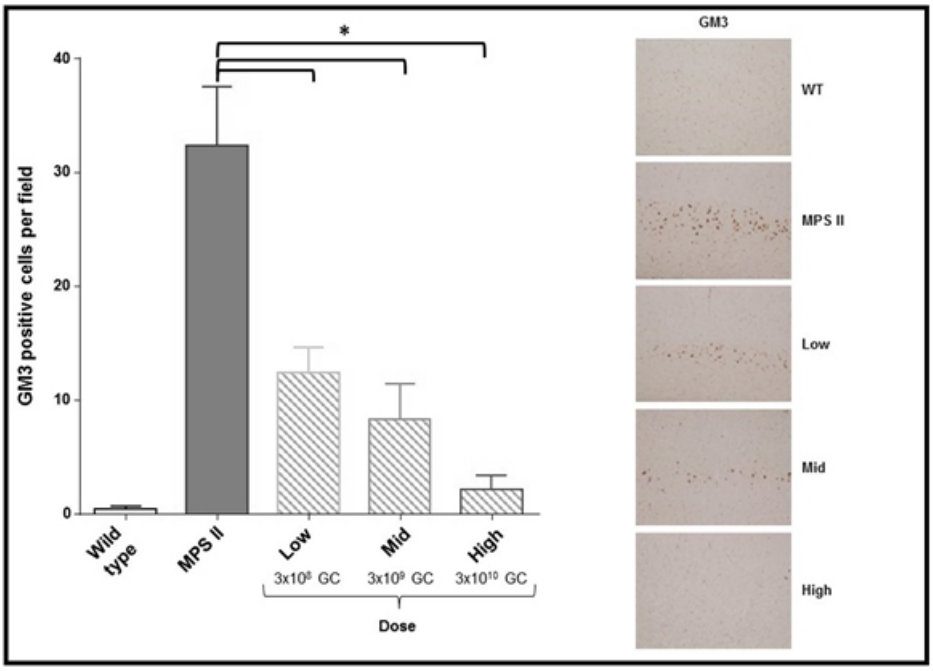
Special Regulatory Status

Orphan Drug Designation

Rare Pediatric Disease Designation

Preclinical studies using NAV Gene Therapy have shown a significant improvement in MPS II mice

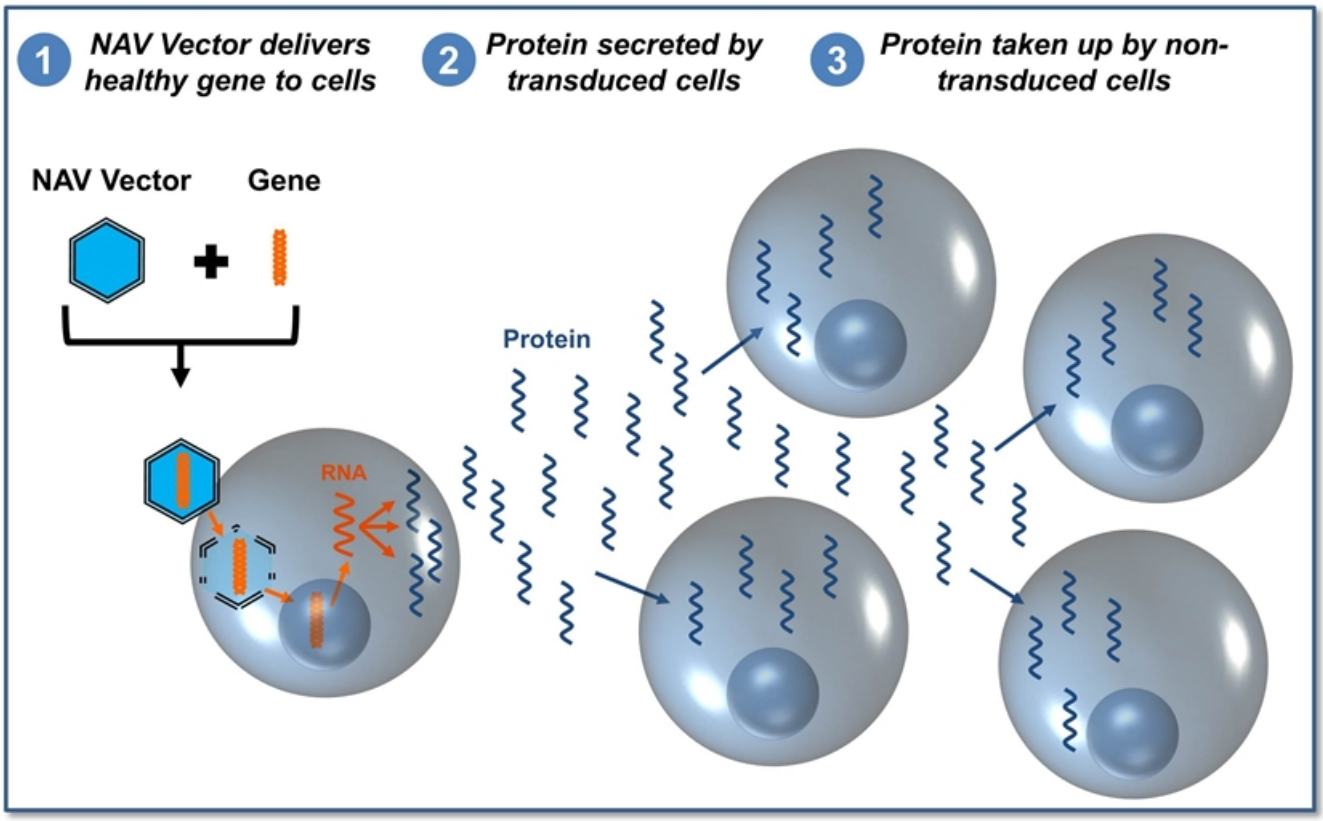
Reduction in Storage Lesions Following AAV9 Delivery



Statistically significant reduction in storage lesions at all dose levels

Source: Hinderer et al. 2016 *Human Gene Therapy*

Cross–correction is a key treatment advantage in MPS diseases

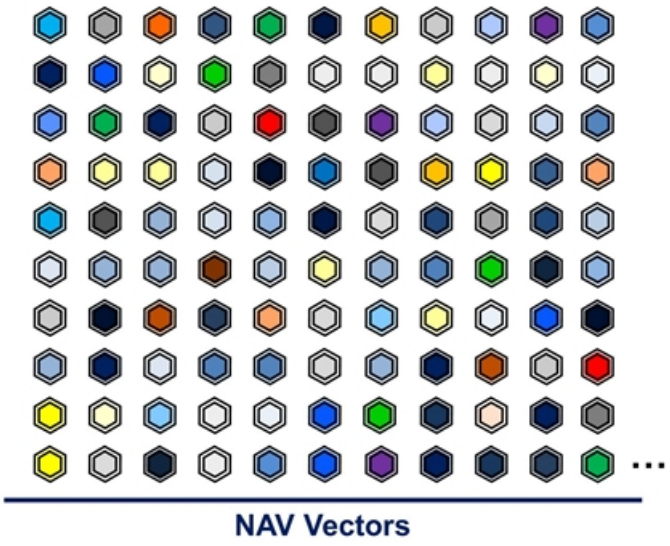


A single transduced cell has potential to correct many other cells

NAV® Technology Platform

The NAV Technology Platform is based on a broad and deep IP portfolio

Exclusive rights to more than 100 patents and patent applications worldwide



- ✓ AAV7, AAV8, AAV9, AAVrh10
- ✓ Over 100 other novel AAV sequences
- ✓ Sequences that are at least 95% identical to these capsids

Key features of REGENXBIO's NAV Technology Platform

Key features

- Higher gene expression
- Longer-term gene expression
- Broad and novel tissue selectivity
- Lower immune response
- Improved manufacturability

The NEW ENGLAND JOURNAL of MEDICINE

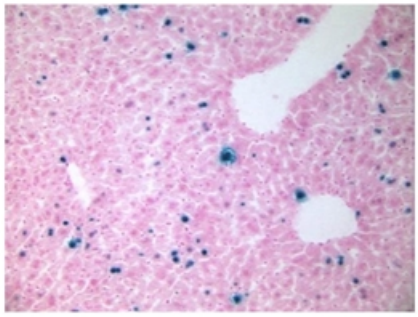
Long-Term Safety and Efficacy of Factor IX Gene Therapy in Hemophilia B

Intravascular AAV9 Preferentially Targets Neonatal Neurons and Adult Astrocytes

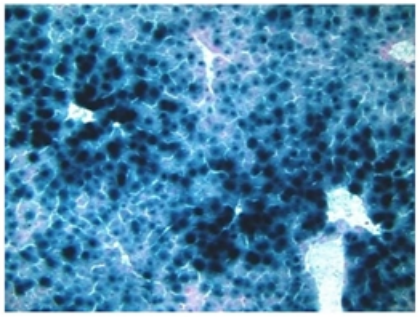
NAV Vectors – higher gene expression than early generation AAV vectors

NAV Vector AAV8: 10x–100x greater gene expression

AAV2

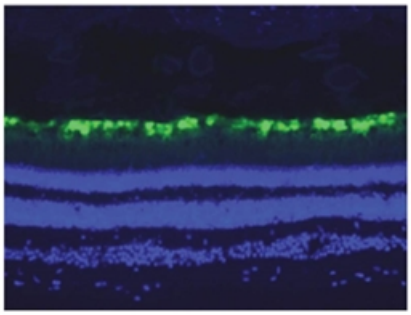


AAV8

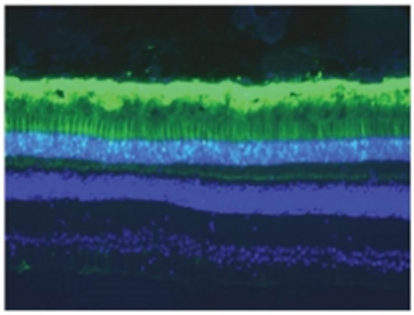


NAV Vector AAV8: More efficient gene delivery to sites of most retinal dystrophies[1]

AAV2



AAV8



[1] Vandenberghe et al. 2011 *Science Translational Medicine*

Team and Conclusion

The REGENXBIO team

Name	Position	Prior / Current Affiliations
Ken Mills	President, CEO & Co-Founder; Director	FOXKISER, MSD
James Wilson, M.D., Ph.D.*	Chief Scientific Advisor & Co-Founder	Perelman School of Medicine, University of Pennsylvania; Massachusetts General Hospital
Vit Vasista	Chief Financial Officer	PRTM, MSD
Stephen Yoo, M.D.	Chief Medical Officer	MedImmune, Abbott
Faraz Ali	Chief Business Officer	bluebirdbio, genzyme
Curran Simpson	SVP, Technical Operations	gsk, Human Genome Sciences
Laura Coruzzi, Ph.D., J.D.	SVP, Intellectual Property	Jones Day
Patrick Christmas, J.D.	SVP, General Counsel	Lumara Health, Wellstat Therapeutics
Kimberly Sloan	SVP, Human Resources	Biocon, dun & bradstreet

* Key advisor to REGENXBIO

Financial results and guidance

Financials as of 06/30/2016 (mm)

Cash:	**$199**
R&D expense:	**$17**
G&A expense:	**$12**
Net loss:	**$25**
Basic shares:	**26.5**

2015 financial highlights

- Over $100 million raised in two private financing rounds
- Closed initial public offering in September, raising over $159 million in gross proceeds
- Ended the year with over $216 million in cash

Program guidance

RGX-501	▪ Recruiting patients, Phase I/II trial initiation anticipated 2H 2016
RGX-314	▪ IND submission anticipated Q1 2017
RGX-111	▪ IND submission anticipated 1H 2017
RGX-121	▪ IND submission anticipated 1H 2017

2016 financial guidance

- As of August 9, 2016, expect 2016 cash burn to be between $60 million and $70 million

REGENXBIO: adeno-associated virus (AAV) gene therapy company developing life-altering treatments for patients with severe diseases

Proprietary NAV® Technology Platform includes exclusive worldwide rights to over 100 AAV vectors, including AAV7, AAV8, AAV9 and AAVrh10

NAV Technology Platform proof-of-concept and durability supported by data from multiple clinical trials

Lead program clinical trial initiation anticipated in 2H 2016 and expect to have active INDs for our four lead programs in 2017

24 partnered product candidates being developed by third-party licensees, with four in clinical trials[1]

Management team and scientific advisors are leaders in gene therapy

[1] As of June 30, 2016

REGENXBIO®

Leader in AAV gene therapy

Thank You