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): April 20, 2016

bluebird bio, Inc.
(Exact name of registrant as specified in its charter)

DELAWARE	001-35966	13-3680878
(State or other jurisdiction of incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

150 Second Street Cambridge, MA	02141
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code **(339) 499-9300**

Not Applicable
(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:

☐ 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

On April 20, 2016, bluebird bio, Inc. ("bluebird") conducted an investor webcast summarizing clinical data from its Starbeam clinical study of its Lenti-D product candidate, presented at the American Academy of Neurology (AAN) 2016 Annual Meeting in Vancouver, British Columbia, Canada on April 20, 2016. A copy of the presentation is being furnished as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Investor presentation provided by bluebird bio, Inc. on April 20, 2016.

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.

bluebird bio, Inc.

Date: April 20, 2016

By: /s/ Jason F. Cole
Jason Cole
Chief Legal Officer

EXHIBIT INDEX

Exhibit No.	Description
99.1	Investor presentation provided by bluebird bio, Inc. on April 20, 2016.

Exhibit 99.1

bluebirdbio®

Starbeam Study Interim Clinical Data

American Academy of Neurology Annual Meeting
April 20, 2016

Nasdaq : BLUE

Forward-Looking Statement

These slides and the accompanying oral presentation contain forward-looking statements and information. The use of words such as “may,” “might,” “will,” “should,” “expect,” “plan,” “anticipate,” “believe,” “estimate,” “project,” “intend,” “future,” “potential,” or “continue,” and other similar expressions are intended to identify forward-looking statements. For example, all statements we make regarding the initiation, timing, progress and results of our clinical studies of our Lenti-D product candidate and our research and development programs, our ability to advance product candidates into, and successfully complete, clinical studies, and the timing or likelihood of regulatory filings and approvals are forward-looking. All forward-looking statements are based on estimates and assumptions by our management that, although we believe to be reasonable, are inherently uncertain. All forward-looking statements are subject to risks and uncertainties that may cause actual results to differ materially from those that we expected. These statements are also subject to a number of material risks and uncertainties that are described in our most recent annual report on Form 10-K, as well as our subsequent filings with the Securities and Exchange Commission. Any forward-looking statement speaks only as of the date on which it was made. We undertake no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise, except as required by law.

Nasdaq: BLUE

bluebird bio: Why We Do What We Do

Our Vision – Make Hope a Reality

Ethan

Aidan

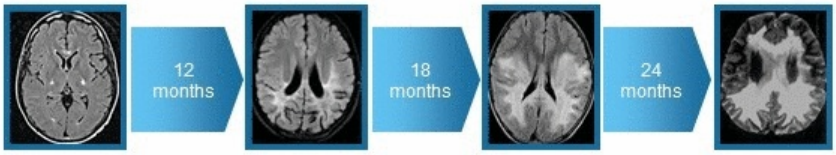
Cameron

Committed to transforming the lives of patients with severe genetic and rare diseases

Starbeam Study

Cerebral Adrenoleukodystrophy (CALD): Disease Overview

Natural Course of Disease



DISEASE

Ultra-orphan, X-linked, monogenic, neurological disorder

Mutated gene results in toxic buildup of very long chain fatty acids

Leads to cerebral inflammation & demyelination

CURRENT TREATMENTS

Untreated CALD leads to dismal outcomes (vegetative state and death)

Allogeneic stem cell transplant standard for CALD (if possible)

EPIDEMIOLOGY

CALD most severe form of ALD

ALD incidence: 1 in ~21,000 (male live births)

Cerebral disease

- CALD accounts for 30-40% of ALD
- AMN accounts for 40-45% of ALD with 40% cerebral
- ACALD accounts for ~25% of ALD

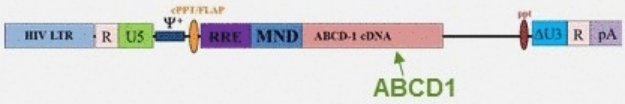
Autologous HSC Gene Therapy in CALD

Need for options beyond allogeneic HCT

- >10% treatment-related mortality[2,3,4]
- 45%–75% experience moderate to severe GvHD[2,3,4,5]
- Full HLA donor match is required for best outcomes[1,2,3]
  - 49% of ALD transplants performed with mismatched, unrelated donors
  - Time to find donor may delay treatment beyond effective window

Lenti-D vector

self-inactivating lentiviral vector delivering a functional copy of *ABCD1* to autologous HSCs



Science
AAAS

Hematopoietic Stem Cell Gene Therapy with a Lentiviral Vector in X-Linked Adrenoleukodystrophy

Nathalie Cartier,[1,2*] Salima Hacein-Bey-Abina,[3,4,5*] Cynthia C. Bartholomae,[6] Gabor Veres,[7] Manfred Schmidt,[6] Ina Kutschera,[6] Michel Vidaud,[1] Ulrich Abel,[6] Liliane Dal-Cortivo,[3,5] Laure Caccavelli,[3,5] Nizar Mahlaoui,[8] Véronique Kiermer,[9] Denice Mittelstaedt,[10] Céline Bellesme,[2] Najiba Lahlou,[11] François Lefrère,[3] Stéphane Blanche,[4] Muriel Audit,[12] Emmanuel Payen,[13,14] Philippe Leboulch,[13,14,15] Bruno l'Homme,[3] Pierre Bougnères,[2] Christof Von Kalle,[6] Alain Fischer,[4,8] Marina Cavazzana-Calvo,[3,4,5*] Patrick Aubourg[1,2*†]

Cartier et al., *Science* **326**, 818 (2009)

Proof of concept study reported 2009

- 2 subjects with stabilization of neurodegeneration 14 to 16 months after gene therapy

1. GdE+ HCT-treated (N=23) subjects with Loes score ≤9 & NFS ≤1, from time of diagnosis or time of HCT, all donor sources. bluebird bio study ALD-101, manuscript in preparation. 2. Miller et al, Blood 2011. 3. Peters et al, Blood 2004. 4.Beam et al, Biol Blood Marrow Transplant. 2007. 5: Baumann et al, Eur J Pediatr, 2003. 6: Martin et al, Biol Blood Marrow Transplant, 2006.

Measuring Neurologic Progression in CALD
Functional Outcomes

Neurological Function Score (NFS)[1]

- 15 domains; total possible score of 25
- **70% maintain stable NFS[2] @ 2 years post allo-HCT[3]**

Major Functional Disabilities (MFDs)

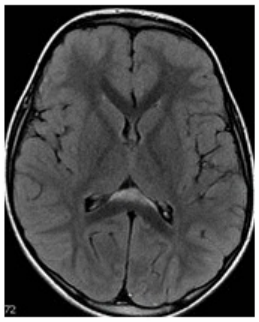
- 6 most serious symptoms
- **81% MFD-free survival @ 2 years post allo-HCT[3]**

NFS Component	Score	NFS Component	Score
Hearing/auditory processing problems	1	Walking difficulties/ spasticity	1
Aphasia/apraxia	1	Spastic gait (needs assistance)	2
Loss of communication	**3**	**Wheelchair dependence**	**2**
Vision impairment	1	**No voluntary movement**	**3**
Cortical blindness	**2**	Episodes of incontinence	1
Swallowing dysfunctions	2	**Total incontinence**	**2**
Tube feeding	**2**	Non-febrile seizures	1
Running difficulties	1	**Possible Total**	**25**

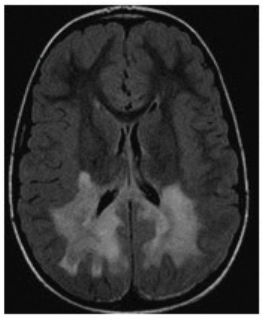
1. Moser et al. Neuropediatrics 2000;31(5):227-239. 2. "Stable NFS" = change post-allo-HCT of ≤ 3 NFS points and an absolute NFS ≤ 4 at Month 24 (±6 months). 3. GdE+ HCT-treated (N=23) subjects with Loes score ≤9 & NFS ≤1, from time of diagnosis or time of HCT, all donor sources. bluebird bio study ALD-101, manuscript in preparation.

Measuring Neurologic Progression in CALD
MRI Measures

Loes MRI severity score[1]**:** measurement of white matter changes by degree and extent of pathological hyperintense regions (0-34)

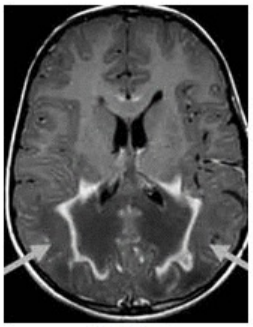


Loes score = 1



Loes score = 15

Gadolinium enhancement: indicator of active inflammation in untreated patients (+/-)



GdE+

Images Courtesy of Dr. Florian Eichler

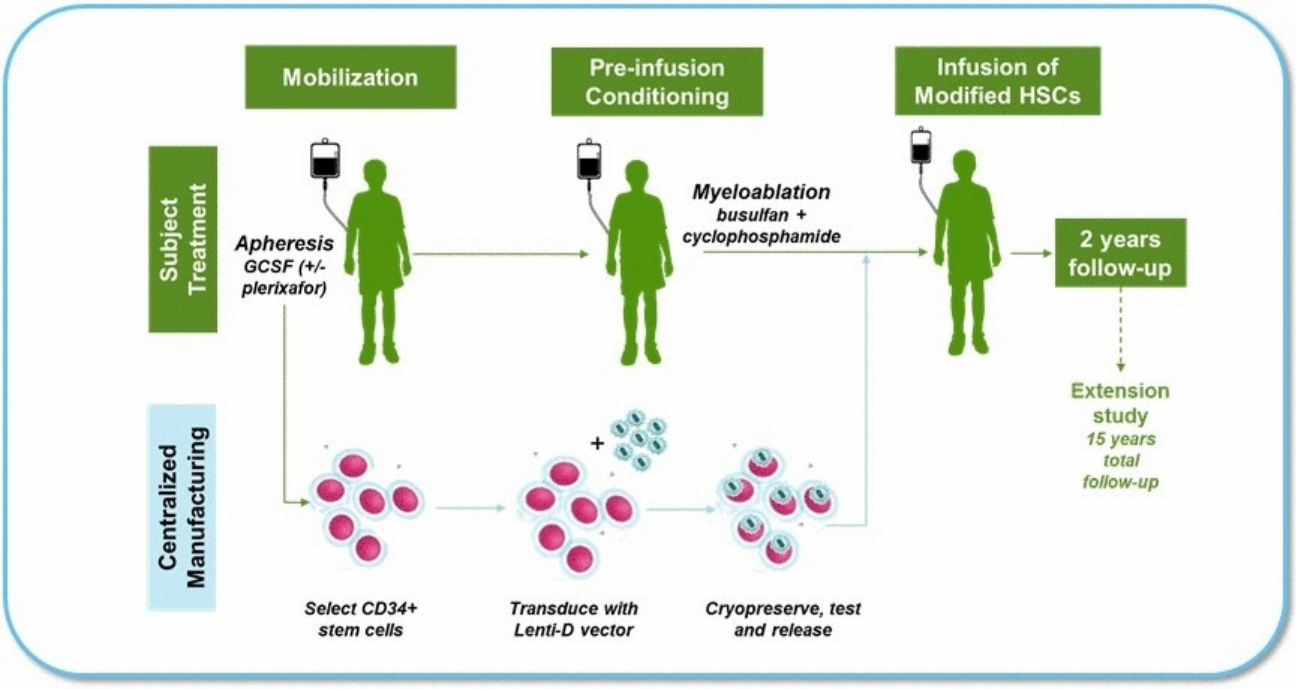
1. Loes et al. AJNR Am J Neuroradiol 1994;15(9):1761-1766.

Starbeam Study – Ongoing Phase 2/3 Study

Open label, multi-center, single arm, global study

Design	Primary Endpoint	Secondary Endpoints
• 15 patients (18 enrolled) • Age ≤ 17 • Gad Positive • Loes Score 0.5 – 9 • NFS ≤ 1 • No HLA-matched sibling donor	• % of Boys With Major Functional Disabilities at 24 Months After Transplant	• Neurological Functional Score (NFS) • Gad +/- • Loes Score • Safety

Starbeam Treatment Protocol Overview



Subject and Cellular Product Characteristics

N=18 consented (formally screened) and N=17 infused subjects

- 17 subjects treated between October 2013 and July 2015
- Median follow-up 16 months (6-24m) as of March 31, 2016
  - 3 completed 24 months
  - 10 with 12 to <24 months follow-up
  - 4 with ≥6 to <12 months follow-up

Baseline Characteristics	
Parameter	**Median (range)**
Age at consent (years)	6.0 (4-13)
Time from CALD diagnosis to treatment (months)	6.8 (2.5-65.8)
Time from enrollment to treatment (months)	2.2 (1.9-2.9)
Baseline Loes score	2.0 (1.0-7.5)
Baseline NFS score	0 (0-0)
MFDs present at baseline	None

Data as of March 31, 2016

Successful Autologous HSCT Procedure

Parameter	Median (range)
Drug Product Cell Dose (x10^6/kg)	10.5 (6.0-19.4)
Neutrophil engraftment[1] (with and without GCSF)	Day +13 (11-39)
Platelet engraftment[2]	Day +29 (16-55)

1. 3 consecutive ANC values of ≥ 0.5 x 10^9/L obtained on different days. 2. 3 consecutive platelet values ≥ 20 x 10^9 /L obtained on different days (unsupported)

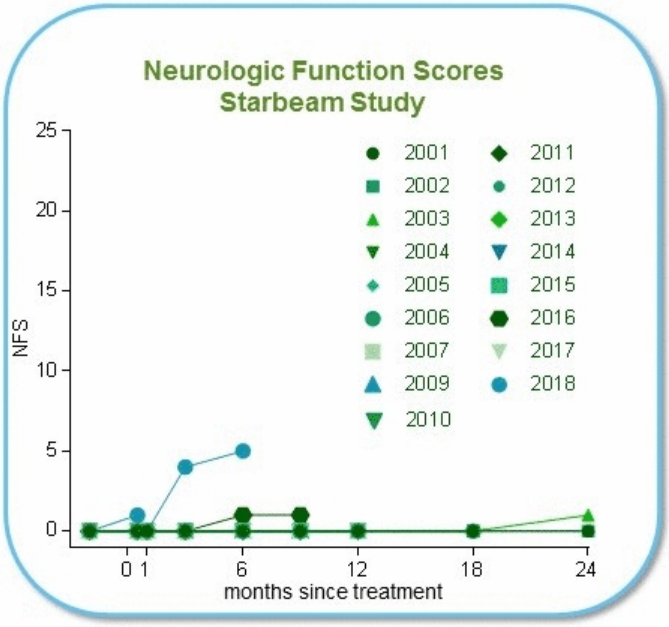
Grade ≥3 non-lab AEs in >1 subject	D-10 to D+43 (N=17)	≥D+44 (N=17)
Febrile neutropenia	13 (76%)	0
Stomatitis	5 (29%)	0
Nausea	3 (18%)	0
Vomiting	2 (12%)	0
Decreased appetite	6 (35%)	0
Epistaxis	2 (12%)	0

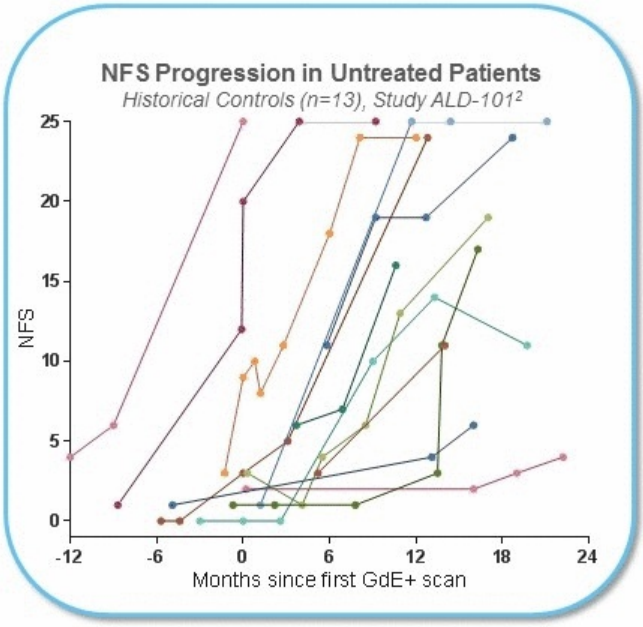
- Study procedures generally well tolerated
- No graft failure or GvHD
- No molecular evidence of clonal dominance or insertional oncogenesis
- No replication competent lentivirus detected to date

- Adverse events consistent with myeloablative conditioning
- Two AEs considered possibly related to drug product: BK-mediated viral cystitis (SAE, grade 3) and tachycardia (grade 1)
- All AEs and SAEs resolved with standard measures

Data as of March 31, 2016

All Patients Remain Free from Major Functional Disabilities, 16/17 Have Stable NFS[1]

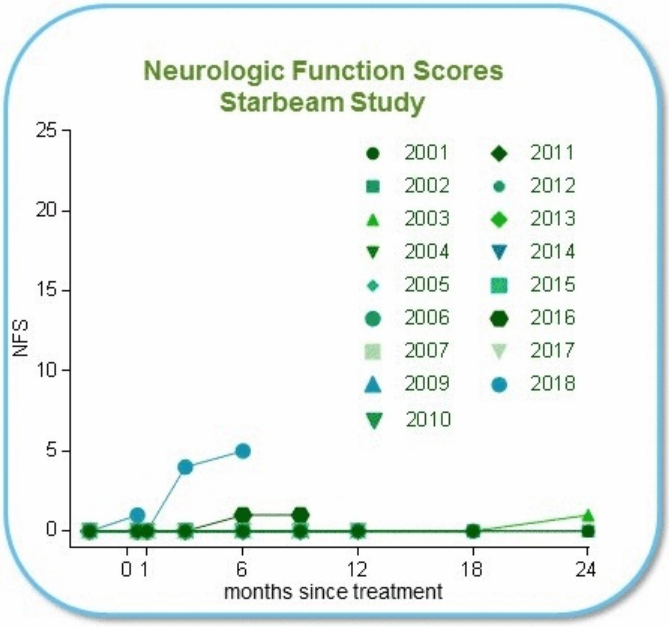


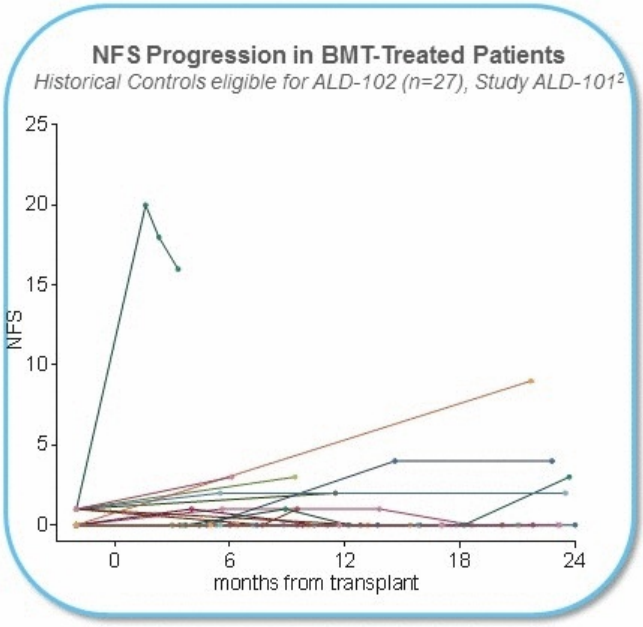


1. Stable NFS: change of <3 points and an absolute NFS ≤ 4. 2. GdE+ untreated subjects regardless of stage of disease with more than 1 NFS score reported (N=13), from time of first GdE+ MRI. Most recent score within 1 year prior to HCT reported as screening score. bluebird bio study ALD-101, manuscript in preparation.

Data as of March 31, 2016

All Patients Remain Free from Major Functional Disabilities, 16/17 Have Stable NFS[1]

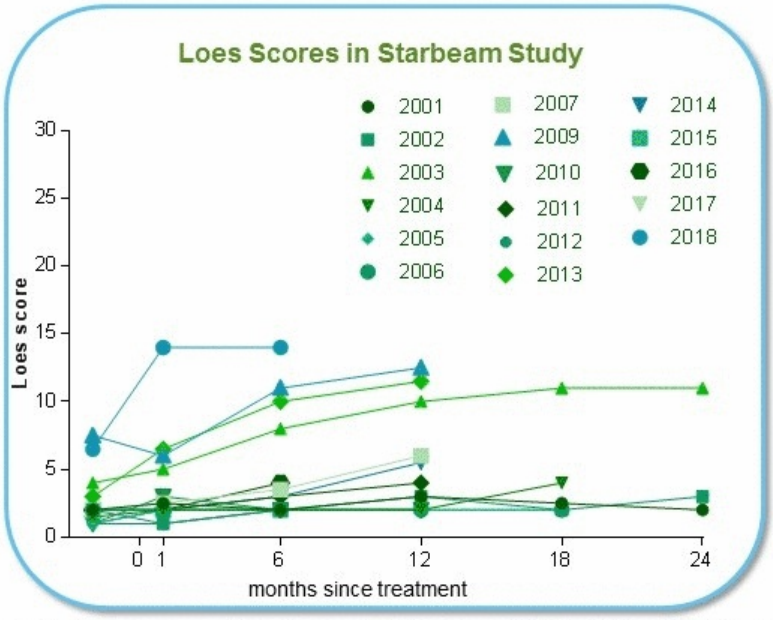


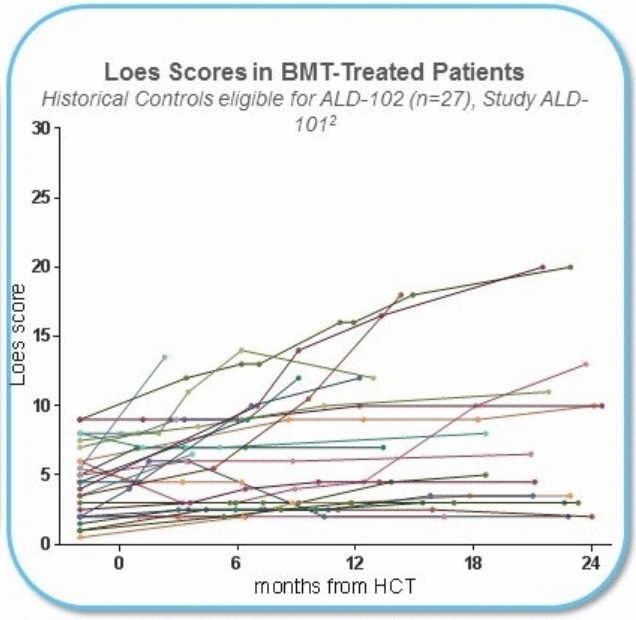


1. Stable NFS: change of <3 points and an absolute NFS ≤ 4. 2. GdE+ HCT treated (N=27) subjects with Loes score ≤9 & NFS ≤1, from time of HCT, all donor sources. Most recent score within 1 year prior to HCT reported as screening score. bluebird bio study ALD-101, manuscript in preparation.

Data as of March 31, 2016

Loes Scores Have Stabilized[1] in 14/17 Patients



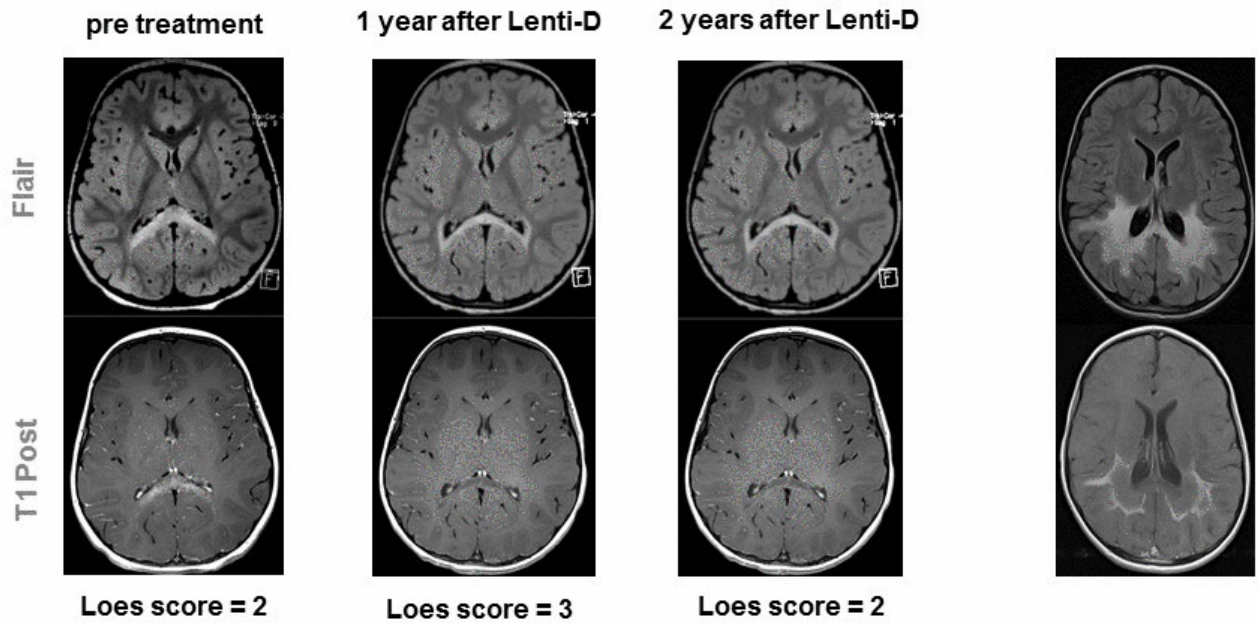


1. Stable Loes score: change of ≤ 5 points or an absolute Loes Score ≤ 9. 2. GdE+ HCT-treated (N=27) subjects with Loes score ≤9 & NFS ≤1, from time of HCT, all donor sources. bluebird bio study ALD-101, manuscript in preparation.
Data as of March 31, 2016

Neuroimaging Outcomes Demonstrate Halting of Disease Progression after Lenti-D Treatment

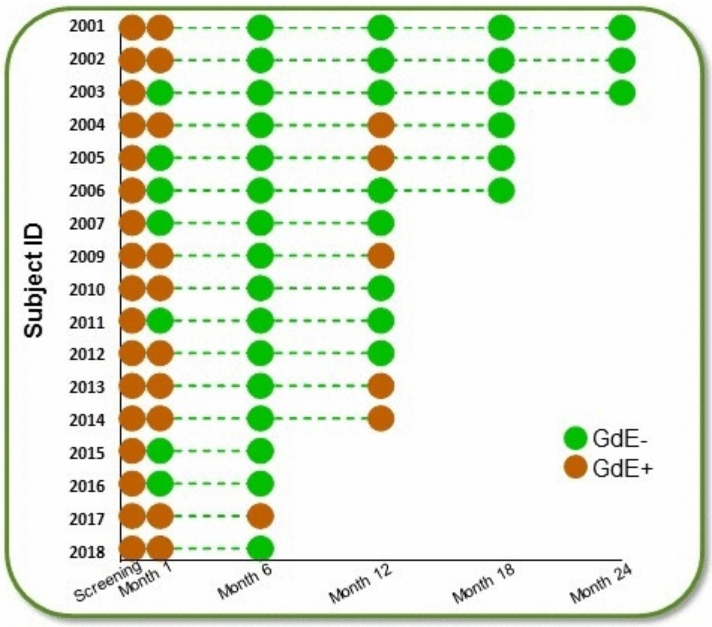
Subject 2001: First Patient Treated in Starbeam Study

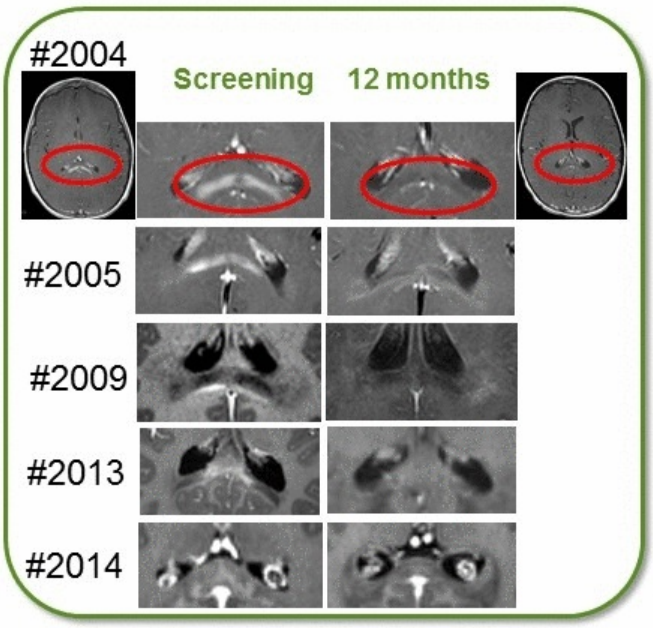
Representative Untreated patient



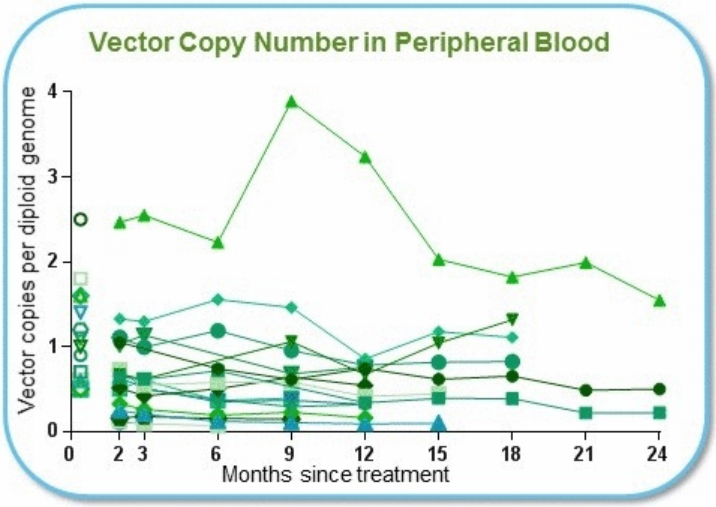
Data as of March 31, 2016

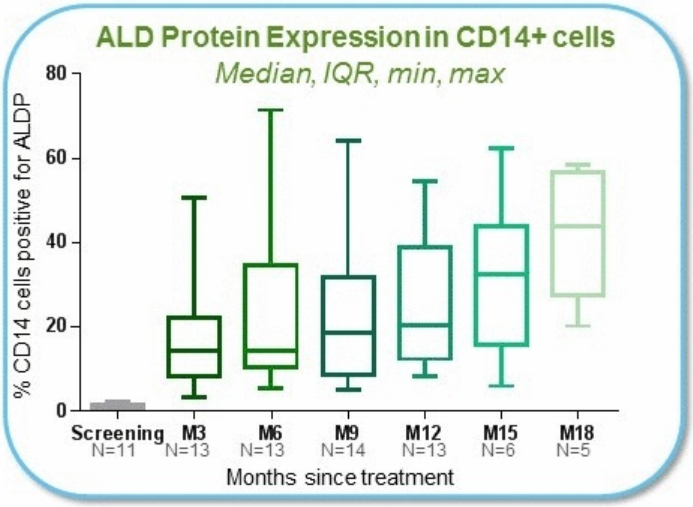
Resolution of Gadolinium Enhancement



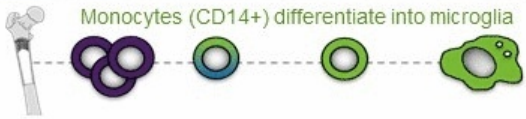


Persistence of Vector-Marked Cells and ALDP Expression





- Median VCN in drug product: 1.0 (0.5-2.5)
- All samples have detectable VCN and ALDP expression at latest follow-up



Summary

Initial Starbeam results suggest early treatment with Lenti-D gene therapy may halt neuro-inflammation and demyelination in most CALD patients, with promising safety

- All subjects remain free of Major Functional Disabilities (MFD) to date
- Stabilization of NFS achieved in 94% (16/17) and Loes score achieved in 82% (14/17)
- Resolution of gadolinium enhancement by month 6 in 94% (16/17)
- Re-appearance of diffuse gadolinium enhancement in 5 subjects, resolved in those (n=2) who have later follow-up

No deaths, graft failure, or GvHD reported to date

- AE profile consistent with myeloablative conditioning with busulfan and cyclophosphamide

Lenti-D gene therapy may offer an alternative to allogeneic bone marrow transplant, particularly for patients with no matched sibling donor

- Additional follow-up is needed to fully assess efficacy, durability of effect and long-term safety

Data as of March 31, 2016

Newborn Screening for ALD

Early detection is critical for boys with CALD

- Rapidly degenerative disease; treatment is most successful in boys who are diagnosed early
- Newborn screening allows boys born with ALD to be monitored for development of symptoms and treated early

The federal government now recommends that states add ALD to their newborn screening panels, and it is up to the states to implement screening. Advocates continue to work on a state-by-state basis to get broader adoption.

New York has already begun screening
- Total number of newborns screened in 2014 and 2015 was 469,515

Connecticut recently started screening

California, Illinois, New Jersey have agreed to add ALD to their state's newborn screening panels

Note: not all boys identified with ALD will convert to cerebral ALD. Literature suggests that 30 to 40% of ALD cases will convert to CALD.

brian's hope | The STOP ALD Foundation | ALDConnect | Aiden Jude Steger Foundation

Anticipated Significant 2016 Milestones

Cash Runway Through 2018


bluebirdbio®
Making Hope a Reality
Transforming the Lives of Patients
with Severe Genetic and Rare Diseases
Nasdaq : BLUE