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): June 14, 2019

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)**	**(IRS Employer Identification No.)**
60 Binney Street, Cambridge, MA		**02142**
(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 (see General Instructions 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))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.01 par value per share	BLUE	The NASDAQ Global Select Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On June 14, 2019, bluebird bio, Inc. ("bluebird") will conduct an investor webcast providing updates in its programs in transfusion-dependent beta-thalassemia and sickle cell disease, including summaries of clinical data from its clinical studies HGB-206, HGB-204, HGB-207, and HGB-212, being presented at the 24th Congress of the European Hematology Association in Amsterdam, The Netherlands. A copy of the presentation is furnished to this Current Report as Exhibit 99.1

The information in Item 7.01 of this Current Report on Form 8-K and Exhibit 99.1 attached hereto is intended to be furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934 (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 or the Exchange Act, except as expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

On June 14, 2019, bluebird issued press releases announcing updated clinical data being presented at the 24th Congress of the European Hematology Association in Amsterdam, The Netherlands from the HGB-206 clinical study in sickle cell disease, and the HGB-204, HGB-207, and HGB-212 clinical studies in transfusion-dependent beta-thalassemia. The full text of bluebird's press releases is filed as Exhibits 99.2 and 99.3 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 June 14, 2019.
99.2	Press release issued by bluebird bio, Inc. on June 14, 2019.
99.3	Press release issued by bluebird bio, Inc. on June 14, 2019.

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.

Date: June 14, 2019

bluebird bio, Inc.

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

Exhibit 99.1

bluebirdbio®
recode for life™

EHA Data Review & ZYNTEGLO® Approval Webcast

June 14, 2019

LET'S RECODE THE STORY

NASDAQ: BLUE

Forward-looking statements

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 preclinical and clinical studies and our research and development programs, our ability to advance product candidates into, and successfully complete, clinical studies, the timing or likelihood of regulatory filings and approvals, and the timing and likelihood of entering into contracts with payors for value-based payments over time or reimbursement approvals, and our commercialization plans for approved products 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 quarterly report on Form 10-Q, 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.

WE **RECODE** FOR LIFE

Our ambition is to recode science, systems and the status quo, so **lives can be lived fully.**






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LET'S RECODE THE SYSTEM

Today's Agenda

SCD Data: HGB-206 Group C	Dave Davidson, MD *chief medical officer*
TDT Data: Northstar (HGB 204), Northstar-2 (HGB-207) and Northstar-3 (HGB-212)	Dave Davidson, MD *chief medical officer*
ZYNTEGLO® Launch Update	Nick Leschly *chief bluebird &* Alison Finger *chief commercial officer*
Q&A	

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Sickle Cell Disease (SCD)

HGB-206 Group C

NASDAQ: BLUE

Living with Sickle Cell Disease

Sickle Cell Disease

- Severe blood disorder that causes anemia, frequent pain crises and shortened lifespan
- Global annual birth incidence ~ 300,000 - 400,000
- Mean age of death in the U.S. is 44 years[1]

Bridgett's Experience

- Diagnosed at 17
- Had over 140 gallstones before diagnosis
- First transfusion at 19, received over 300 transfusions
- Chronic pain
- Constantly concerned about what may trigger the next crisis

[1]Paulukonis et al, California's Sickle Cell Data Collection Cohort, 2005-2015* ASH 2017*

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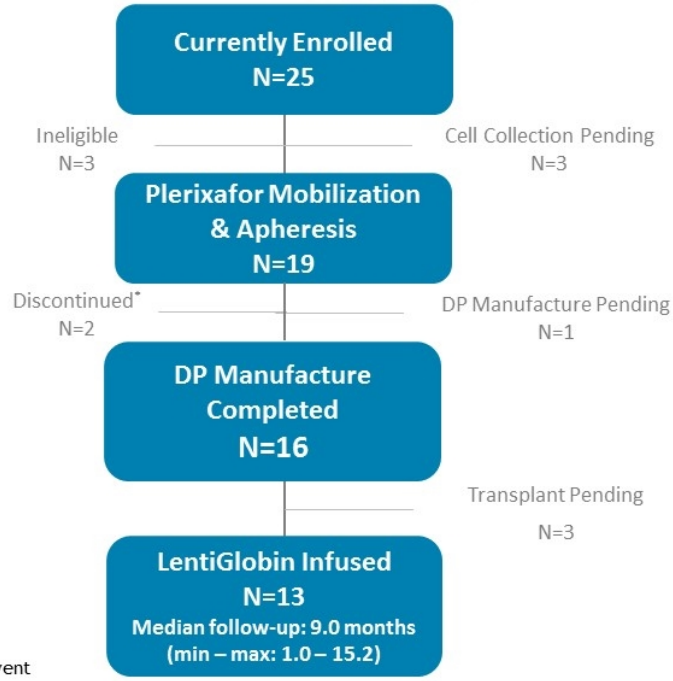
HGB-206 Group C: Disposition
Currently enrolling



*1 withdrew consent, 1 discontinued due to adverse event
Definitions: HSCs, hematopoietic stem cells

Data as of 7 March 2019

HGB-206 Group C: Patient characteristics

N=19 patients who started cell collection

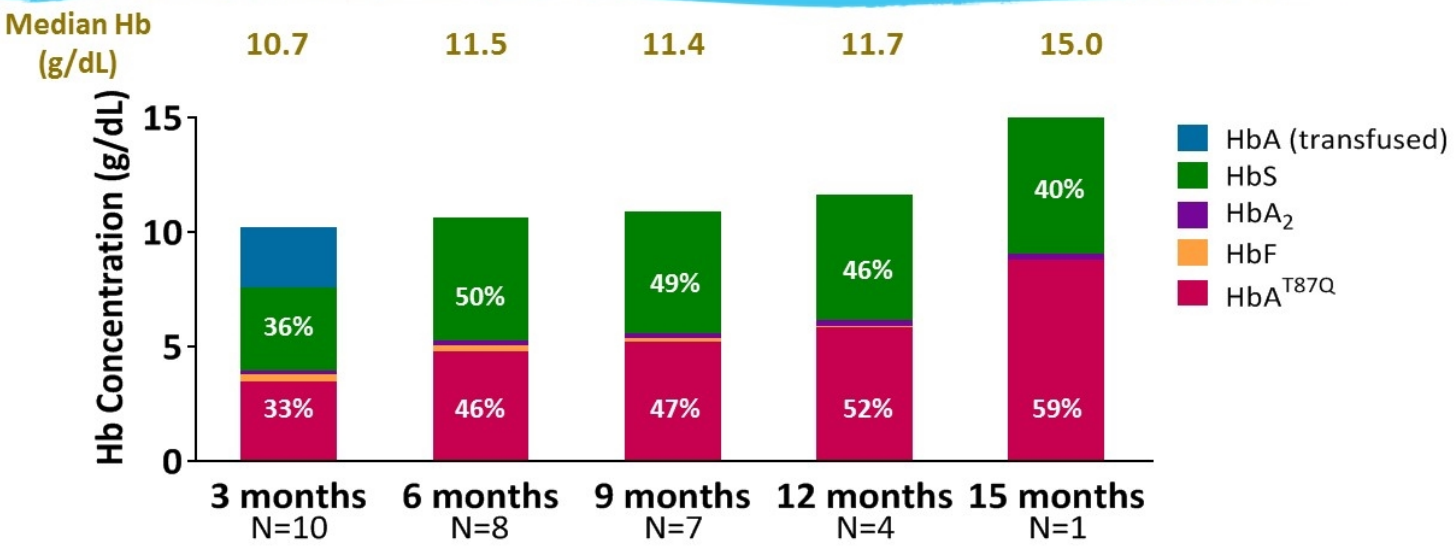
Parameter	Group C N=19
Age at consent, years median (min – max)	**26** (18 – 36)
Gender	**8F 11 M**
Genotype, β^S/β^S	**19**
SCD History	
Hydroxyurea[#], n	**11**
VOCs[*], n Annualized no. of events, median (min – max)	**15** **4.0** (2.0 – 13.5)
ACS[†], n Annualized no. of events, median (min – max)	**2** **1** (1 – 1)
Stroke, n	**3**
TRJV > 2.5 m/s, n	**1**

*≥ 2 events/year in preceding 2 years; †≥ 2 episodes in preceding 2 years, with ≥ 1 episode in the past year or in the year prior to the initiation of regular transfusions;
#Within 30 days prior to informed consent

Definitions: ACS, acute chest syndrome; F, female; M, male; TRJV, tricuspid regurgitant jet velocity; VOC, vaso-occlusive crisis

Data as of 7 March 2019

HGB-206 Group C: Median HbS ≤ 50% of total Hb in patients with ≥ 6 months of follow-up post LentiGlobin treatment



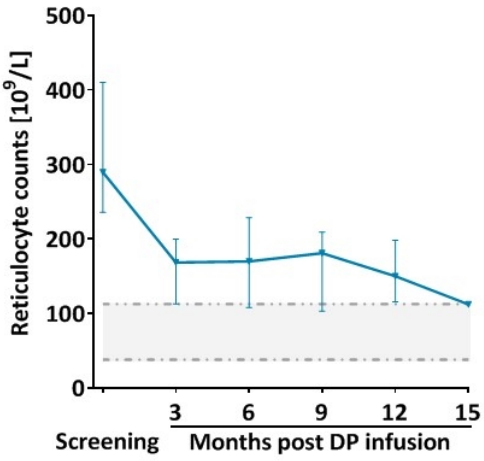
Total Hb and HbA^{T87Q} ranged from 10.2 – 15.0 g/dL and 4.5 – 8.8 g/dL, respectively, at last visit in patients with ≥ 6 months of follow-up

Definitions: % represent median Hb fractions as % of total; Hb, hemoglobin

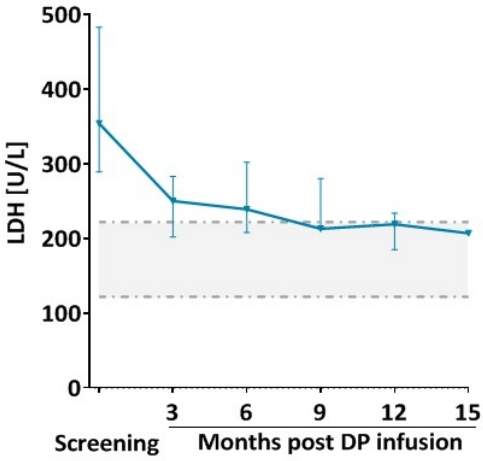
Data as of 7 March 2019 [9]

HGB-206 Group C: Decreased hemolysis following LentiGlobin treatment

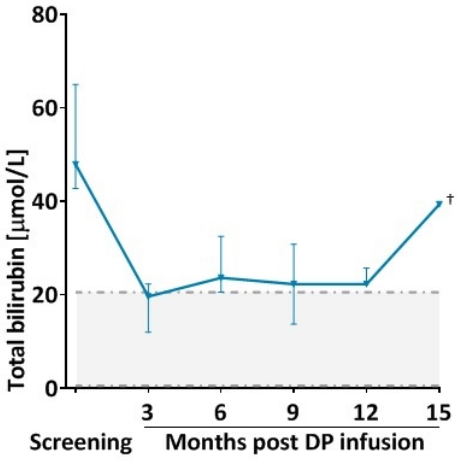
Reticulocyte Counts



Lactate Dehydrogenase



Total Bilirubin



N*	13	10	8	7	5	1
	11	10	7	7	5	1
	13	10	8	7	5	1

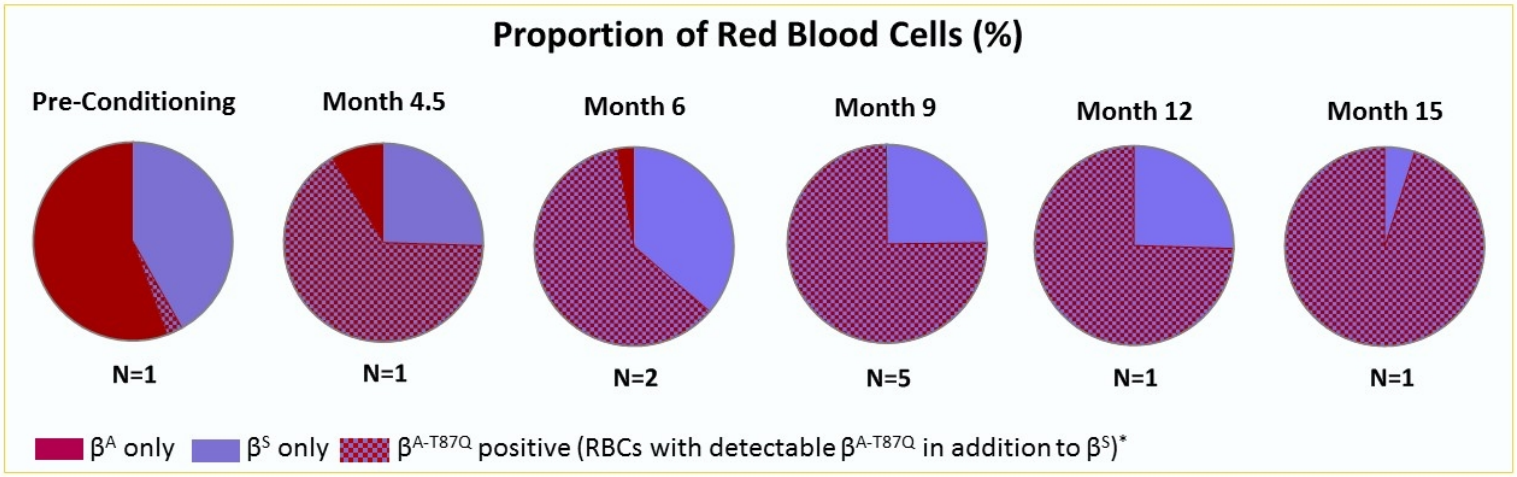
Median (Q1, Q3) depicted; Dot-dash lines denote lower and upper limits of normal values; *Shows number of patients for whom data are available; † Total bilirubin at last follow-up remains > 2-fold lower than at screening

Definition: LDH, lactate dehydrogenase

Data as of 7 March 2019

HGB-206 Group C: On average, ≥ 70% of RBCs from patients treated with LentiGlobin contain $\beta^{A\text{-}T87Q}$ by month 9

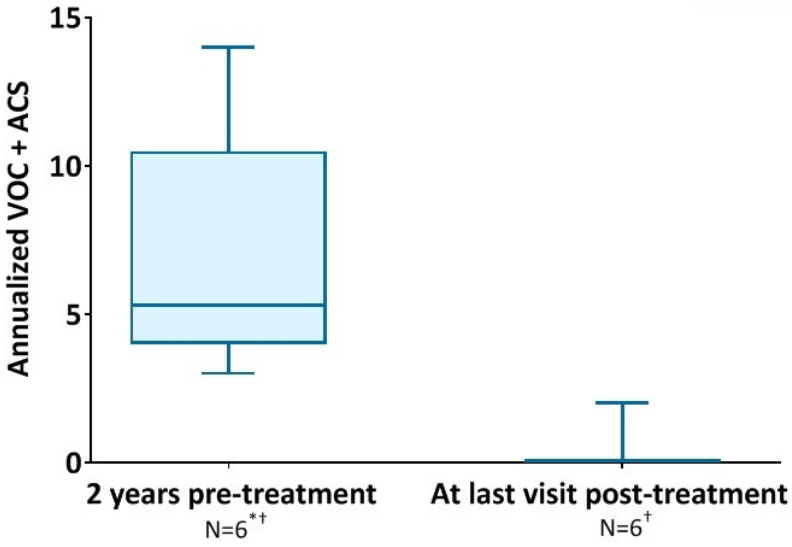
- Single RBC western blot assay was performed in multiple patient samples



Mean is depicted - if N=1, data show technical replicates; *Pre-conditioning sample does not contain any $\beta^{A\text{-}T87Q}$, signal represents false positives
Definition: RBCs, red blood cells

Data as of 7 March 2019

HGB-206 Group C: Reduction in annualized rate of VOC plus ACS post treatment



	2 years pre-treatment	At last visit post-treatment
	N=6*†	N=6†
Median	**5.3**	**0**
(min – max)	**(3 – 14)**	**(0 – 2)**

- No ACS or serious VOCs occurred in any Group C patient post-LentiGlobin treatment to date
- 1 non-serious Grade 2 VOC was observed in 1 pt ~3.5 months post DP infusion

Investigator-reported adverse events of VOC or ACS are shown;
*Patients with ≥ 1 VOC/ACS in the 2 years before Informed Consent; †Patients with ~ ≥ 6 months of follow-up post DP infusion
Definitions: ACS, acute chest syndrome; DP, drug product; VOCs, vaso-occlusive crises

Data as of 7 March 2019

HGB-206 Group C: Safety profile consistent with myeloablative busulfan conditioning

Non-hematologic grade ≥ 3 AEs* *Post DP infusion in ≥ 2 patients*	**N=13 n (%)**
Febrile neutropenia	10 (77)
Stomatitis	7 (54)
Abdominal pain upper	2 (15)
Alanine aminotransferase increased	2 (15)
Blood bilirubin increased	2 (15)
Nausea	2 (15)
Serious AEs* *Post DP infusion in ≥ 2 patients*	**N=13 n (%)**
Nausea	2 (15)
Vomiting	2 (15)

- Serious AEs post DP infusion were reported in 6 patients
- No DP-related adverse events
- No cases of veno-occlusive liver disease observed to date
- No graft failure or deaths reported
- No vector-mediated RCL detected and no evidence of clonal dominance across LentiGlobin studies†
- No further cases of MDS have been observed across studies of LentiGlobin†▪

*Hematologic AEs commonly observed post-transplant have been excluded;
†As of 20 Sep 2017 (HGB-205); 13 Dec 2018 (HGB-204, HGB-207), and 12 Apr 2019 (HGB-212)
▪One patient in Group A was reported to have MDS at last data update (ASH 2018). There was no evidence of LVV-mediated oncogenesis and the MDS SAE was considered unlikely related to LentiGlobin gene therapy.
Definitions: AE, adverse event; DP, drug product; RCL, replication competent lentivirus

Data as of 7 March 2019

Accelerated development plan using novel composite primary endpoint based on hemoglobin

EXPANDED

Updated Primary Endpoint

Up to additional 21 patients

Expanded age range

HGB-206 Group C

Sickle Cell Disease, history of vaso-occlusive events (VOEs) over 24 months

Ongoing Phase 1/2, single arm, multi-center, U.S. study
N=41 (Group C)

- Primary Endpoint: HbA^{T87Q} and Total Hb
- Key Secondary Endpoint:
  - Reduction in severe VOEs
- ≥12 years of age - ≤50 years of age

HGB-210

Sickle Cell Disease, history of VOEs over 24 months

Phase 3, single arm, multi-center, global study

- Primary Endpoint: HbA^{T87Q} and Total Hb
- Key Secondary Endpoint:
  - Reduction in severe VOEs

NEW

Planned for 2019

Additional Clinical Investigation in Other Patient Types and Ages Planned

Plans Based on Ongoing Engagement with Regulators

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Transfusion-Dependent β-thalassemia (TDT)

NASDAQ: BLUE

Conditional approval granted in EU for patients with TDT and non-β^0/β^0 genotypes

zynteglo®
(autologous CD34$^+$ cells encoding $\beta^{A\text{-}T87Q}$-globin gene)

Gene therapy for patients 12 years and older with transfusion-dependent β-thalassemia (TDT) who do not have a β^0/β^0 genotype, for whom hematopoietic stem cell (HSC) transplantation is appropriate but a human leukocyte antigen (HLA)-matched related HSC donor is not available

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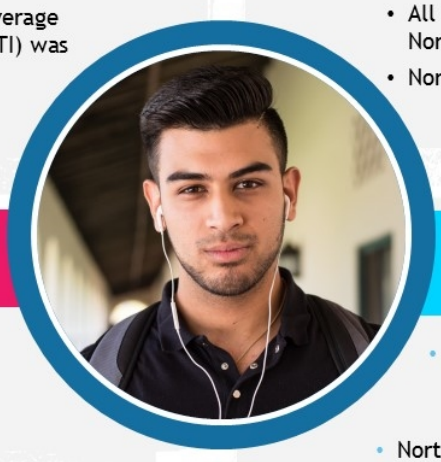
ZYNTEGLO® is the first and only one-time therapy for TDT now approved in the EU for people with TDT and non-β^0/β^0 genotypes

ZYNTEGLO has the potential to increase total Hb to normal levels

- Northstar-2 (HGB-207): Median weighted average total Hb during transfusion independence (TI) was 12.4 g/dL (n=4)

Following engraftment and achievement of TI, the effects of ZYNTEGLO are expected to be lifelong

- All non- β^0/β^0 patients in Northstar (HGB- 204) and Northstar-2 who achieved TI, maintained TI
- Northstar: TI maintained up to 3.8 years
  - Northstar: Reduction in iron overload seen at 4 years (n=4)



The majority of evaluable patients achieved TI

- Northstar and HGB-205: 11/14 patients with non-β^0/β^0 genotypes achieved TI
- Northstar-2: 4/5 patients achieved TI

Gene therapy derived Hb (HbA^{T87Q}) supports total Hb production soon after infusion

- Northstar-2: Median total hemoglobin at 6 months: 11.9g/dL; HbA^{T87Q} was 9.5 g/dL (n=11)
- Northstar, non-β^0/β^0 patients: Median 6 month Hb was 9.7 g/dL; HbA^{T87Q} was 4.7 g/dL (n=10)

Full Indication: Gene therapy for patients 12 years and older with transfusion-dependent β-thalassemia (TDT) who do not have a β^0/β^0 genotype, for whom hematopoietic stem cell (HSC) transplantation is appropriate but a human leukocyte antigen (HLA)-matched related HSC donor is not available

Data as of 13 December 2018

Broad clinical development program continues

NORTHSTAR STUDY	HGB-204 *Complete*	• Original manufacturing process • All genotypes • N=18
HGB-205	HGB-205 *Complete*	• Original manufacturing process • N=4
NORTHSTAR-2 STUDY	HGB-207	• Refined manufacturing process • Non-β^0/β^0 genotypes • N=23
NORTHSTAR-3 STUDY	HGB-212	• β^0/β^0 genotypes • Refined manufacturing process • N=15

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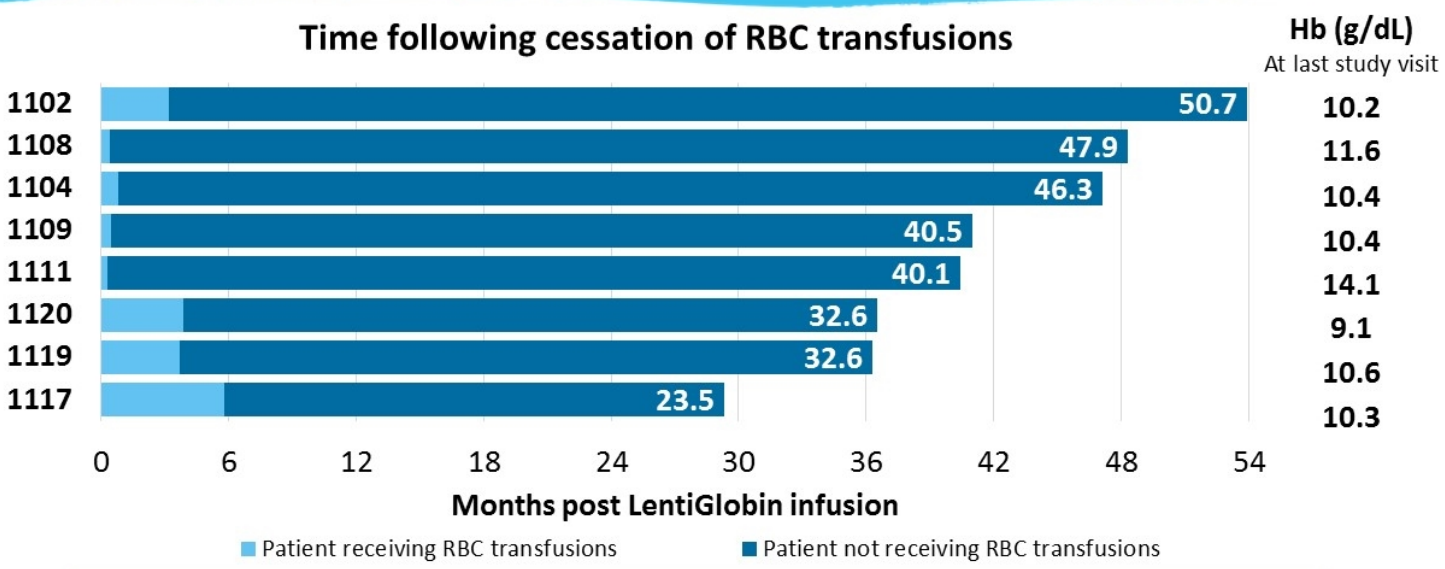
HGB-204: 8/10 patients with non-β^0/β^0 genotypes achieved transfusion independence

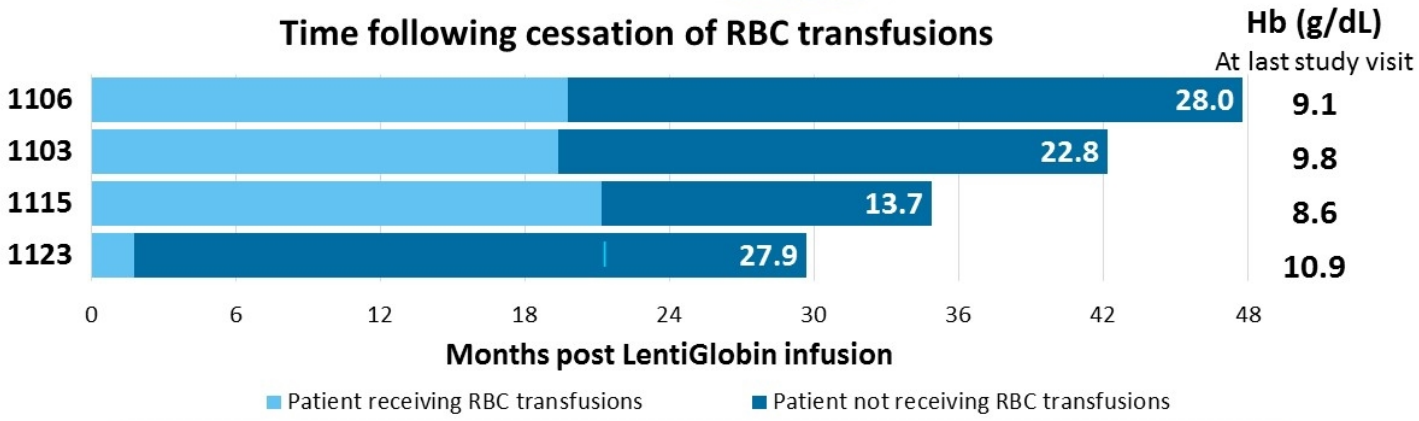


Median duration of TI: 38.0 months (min – max: 21.2 – 45.3 months); responses are ongoing
Median weighted average Hb during TI: 10.3 g/dL (min – max: 9.3 – 13.2 g/dL)

Definitions: Hb, hemoglobin; RBC, red blood cell; TI, transfusion independence (weighted average Hb ≥ 9 g/dL without RBC transfusions for ≥ 12 months)

Data as of 13 December 2018

HGB-204: 4/8 patients with β^0/β^0 genotypes have been transfusion free for > 12 months



3/8 patients with β^0/β^0 genotypes have achieved transfusion independence
(weighted average Hb ≥ 9 g/dL without any red RBC transfusions for ≥ 12 months)

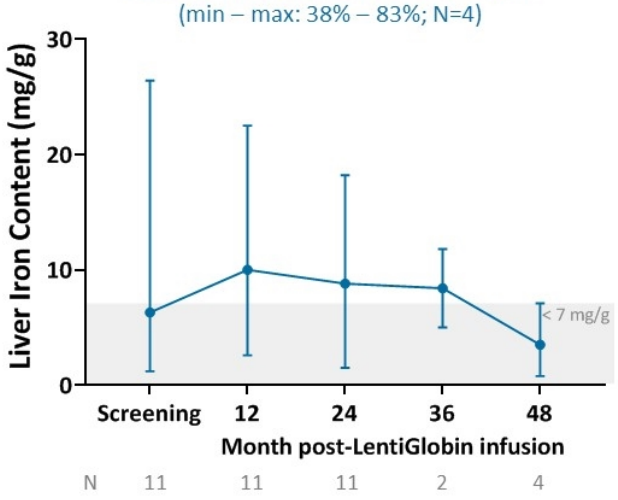
Median duration of TI: 16.4 months (min – max: 16.1 – 20.8 months) All responses are ongoing

Median weighted average Hb during TI: 9.9 g/dL (min – max: 9.5 – 10.1 g/dL)

| Patient had a single transfusion for an acute event of cat scratch disease. Hb, hemoglobin; RBC, red blood cell; TI, transfusion independence

HGB-204: Liver iron concentration decreased in patients who achieved transfusion independence

56% median reduction in LIC between baseline and M48 with re-initiation of iron chelation
(min – max: 38% – 83%; N=4)



N	11	11	11	2	4

Patients re-initiated iron chelation therapy a median of 13 months after LentiGlobin infusion (min – max: 2 – 15 months)

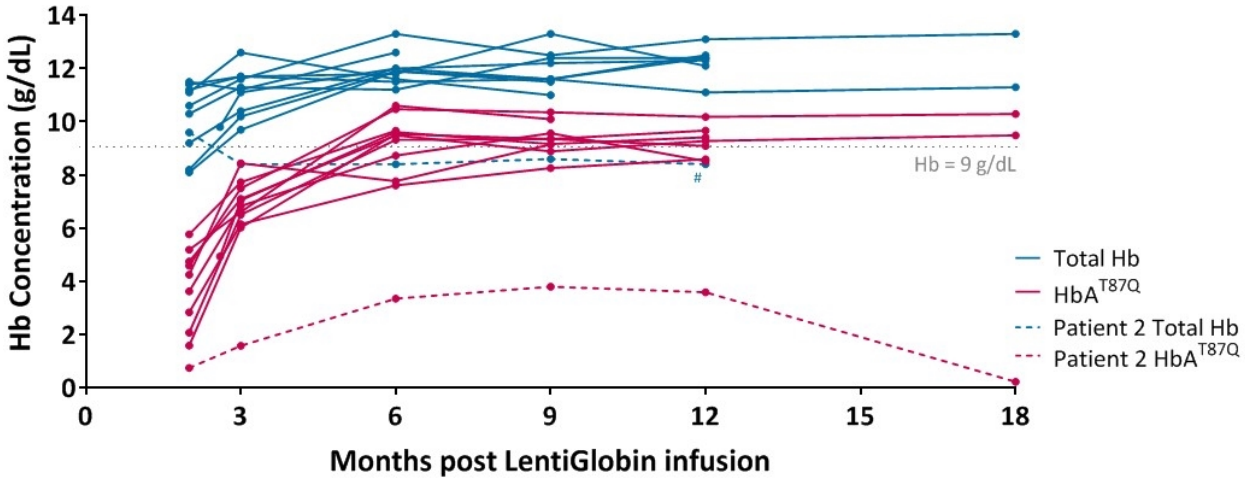
Medians (min, max) depicted
Definitions: LIC, liver iron concentration; M, month

Data as of 13 December 2018

HGB-207: Stable total Hb and gene therapy-derived HbA^{T87Q} in 10/11 patients with ≥ 6 months follow-up

Median Hb in patients free from transfusions at last study visit (g/dL)

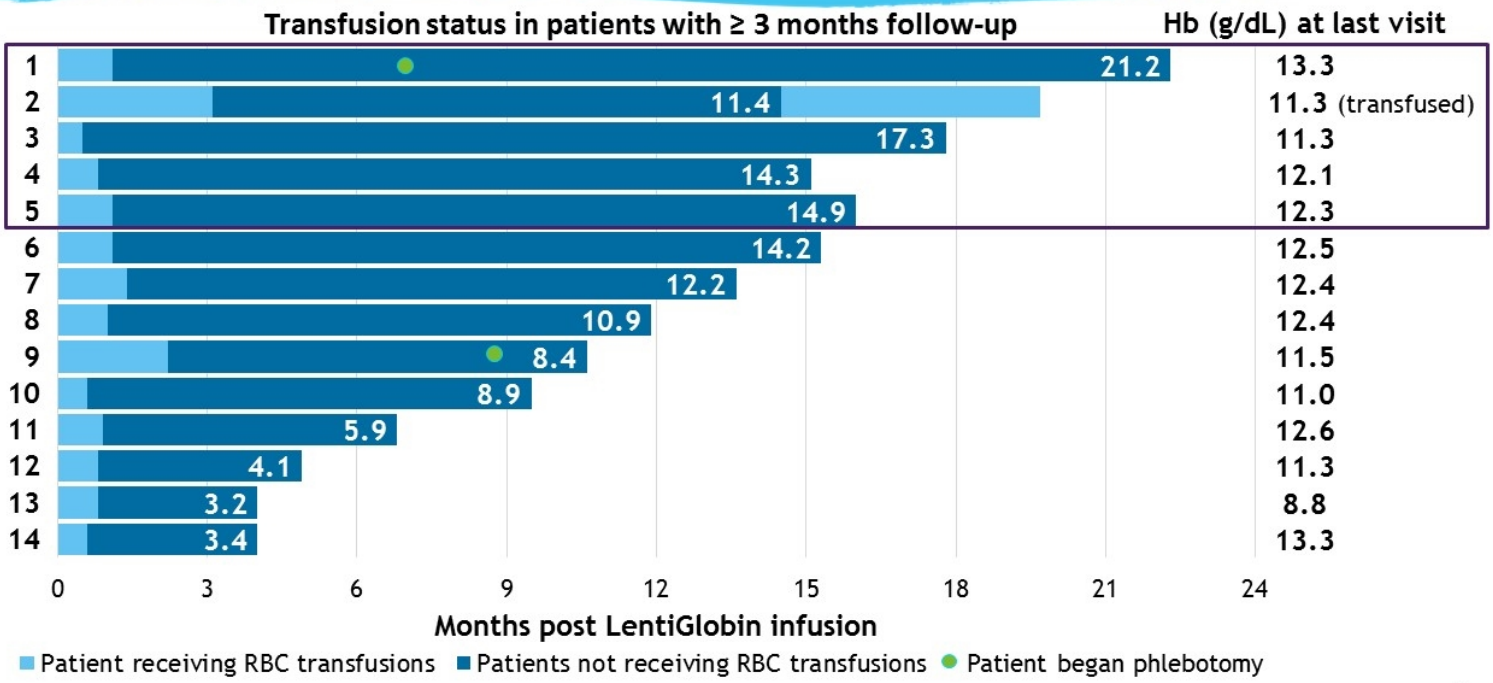
	M6 (n=10)	M9 (n=9)	M12 (n=7)	M18 (n=2)
Total Hb	11.9	11.6	12.4	12.3
HbA^{T87Q}	9.5	9.4	9.3	9.9



#Last Hb before patient restarted red blood cell transfusions
Definitions: Hb, hemoglobin

Data as of 13 December 2018

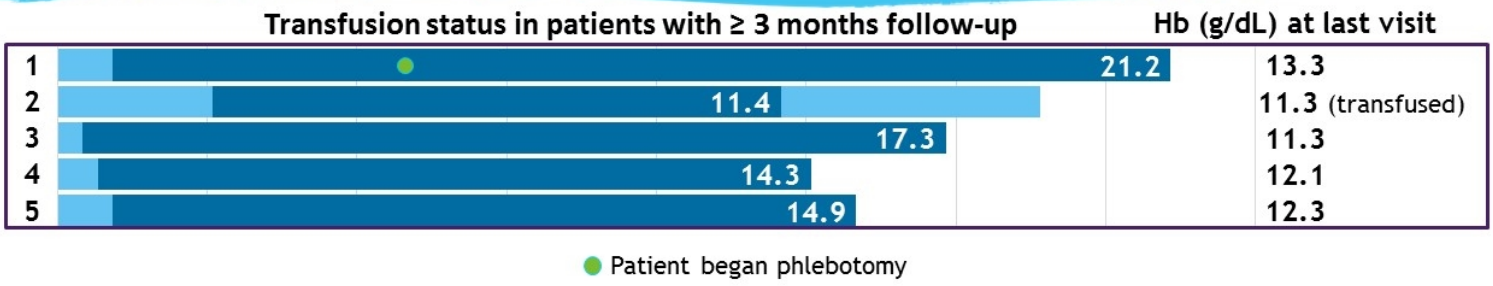
HGB-207: 8.8 – 13.3 g/dL total Hb in patients who have stopped RBC transfusions for ≥ 3 months (n=13)



Definitions: Hb, hemoglobin; RBC, red blood cell

Data as of 13 December 2018

HGB-207: 4/5 (80%) evaluable patients achieved the primary endpoint of transfusion independence

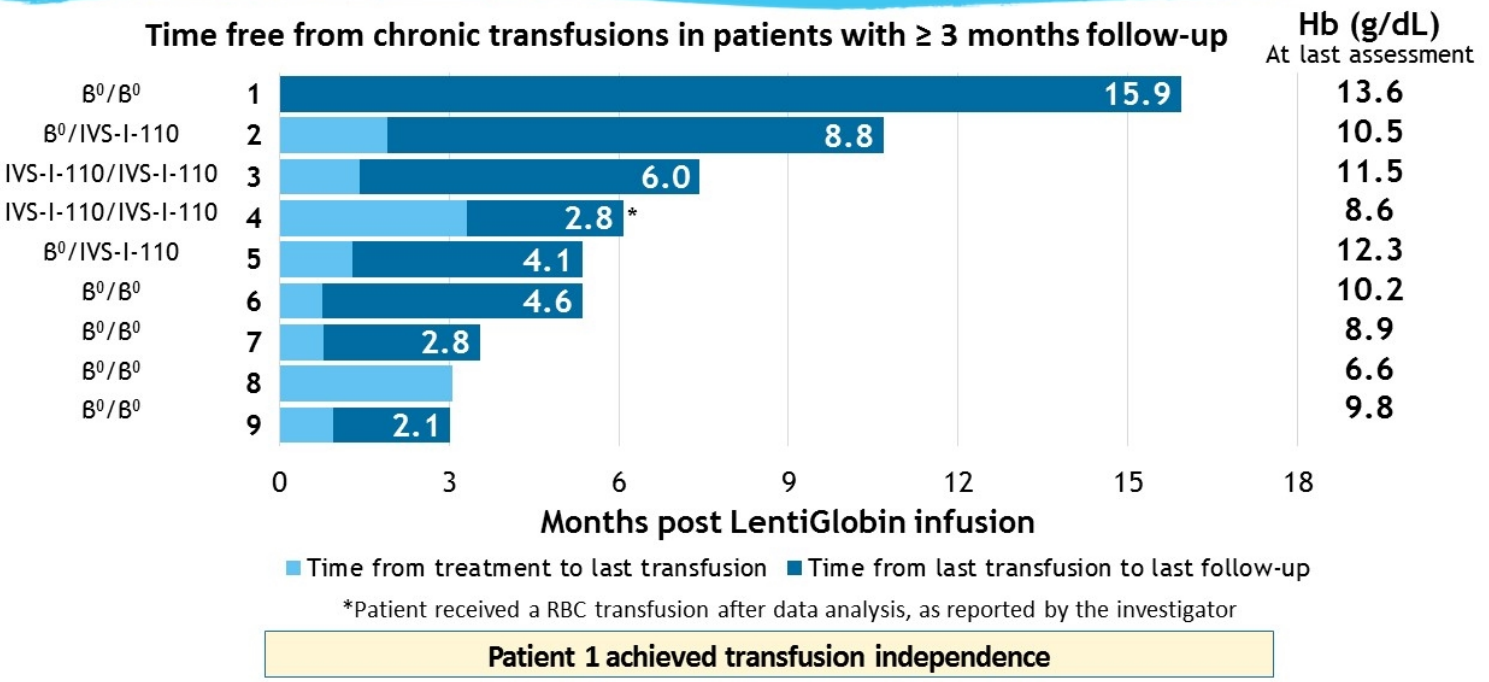


- **4/5 (80%) evaluable patients achieved the primary endpoint of transfusion independence (TI)**
  Weighted average hemoglobin ≥ 9 g/dL without any transfusions for ≥ 12 months
- **Median duration of TI: 13.6 months** (min – max: 12.0 – 18.2 months)
  All responses are ongoing
- **Median weighted average Hb during TI of 12.4 g/dL** (min – max: 11.5 – 12.6 g/dL)

Definitions: Hb, hemoglobin; RBC, red blood cell

Data as of 13 December 2018

HGB-212: Hb of 10.2 – 13.6 g/dL in patients off RBC transfusions for ≥ 3 months (n=5)



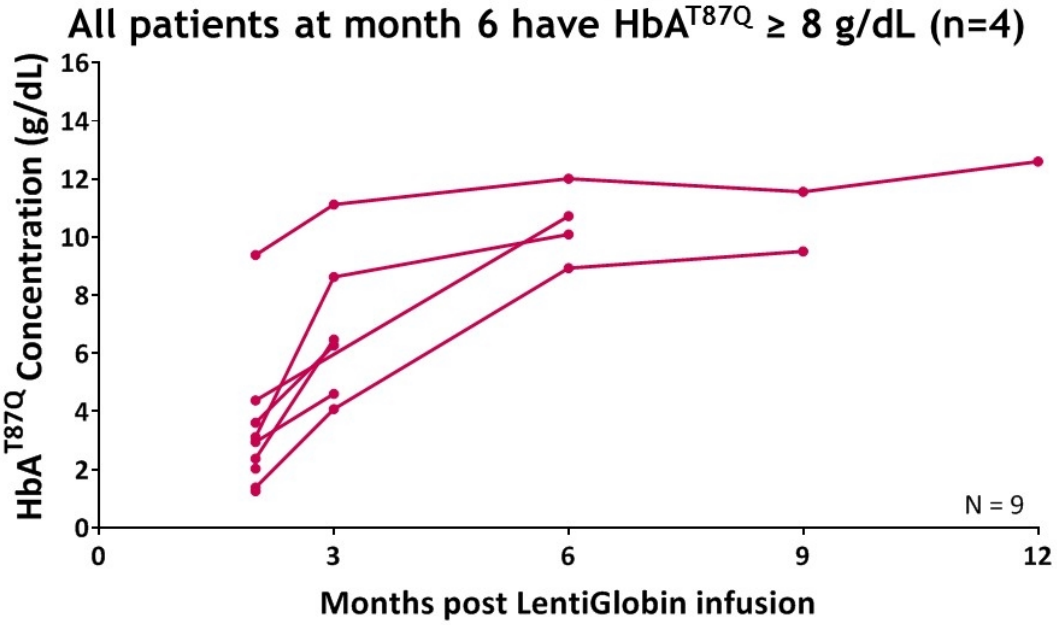
*Patient received a RBC transfusion after data analysis, as reported by the investigator

Patient 1 achieved transfusion independence

Definitions: Hb, hemoglobin; RBC, red blood cell

Data as of 12 April 2019

HGB-212: HbA^{T87Q} in patients following treatment with LentiGlobin



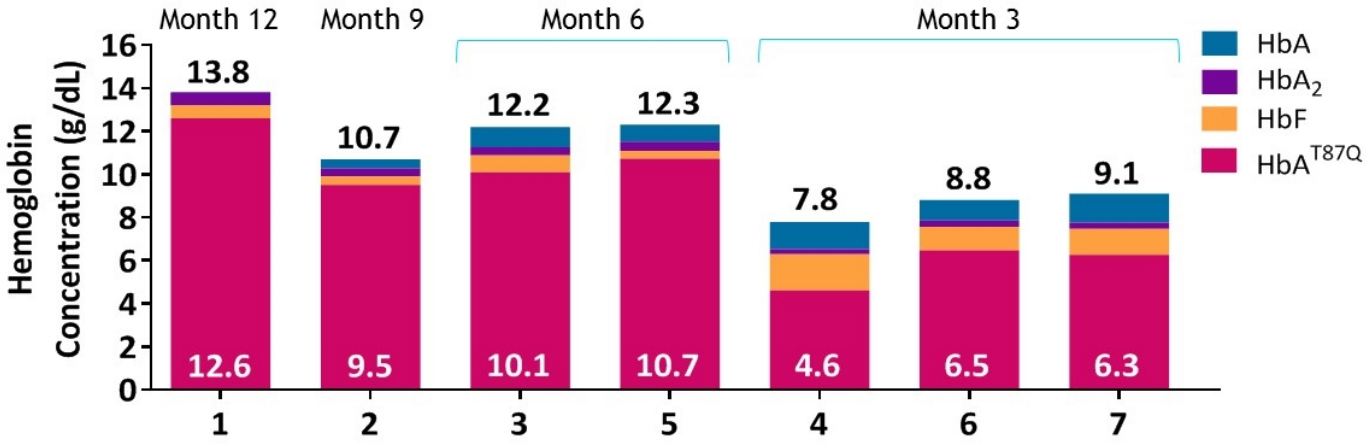
Definition: Hb, hemoglobin

Data as of 12 April 2019

HGB-212: Gene therapy-derived HbA^{T87Q} significantly contributes to Hb

59 – 91% of total Hb is HbA^{T87Q}

Hb fractions in patients with ≥ 3 month visit



	1	2	3	5	4	6	7
Genotype	β^0/β^0	β^0/ IVS-I-110	IVS-I-110/ IVS-I-110	β^0/ IVS-I-110	IVS-I-110/ IVS-I-110	β^0/β^0	β^0/β^0
DP/Peripheral VCN	3.1/2.8	3.65/3.3	3.1/5.1	4.3/2.0	2.1/0.4	2.25/0.8	3.0/0.5

Definitions: DP, drug product; Hb, hemoglobin; VCN, vector copy number

Data as of 12 April 2019

Clinical data supports patient and physician desired outcomes in TDT & SCD

HGB-206 Phase 1

- ✓ *In patients who were at least 6 months post-treatment, median level of abnormal sickle hemoglobin (HbS) was reduced to ≤ 50% of total Hb*
- ✓ *At up to 15 months post-treatment there were no reports of serious vaso-occlusive crisis or acute chest syndrome in Group C*

NORTHSTAR STUDY Phase 1/2

- ✓ *Up to 3.8 years of transfusion independence in Phase 1/2 (HGB-204) study in patients with TDT who do not have a β^0/β^0 genotype*

NORTHSTAR-2 STUDY Phase 3

- ✓ *80% of evaluable patients achieved transfusion independence in ongoing (HGB-207) study of patients with TDT who do not have a β^0/β^0 genotype*
- ✓ *13/14 were free from transfusions for at least 3 months with total Hb from 8.8-13.3 g/dL at the time of the last study visit*

NORTHSTAR-3 STUDY Phase 3

- ✓ *Total hemoglobin levels of 10.2 - 13.6 g/dL in patients who have β^0/β^0 genotype or IVS-I-110 mutation and were free of transfusions for at least three months in ongoing Phase 3 (HGB-212) study*

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Commercial launch update

A system NOT setup for one-time potentially curative treatments

CNBC

"The debate over price is fundamentally a debate over access. Gene therapies and other treatments that can cost millions of dollars can still be a relative bargain for what they give patients and society if they're able to cure a disease that would severely limit or even end life."

Scott Gottlieb, M.D. Former FDA Commissioner

HEALTH PAYER INTELLIGENCE

"While … therapies that are in the pipeline offer the promise of dramatic health improvements, their upfront costs are significant, which makes it imperative that we work together to find creative, value-based payment approaches that tie reimbursement level to both short-term and long-term efficacy."

Michael Sherman, M.D.
Harvard Pilgrim Chief Medical Officer

FiercePharma

"Gene therapy either works or it doesn't… If the product succeeds, it should be reimbursed at a robust level, because the pharmaco-economics over the course of time are extremely positive. If it doesn't work, the payer, whether it's public or private, shouldn't have to bear the burden. We're moving in that direction."

Peter Pitts
Former FDA Assistant Commissioner

TRADITIONAL CHRONIC FOR LIFE MODEL

bluebirdbio
recode for life

Our commitment to recode the status quo

BLUE VALUE PRINCIPLES

- Focus on patient innovation and access
- Creative and disruptive
- Flexible and share risk
- Transparent, proud and proactive
- Don't do silly short-sighted stuff

&

Unapologetically fund & reward innovation that matters

Focus on **real value** delivered to the patient & system

Don't truncate value because it's a one-time potentially curative treatment

Don't price at what you can get away with or what the market can bear

bluebirdbio
recode for life

Our approach - VALUE-BASED PAYMENT over time based on OUTCOME

	OBJECTIVE	STRATEGIC APPROACH
1	FAIR VALUE RECOGNITION	✓ *Lifetime cost-time effectiveness timeframe* ✓ *Base value only on patient QOL and Life Extension*
2	SHARED RISK	✓ *Pay ONLY IF the treatment works* ✓ *Put UP TO 80% of the price at risk based on success*
3	PER PATIENT AFFORDABILITY	✓ *Spread payments over UP TO A FIVE YEAR period* ✓ *NO PRICE INCREASES above CPI*
4	HEALTH SYSTEM AFFORDABILITY	✓ *NO COST after payment period (vs. for life)* ✓ *Recode system to catalyze change*

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Keeping it Focused on the Patient: Living with TDT

- **Potentially fatal genetic disease caused by mutations in the β-globin gene that result in reduced or absent hemoglobin**
- **Despite advances in iron management, TDT patients suffer from serious complications and organ damage caused by excess iron**
- **TDT patients have a lifelong challenge and currently rely on chronic treatments that accumulate in costs over decades**

LAURICE'S EXPERIENCE:

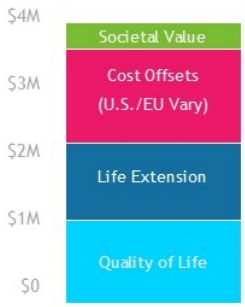
- Hemoglobin of 6.9 g/dL growing up [normal range for females: 12.1-15.1 g/dL][1]
- Congestive heart failure at 9 and 25
- Splenectomy at 10, tonsillectomy at 13, gall bladder removal at 22
- Severe osteoporosis
- Chronic pain
- Under care of PCP, cardiologist, hematologist, endocrinologist, and a pain specialist
- Lost many friends with TDT

1. National Institutes of Health (NIH). *Hemoglobin*. https://medlineplus.gov/ency/article/003645.htm.

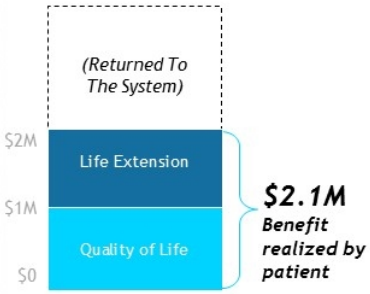
What has (and has not) gone into assessing the value of ZYNTEGLO®?

We measure the value of ZYNTEGLO based on impact on patients:
Life extension and quality of life improvements*

Traditional
All Inclusive Calculation



ZYNTEGLO
Intrintic Value



ZYNTEGLO
Actual Price Considerations

- The expected lifelong clinical benefits of ZYNTEGLO drive its intrinsic value
- The resulting cost offsets are returned to the healthcare system
- The ZYNTEGLO payment model protects health care systems from bearing the cost of ineffective therapy
- ZYNTEGLO is a good health care investment and is cost-effective when considering a range of accepted thresholds in Europe

bluebirdbio recode for life™

*We have quantified the impact on patient quality of life, survival, treatment cost and society using established health economic modeling techniques.

ZYNTEGLO® payment and pricing: value & outcome based, 5 year cap @ risk

- ✓ First Year Payment: €315K*
- ✓ Five Year Total Payment With 100% Success: €1.575M

A one-time treatment expected to deliver lifelong benefit with 5 years of cost versus continual, lifelong treatment and cost

*Based on exchange rate of 1 Euro = $1.13196 USD on June 12, 2019, First Year Payment in USD terms is: $356,567; Five Year Total Payment With 100% Success: $1,782,837

What are next steps and how is launch readiness progressing?

EC Decision

- Team in place; completing set-up and working to activate QTCs
- Actively engaging payers
- Progressing forward with dossier submissions
- Working in collaboration with EMA to finalize commercial drug product specifications and manufacturing parameters

Each Journey is Different

Country-by-Country Recoding Will Play Out Over Time

BLUE style commercial success factors

In the near-term, product revenue is not the most telling indicator on European TDT launch progress

- Payment models may vary by country
- Focus on establishing the commercial model and operations for the long-term

Performance metrics that we will be tracking and sharing

QTC contracts in place

Pricing approval by country

Commercial patient infusions

Learnings and local market insights to inform continuous innovation

Q & A

Exhibit 99.2

bluebird bio Presents New Data for LentiGlobin® Gene Therapy for Sickle Cell Disease (SCD) at 24th European Hematology Association (EHA) Congress

In patients who were at least six months post-treatment with LentiGlobin for SCD, median level of abnormal sickle hemoglobin (HbS) was reduced to ≤50 percent of total Hb

At up to 15 months post-treatment with LentiGlobin, there were no reports of serious vaso-occlusive crisis or acute chest syndrome in Group C

CAMBRIDGE, Mass.—(BUSINESS WIRE)—June 14, 2019—bluebird bio, Inc. (Nasdaq: BLUE) announced new data from patients in Group C of its ongoing Phase 1/2 HGB-206 study of the company's investigational LentiGlobin® gene therapy for sickle cell disease (SCD) today at the 24th European Hematology Association (EHA) Congress in Amsterdam, the Netherlands.

SCD is a serious, progressive and debilitating genetic disease caused by a mutation in the β-globin gene that leads to the production of abnormal sickle hemoglobin (HbS), causing red blood cells (RBCs) to become sickled and fragile, resulting in chronic hemolytic anemia, vasculopathy and painful vaso-occlusive events (VOEs). For adults and children living with SCD, this means unpredictable episodes of excruciating pain due to vaso-occlusion as well as other acute complications—such as acute chest syndrome (ACS), stroke, and infections, which can contribute to early mortality in these patients.

LentiGlobin for SCD adds functional copies of a modified form of the β-globin gene ($\beta^{A\text{-}T87Q}$-globin gene) into a patient's own hematopoietic (blood) stem cells (HSCs). Once patients have the $\beta^{A\text{-}T87Q}$-globin gene, they have the potential to make functional RBCs, with the goal of reducing sickled RBCs, hemolysis, and other complications.

"The latest Group C data from our ongoing Phase 1/2 study show robust production of gene therapy-derived anti-sickling hemoglobin, HbA^{T87Q}, such that patients with six or more months of follow-up after treatment with LentiGlobin for sickle cell disease had median sickle hemoglobin levels reduced to 50 percent or less of total hemoglobin, in the absence of blood transfusions. The potential for gene therapy with LentiGlobin to fundamentally alter the pathophysiology of sickle cell disease was also supported by the normalization of hemolysis markers, increase in total hemoglobin and substantial reduction in vaso-occlusive crises relative to baseline," said David Davidson, M.D., chief medical officer, bluebird bio. "Further insight into these encouraging clinical results was provided by findings from an exploratory assay used to evaluate the expression of HbA^{T87Q}, which demonstrated 70 percent or more of patient red blood cells contain HbA^{T87Q} at nine months after treatment."

Phase 1/2: HGB-206
HGB-206 is an ongoing, Phase 1/2 open-label study designed to evaluate the efficacy and safety of LentiGlobin gene therapy for SCD that includes three treatment cohorts: Groups A, B and C. As of March 7, 2019, 25 patients were enrolled and a total of 13 patients had been treated with LentiGlobin in Group C, with a median post-treatment follow-up of nine months (1.0 – 15.2 months).

"The severity of sickle cell disease is not always recognized, and many people are unaware that individuals are debilitated by the effects of sickle cell disease," said Julie Kanter, M.D., University of

Alabama at Birmingham, Birmingham, Ala. “Group C of the Phase 1/2 HGB-206 study of LentiGlobin now includes multiple patients with at least one year of follow-up, and in these individuals, many with a history of vaso-occlusive crises, their symptoms appear to be resolving. There have been no incidents of acute chest syndrome or serious vaso-occlusive crises reported, and many of their labs are approaching normal.”

Eight of the 13 treated patients in Group C had at least six months of follow-up at the time of the data cutoff. In these patients, production of gene therapy-derived hemoglobin (HbA^{T87Q}) ranged from 4.5–8.8 g/dL and total unsupported hemoglobin (Hb) levels ranged from 10.2–15.0 g/dL at the last study visit.

The median concentration of HbA^{T87Q} continued to increase, accounting for ≥50 percent of total Hb in patients with at least 12 months of follow up (n=4).

No ACS or serious vaso-occlusive crisis (VOC) was reported in patients in Group C at up to 15 months post-treatment with LentiGlobin. In an exploratory analysis, key markers of hemolysis, including reticulocyte counts, lactate dehydrogenase (LDH) and total bilirubin concentration, trended toward normal levels.

As of the data cutoff date, the safety data from all patients in HGB-206 are reflective of underlying SCD, the known side effects of hematopoietic stem cell (HSC) collection and myeloablative conditioning. There have been no serious adverse events (SAEs) related to LentiGlobin for SCD. One mild, non-serious event of hot flush was reported that the investigator considered to be related to LentiGlobin for SCD; it occurred and resolved on the day of drug product infusion and did not require treatment.

Established tools, including high-performance liquid chromatography (HPLC), are used to measure the amount of HbA^{T87Q} in a blood sample. In order to detect HbA^{T87Q} and HbS protein expression at a cellular level, bluebird bio has utilized a new, exploratory assay to demonstrate the pancellular expression of HbA^{T87Q} in patients treated with LentiGlobin. The assay enables detection of HbA^{T87Q} and HbS protein expression at a cellular level. Results from this assay showed that in samples from five patients who were at least nine months post-treatment, on average, at least 70 percent of each patient’s RBCs expressed HbA^{T87Q}.

About LentiGlobin for Sickle Cell Disease

LentiGlobin for sickle cell disease (SCD) is an investigational gene therapy being studied as a potential treatment for SCD. bluebird bio’s clinical development program for LentiGlobin for SCD includes the ongoing Phase 1/2 HGB-206 study.

bluebird bio is conducting a long-term safety and efficacy follow-up study (LTF-303) for people who have participated in bluebird bio-sponsored clinical studies of LentiGlobin for SCD. For more information, visit: https://www.bluebirdbio.com/our-science/clinical-trials.

LentiGlobin for SCD received Orphan Medicinal Product designation from the European Commission for the treatment of SCD.

The U.S. Food and Drug Administration granted Orphan Drug status and Regenerative Medicine Advanced Therapy designation for LentiGlobin for the treatment of SCD.

About bluebird bio, Inc.
bluebird bio is pioneering gene therapy with purpose. From our Cambridge, Mass., headquarters, we're developing gene therapies for severe genetic diseases and cancer, with the goal that people facing potentially fatal conditions with limited treatment options can live their lives fully. Beyond our labs, we're working to positively disrupt the healthcare system to create access, transparency and education so that gene therapy can become available to all those who can benefit.
bluebird bio is a human company powered by human stories. We're putting our care and expertise to work across a spectrum of disorders by researching cerebral adrenoleukodystrophy, sickle cell disease, transfusion-dependent β-thalassemia and multiple myeloma using three gene therapy technologies: gene addition, cell therapy and (megaTAL-enabled) gene editing.
bluebird bio has additional nests in Seattle, Wash.; Durham, N.C.; and Zug, Switzerland. For more information, visit bluebirdbio.com.
Follow bluebird bio on social media: @bluebirdbio, LinkedIn, Instagram and YouTube.
LentiGlobin is a trademark of bluebird bio.

Forward-Looking Statements
This release contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. Any forward-looking statements are based on management's current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. These risks and uncertainties include, but are not limited to: the risk that the efficacy and safety results from our prior and ongoing clinical trials of LentiGlobin for SCD will not continue or be repeated in our ongoing or planned clinical trials of LentiGlobin for SCD; the risk that the current or planned clinical trials of LentiGlobin for SCD will be insufficient to support regulatory submissions or marketing approval in the U.S. and EU; the risk that the production of HbA^{T87Q} may not be sustained over extended periods of time; and the risk that we may not secure adequate pricing or reimbursement to support continued development or commercialization of LentiGlobin for SCD following regulatory approval. For a discussion of other risks and uncertainties, and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the section entitled "Risk Factors" in our most recent Form 10-Q as well as discussions of potential risks, uncertainties, and other important factors in our subsequent filings with the Securities and Exchange Commission. All information in this press release is as of the date of the release, and bluebird bio undertakes no duty to update this information unless required by law.

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Exhibit 99.3

bluebird bio Presents Long-Term Efficacy and Safety Data from Clinical Studies of LentiGlobin® Gene Therapy for Transfusion-Dependent β-Thalassemia (TDT) at 24th European Hematology Association (EHA) Congress

Up to 3.8 years of transfusion independence in Phase 1/2 Northstar (HGB-204) study in patients with TDT who do not have a β^0/β^0 genotype

Four of five evaluable patients achieved transfusion independence in ongoing Phase 3 Northstar-2 (HGB-207) study of patients with TDT who do not have a β^0/β^0 genotype

In patients who were free from transfusions for at least three months total hemoglobin levels were 10.2–13.6 g/dL in the ongoing Phase 3 Northstar-3 (HGB-212) study in patients with TDT who have a β^0/β^0 genotype or IVS-I-110 mutation

CAMBRIDGE, Mass.--(BUSINESS WIRE)—June 14, 2019—bluebird bio, Inc. (Nasdaq: BLUE) announced updated results from the completed Phase 1/2 Northstar (HGB-204) study, and new data from the Phase 3 Northstar-2 (HGB-207) and Phase 3 Northstar-3 (HGB-212) clinical studies of its LentiGlobin™ gene therapy for patients with transfusion-dependent β-thalassemia (TDT), at the 24th European Hematology Association (EHA) Congress in Amsterdam, the Netherlands.

“The maturing data from our clinical studies of LentiGlobin for TDT show that patients across genotypes are able to achieve and maintain transfusion independence with stable production of gene therapy-derived-hemoglobin, HbA^{T87Q}, extending for years,” said David Davidson M.D., chief medical officer, bluebird bio. “In patients who achieve transfusion independence, we have observed decreased liver iron concentration over time and improved markers of erythropoiesis, demonstrating the transformative disease-modifying potential of gene therapy for patients with TDT.”

TDT is a severe genetic disease caused by mutations in the β-globin gene that result in reduced or absent hemoglobin (Hb). In order to survive, people with TDT maintain Hb levels through lifelong chronic blood transfusions. These transfusions carry the risk of progressive multi-organ damage due to unavoidable iron overload.

LentiGlobin for β-thalassemia addresses the underlying genetic cause of TDT by adding functional copies of a modified form of the β-globin gene ($\beta^{A\text{-}T87Q}$-globin gene) into a patient’s own hematopoietic (blood) stem cells (HSCs). This means there is no need for donor HSCs from another person, as is required for allogeneic HSC transplantation (allo-HSCT). Once a patient has the $\beta^{A\text{-}T87Q}$-globin gene, they have the potential to produce HbA^{T87Q}, which is gene therapy-derived-Hb, at levels that eliminate or significantly reduce the need for transfusions.

bluebird bio’s clinical development program for LentiGlobin in TDT includes studies across patient genotypes, including those who do not have a β^0/β^0 genotype as well as those with a β^0/β^0 genotype.

“Patients living with β-thalassemia who have a β^0/β^0 genotype or an IVS-I-110 mutation typically have low levels of endogenous hemoglobin,” said Andreas Kulozik, M.D., Ph.D., Heidelberg University Hospital, Heidelberg, Germany. “Transfusion independence is a goal for the treatment of TDT, regardless of genotype. Early results from the ongoing Phase 3 study in patients with a β^0/β^0 genotype or an IVS-I-110 mutation show gene therapy-derived-hemoglobin significantly contributes to improved total hemoglobin levels.”

Northstar (HGB-204)
The results reported for the completed Phase 1/2 Northstar (HGB-204) study reflect data as of December 13, 2018; of the 18 patients in the study, 10 patients do not have a β^0/β^0 genotype and eight have a β^0/β^0 genotype. All 18 patients have completed the two-year study and enrolled in the long-term follow-up study, LTF-303.

Eight of 10 treated patients who do not have a β^0/β^0 genotype achieved transfusion independence (TI), meaning they had not received a transfusion for at least 12 months or more and maintained a weighted average Hb ≥9 g/dL.

These eight patients had a median weighted average Hb during TI of 10.3 g/dL (min–max: 9.3–13.2 g/dL) and continued to maintain TI for up to 45 months. The patient follow-up period is calculated from infusion of LentiGlobin to the last study visit.

In patients who have a β^0/β^0 genotype, three of the eight achieved TI and maintained a median weighted average Hb ranging from 9.5–10.1 g/dL for a median duration of 16.4 months (min–max: 16.1–20.8 months).
An exploratory assessment was conducted to assess liver iron concentration (LIC) in the 11 patients from the Northstar study who achieved TI. Increased iron levels are a consequence of frequent transfusions. High iron levels can cause organ damage, which many patients with TDT are at risk of and must manage through chelation regimens.
LIC was measured at baseline and then every 12 months after treatment with LentiGlobin. Patients reinitiated iron chelation therapy at a median of 13 months after LentiGlobin infusion (min–max: 2–16 months).
Over time, LIC began to decrease in all 11 patients with the largest decrease observed in patients who had 48 months of data available (n=4). A median 56 percent reduction (min–max: 38–83 percent) was reported in these four patients.
Northstar-2 (HGB-207) Efficacy
As of December 13, 2018, 20 patients who do not have β^0/β^0 genotypes have been treated in the Phase 3 Northstar-2 study. Patient age ranged from 8–34 years, with five pediatric (<12 years) and 15 adolescent/adult (≥12 years) patients.

Four of five evaluable patients achieved TI and maintained a median weighted average Hb of 12.4 g/dL (min–max: 11.5–12.6 g/dL). These four patients continued to maintain TI for a median duration of 13.6 months (min–max: 12–18.2 months) at the time of the data cut off.

Thirteen of 14 patients with at least three months of follow-up were free from transfusions for at least three months. Total Hb levels in these patients ranged from 8.8–13.3 g/dL at the time of the last study visit. HbA^{T87Q} levels were stable over time in patients who were free from transfusions; at Month 6 (n=10) median HbA^{T87Q} was 9.5 g/dL and at Month 12 (n=7) median HbA^{T87Q} was 9.3 g/dL.

An exploratory analysis was conducted with bone marrow from seven patients with 12 months of follow-up after treatment. The samples were evaluated for cellularity and myeloid to erythroid ratio. A low myeloid to erythroid ratio is a key feature of dyserythropoesis, or abnormal bone marrow red blood cell (RBC) production, characteristic of patients with TDT. In these seven patients, all of whom had stopped chronic transfusions, an increase in the myeloid to erythroid ratio was observed, suggesting improvement in RBC production.

Northstar-3 (HGB-212) Efficacy
As of April 12, 2019, 11 patients with TDT and a β^0/β^0 genotype or an IVS-I-110 mutation had been treated in the Phase 3 Northstar-3 study.

The one patient evaluable for TI achieved and maintained it and had a total Hb of 13.6 g/dL at the Month 16 follow-up.

Five patients had stopped transfusions for at least three months and had Hb levels of 10.2–13.6 g/dL at the time of the last study visit (5 – 16 months post-treatment). Of these patients, all of those who reached six months of follow-up (n=4) had HbA^{T87Q} levels of at least 8 g/dL.

LentiGlobin for TDT Safety
Non-serious adverse events (AEs) observed during clinical studies that were attributed to LentiGlobin for TDT were hot flush, dyspnoea, abdominal pain, pain in extremities and non-cardiac chest pain. One serious adverse event (SAE) of thrombocytopenia was considered possibly related to LentiGlobin for TDT.

Additional AEs observed in clinical studies were consistent with the known side effects of HSC collection and bone marrow ablation with busulfan, including SAEs of veno-occlusive disease.

As of the data cut off dates stated above, a total of 49 pediatric, adolescent and adult patients with TDT and a non-β^0/β^0 or β^0/β^0 genotype, including patients with IVS-I-110 mutations, have been treated with LentiGlobin for TDT in the Northstar, Northstar-2 and Northstar-3 studies.

About LentiGlobin for β-Thalassemia
The European Commission (EC) granted conditional marketing authorization for LentiGlobin for TDT, to be marketed as ZYNTEGLO® (autologous CD34+ cells encoding $\beta^{A\text{-}T87Q}$-globin gene) gene therapy, for patients 12 years and older with TDT who do not have a β^0/β^0 genotype, for whom hematopoietic stem cell (HSC) transplantation is appropriate, but a human leukocyte antigen (HLA)-matched related HSC donor is not available.

ZYNTEGLO adds functional copies of a modified form of the β-globin gene ($\beta^{A\text{-}T87Q}$-globin gene) into a patient's own hematopoietic (blood) stem cells (HSCs). Once a patient has the $\beta^{A\text{-}T87Q}$-globin gene, they have the potential to produce HbA^{T87Q}, which is gene therapy-derived-hemoglobin, at levels that

eliminate or significantly reduce the need for transfusions. Upon engraftment and achievement of transfusion independence, effects of ZYNTEGLO are expected to be lifelong.

The EMA previously granted Priority Medicines (PRIME) eligibility and Orphan Medicinal Product designation to ZYNTEGLO for the treatment of TDT. ZYNTEGLO is also part of the EMA's Adaptive Pathways pilot program, which is part of the EMA's effort to improve timely access for patients to new medicines.

The U.S. Food and Drug Administration (FDA) also granted ZYNTEGLO Orphan Drug status and Breakthrough Therapy designation for the treatment of TDT.
LentiGlobin for TDT continues to be evaluated in the ongoing Phase 3 Northstar-2 and Northstar-3 studies and the long-term follow-up study LTF-303. For more information about the ongoing clinical studies, visit www.northstarclinicalstudies.com or clinicaltrials.gov and use identifier NCT01745120 for Northstar (HGB-204), NCT02906202 for Northstar-2 (HGB-207), NCT03207009 for Northstar-3 (HGB-212) and NCT02633943 for LTF-303.

About bluebird bio, Inc.

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bluebird bio is a human company powered by human stories. We're putting our care and expertise to work across a spectrum of disorders by researching cerebral adrenoleukodystrophy, sickle cell disease, transfusion-dependent β-thalassemia and multiple myeloma using three gene therapy technologies: gene addition, cell therapy and (megaTAL-enabled) gene editing.
bluebird bio has additional nests in Seattle, Wash.; Durham, N.C.; and Zug, Switzerland. For more information, visit bluebirdbio.com.
Follow bluebird bio on social media: @bluebirdbio, LinkedIn, Instagram and YouTube.
ZYNTEGLO and LentiGlobin are trademarks of bluebird bio.
The full common name for ZYNTEGLO: A genetically modified autologous CD34+ cell enriched population that contains hematopoietic stem cells transduced with lentiviral vector encoding the $\beta^{A\text{-}T87Q}$-globin gene.

Forward-Looking Statements

This release contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995. Any forward-looking statements are based on management's current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. These risks and uncertainties include, but are not limited to: the risk that the efficacy

and safety results from our prior and ongoing clinical trials of LentiGlobin for TDT will not continue or be repeated in our ongoing or planned clinical trials of LentiGlobin for TDT; the risk that the current or planned clinical trials of LentiGlobin for TDT will be insufficient to support future regulatory submissions in the U.S. and EU or additional marketing authorizations; the risk that the production of HbA^{T87Q} may not be sustained over extended periods of time; and the risk that we may not secure adequate pricing or reimbursement to support continued development or commercialization of LentiGlobin for TDT. For a discussion of other risks and uncertainties, and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the section entitled "Risk Factors" in our most recent Form 10-Q as well as discussions of potential risks, uncertainties and other important factors in our subsequent filings with the Securities and Exchange Commission. All information in this press release is as of the date of the release, and bluebird bio undertakes no duty to update this information unless required by law.

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