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): December 9, 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 (Par Value $0.01)	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 December 9, 2019, bluebird bio, Inc. ("bluebird") conducted an investor webcast presenting updated clinical data for the LentiGlobin product candidate in the treatment of patients with transfusion-dependent β-thalassemia (HGB-204, HGB-207, and HGB-212), for the LentiGlobin product candidate in the treatment of patients with sickle cell disease (HGB-206), and for the bb21217 product candidate in the treatment of patients with relapsed-refractory multiple myeloma (CRB-402), as presented at the 61st Annual Meeting of the American Society of Hematology ("ASH") in Orlando, Florida, as well as clinical data from KarMMa, a pivotal phase 2 study of idecabtagene vicleucel for the treatment of patients with relapsed and refractory multiple myeloma. A copy of the presentation is being furnished as Exhibit 99.1 to this Current Report on Form 8-K.

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 December 9, 2019, bluebird issued a press release announcing data presented at ASH of new data from ongoing studies of LentiGlobin gene therapy for β-thalassemia (betibeglogene autotemcel) in pediatric, adolescent and adult patients who have transfusion-dependent β-thalassemia (TDT),

Also on December 9, 2019, bluebird issued a press release announcing data presented at ASH from the ongoing Phase 1 clinical study of bb21217 (CRB-402) of its investigational next-generation anti-BCMA CAR T cell therapy being studied in patients with relapsed-refractory multiple myeloma.

The full text of bluebird's press releases regarding these announcements are filed as Exhibits 99.2 and 99.3 to this Current Report on Form 8-K and are 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 December 9, 2019.
99.2	Press release issued by bluebird bio, Inc. on December 9, 2019 regarding data from HGB-204, HGB-207 and HGB-212 studies.
99.3	Press release issued by bluebird bio, Inc. on December 9, 2019 regarding data from CRB-402 clinical study.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

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 thereunto duly authorized.

bluebird bio, Inc.

Date: December 10, 2019

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

Exhibit 99.1

bluebirdbio®
recode for life™

ASH 2019

december 9, 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,” “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 initiate or complete, clinical studies, the timing or likelihood of regulatory filings and approvals or the requirements that may be imposed, 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.

LET'S RECODE THE SYSTEM

today's agenda

welcome	Nick Leschly *chief bluebird*
LentiGlobin for TDT data	Dave Davidson, M.D. *chief medical officer*
LentiGlobin for SCD data	Mohammed Asmal, M.D., Ph.D. *vice president, head of SGD clinical research*
bb21217 and KarMMa topline data	Dave Davidson, M.D. *chief medical officer*
Q&A	Chip Baird, *chief financial officer* Philip Gregory, D. Phil., *chief scientific officer* Alison Finger, *chief commercial officer*

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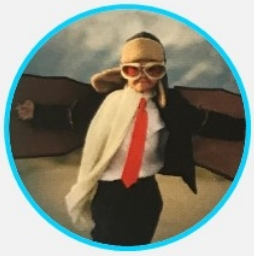
WE **RECODE** FOR LIFE

RADICAL CARE

We care in a way that's intense and truly sets us apart.

THIS IS PERSONAL

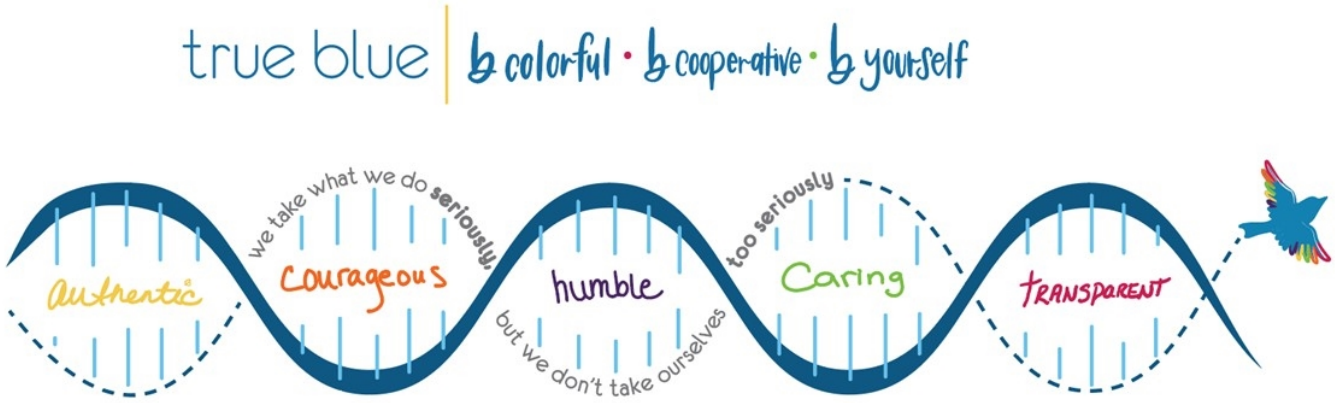
Gene therapy is about saving lives one person at a time. And we are, each of us, personally all in.



PIONEERS WITH PURPOSE

We're exploring new frontiers for the sake of patients.

We live by our non-negotiables



Key questions for today

LentiGlobin TDT	• Do the updated data in patients with non-β^0/β^0 genotypes reinforce the potential for patients to achieve and maintain transfusion independence (TI)? • Do the emerging data in patients with β^0/β^0 genotypes suggest that these patients may achieve TI?
LentiGlobin SCD	• With more patients and more follow up, are we continuing to see profound impact on important clinical manifestations of disease (VOEs, hemolytic anemia)? • Do the laboratory tools we have developed conclusively demonstrate that LentiGlobin for SCD changes the fundamental pathophysiology of the sickle RBC?
bb21217	• Does enriching 21217 drug product for memory-like t cells translate to greater T cell persistence and longer durations of response?
ide-cel KarMMa	• Are KarMMa data supportive of ide-cel as a meaningful advance for patients and as a potential new standard of care for refractory multiple myeloma? • Is the program on track for 1H:2020 filing?

Transfusion-Dependent ß-Thalassemia (TDT)

- A severe, progressive, genetic disease that leads to severe anemia, lifelong transfusion dependence, unavoidable iron overload, serious comorbidities, and a shortened lifespan.
- Global prevalence estimated at ~288,000[1]
- The U.S. prevalence of beta-thalassemia major is estimated at least 1,000 people[2,3]
- European prevalence is variable by country ranging from <1,000[4] patients in nonendemic countries, to ~6,500[5] patients in endemic countries

program overview

- EU approval granted June 2019
- US rolling BLA submission to begin by YE 2019
- Studies ongoing:
  - Northstar-2 (HGB-207)
  - Northstar-3 (HGB-212)
  - Long-term follow-up: LTF-303

[1]Biffi A. Gene Therapy as a Curative Option for beta-Thalassemia. *N Engl J Med*. 2018;378(16):1551-1552.
[2]SayaniFA, Kwiatkowski JL. Increasing prevalence of thalassemia in America: implications for primary care. Ann Med. 2015;47(7):592-604.
[3]Centers for Disease Control and Prevention. Living with thalassemia. 2018; https://www.cdc.gov/features/international-thalassemia/index.html. Accessed May 11, 2018.
[4]CarioH, StahnkeK, Sander S, KohneE. Epidemiological situation and treatment of patients with thalassemia major in Germany: results of the German multicenter ß-thalassemia study. Ann Hematol. 2000;79(1):7-12.
[5]AngelucciE, AntmenAB, LosiS, Burrows N, BartiromoC, Hu XH. Direct medical care costs associated with ß-Thalassemia care in Italy. Blood. 2017;130(Suppl 1):92-5599.

Completed studies of LentiGlobin for TDT reinforce long term durability of clinical outcomes

NORTHSTAR STUDY	HGB-204 *Complete*	• Up to 5 years follow-up with stable HbA^{T87Q} and total Hb • 8/10 non-β^0/β^0; 3/8 β^0/β^0 remain TI as of data cut-off • Reduction in liver iron content; cardiac iron remains stable in normal range as of data cut-off
HGB-205	HGB-205 *Complete*	• Stable HbA^{T87Q} and total Hb at up to 5+ years follow-up • 3/4 non-β^0/β^0 remain TI as of data cut-off • Substantial improvement in underlying dyserythropoiesis
NORTHSTAR-2 STUDY	HGB-207 *non-β^0/β^0 genotypes*	• 21/23 patients treated • 9/10 patients achieved transfusion independence (TI) • Total unsupported Hb is near-normal in most patients as of data cut-off
NORTHSTAR-3 STUDY	HGB-212 *β^0/β^0 genotype or IVS-I-110 mutations*	• 13/18 patients treated • 2/2 patients achieved transfusion independence

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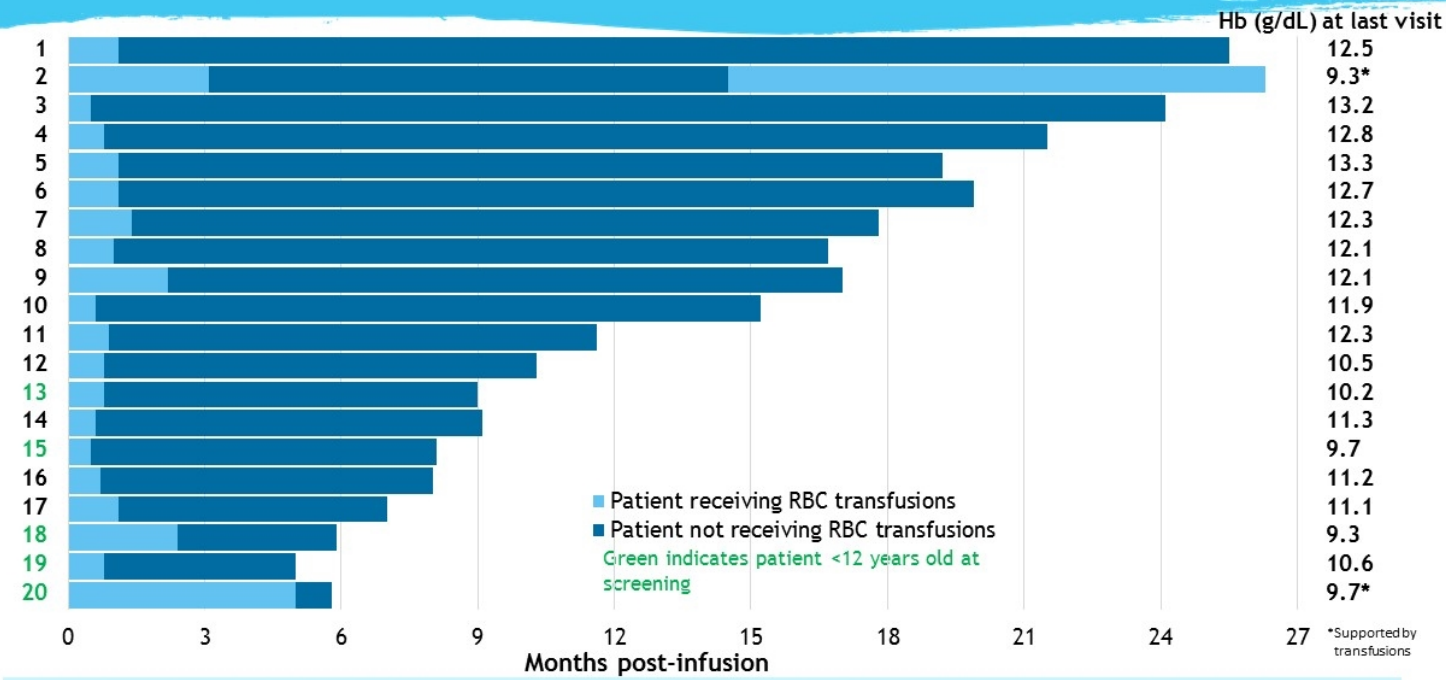
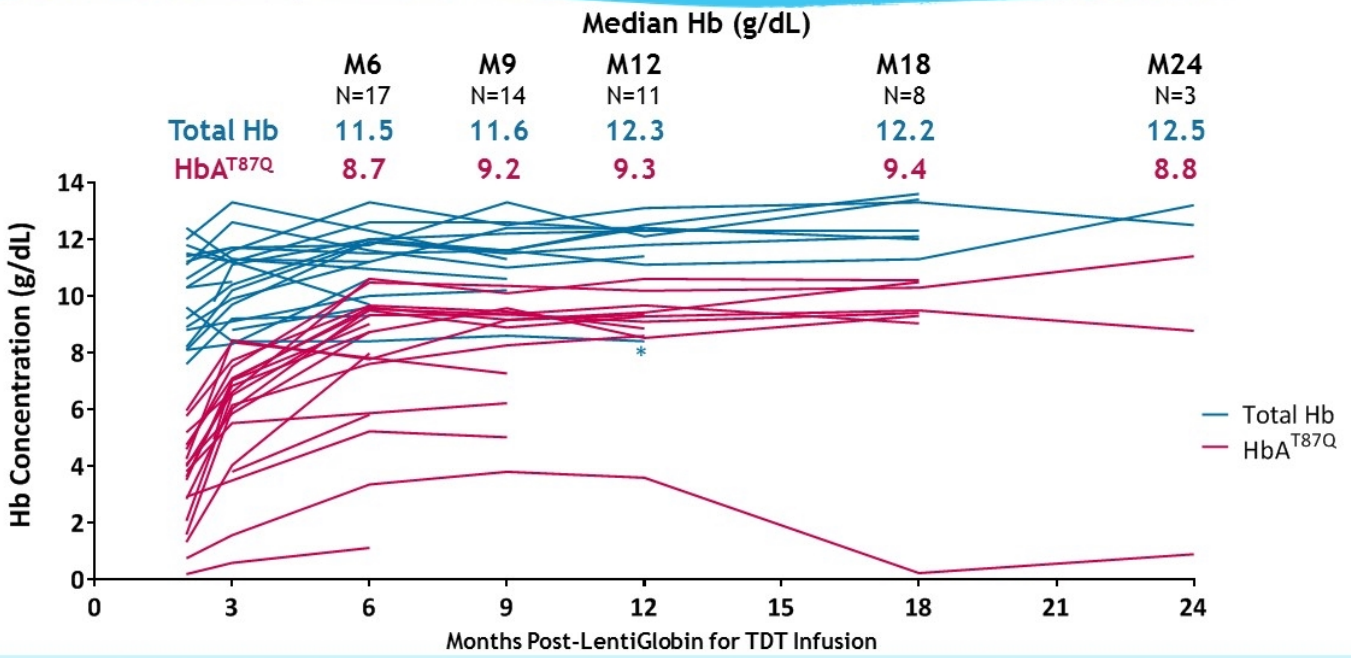
HGB-207: 90% (18/20) of patients with > 3 months follow-up are off pRBC transfusions



The safety profile of LentiGlobin for TDT remains generally consistent with that of myeloablative busulfan conditioning.

Data as of 12 June 2019

HGB-207: stable total Hb and gene therapy-derived HbA^{T87Q} in the majority of patients

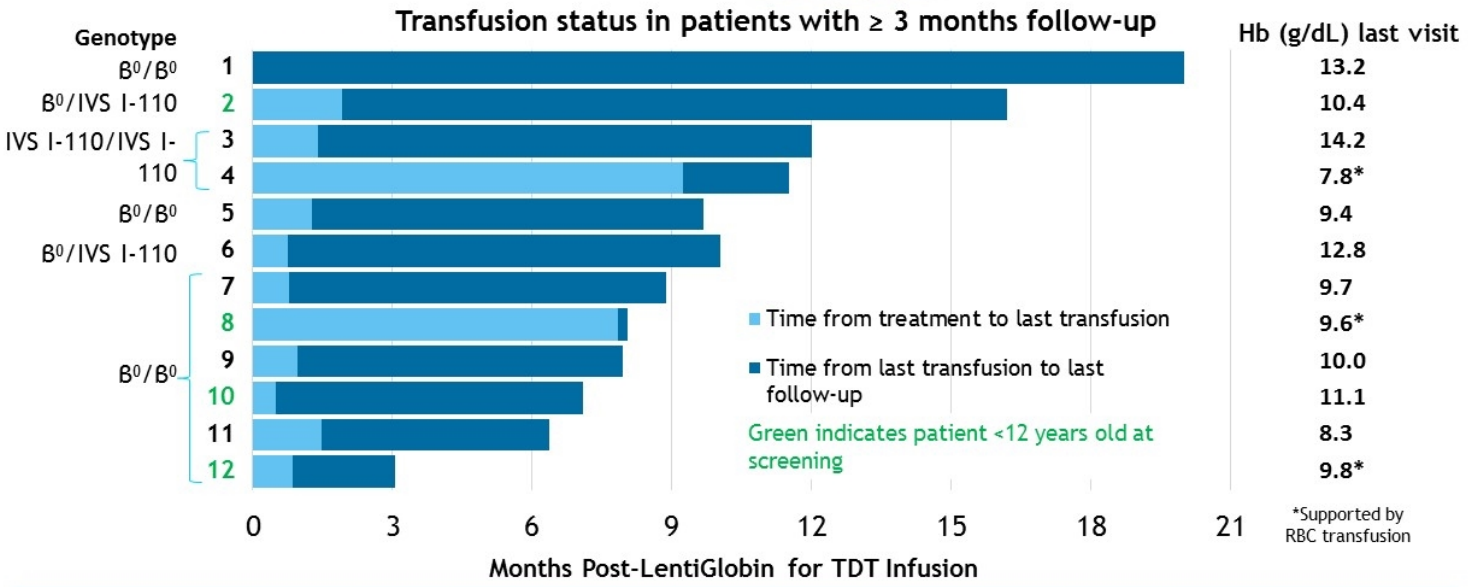


HGB-207 utilized a range of manufacturing process conditions as the commercial manufacturing process was defined

*Last Hb before patient 2 restarted red blood cell transfusions. Hb, hemoglobin. Median total Hb values include Patient 2 who was on transfusions

Data as of 12 June 2019

HGB-212: 9/11 patients with ≥ 6 months follow-up have been off transfusions for ≥ 3 months

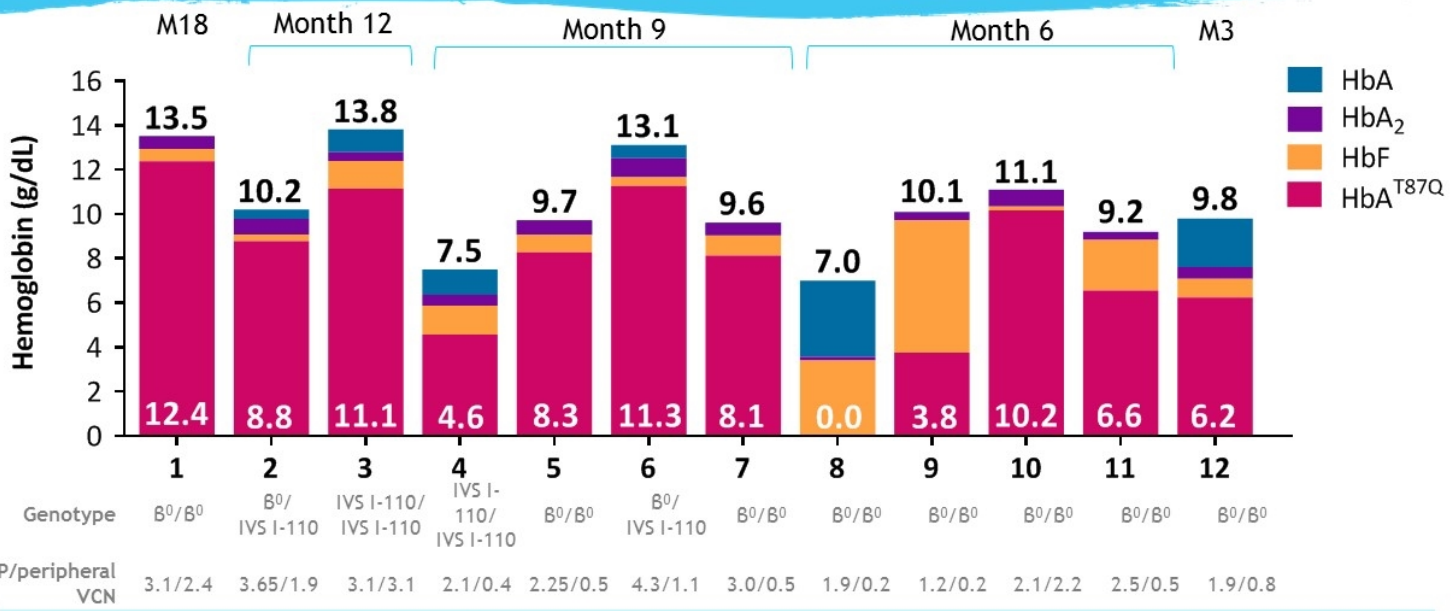


Patients 1 and 2 achieved and maintained transfusion independence Weighted average Hb ≥ 9 g/dL without RBC transfusions for ≥ 12 months

Hb, hemoglobin; RBC, red blood cell

Data as of 30 September 2019

HGB-212: HbA^{T87Q} supports target total Hb in most patients with minimal endogenous HbA ≥ 3 months after treatment



HGB-212 utilized a range of manufacturing process conditions as the commercial manufacturing process was defined

The safety profile of LentiGlobin for TDT remains generally consistent with that of myeloablative busulfan conditioning

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

Data as of 30 September 2019

50+ patients treated across program for LentiGlobin in TDT

Completed HGB-204 and HGB-205 studies with up to 5+ years of data reinforce durability of treatment

90% of evaluable patients who do not have a β^0/β^0 genotype achieved TI in HGB-207 study

9 of 11 patients with at least 6 months of follow-up in HGB-212 did not receive a transfusion for more than 3 months as of last follow-up

Safety profile of LentiGlobin for TDT treatment is consistent with that of busulfan conditioning

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Sickle Cell Disease (SCD)

- A serious, progressive, unpredictable, and debilitating genetic disease caused by abnormal sickle hemoglobin
- Results in chronic hemolytic anemia, repeated painful vaso-occlusive events and persistent vasculopathy that frequently leads to early morbidity and mortality
- Global annual birth incidence ~ 300,000 – 400,000[1,2]
- U.S. prevalence estimated at 72,000 – 100,000[3,4]
- Mean age of death in the U.S. is 44 years[5]

program overview

- Plan to pursue development path based on hematological primary endpoint
- Phase 3 HGB-210 study to be open and enrolling patients by early 2020
- HGB-206 target enrollment achieved

[1]Kato GJ, Piel FB, Reid CD, et al. Sickle cell disease. *Nat Rev Dis Primers*. 2018;4:18010.
[2]Piel FB, Patil AP, Howes RE, et al. Global epidemiology of sickle haemoglobin in neonates: a contemporary geostatistical model-based map and population estimates. *Lancet*. 2013;381(9861):142-151.
[3]Hassell KL. Population estimates of sickle cell disease in the U.S. *Am J Prev Med*. 2010;38(4 Suppl):S512-521.
[4]CDC Data and Statistics on Sickle Cell Disease. https://www.cdc.gov/ncbddd/sicklecell/data.html
[5]Paulukonis et al, California's Sickle Cell Data Collection Cohort, 2005-2015* ASH 2017*

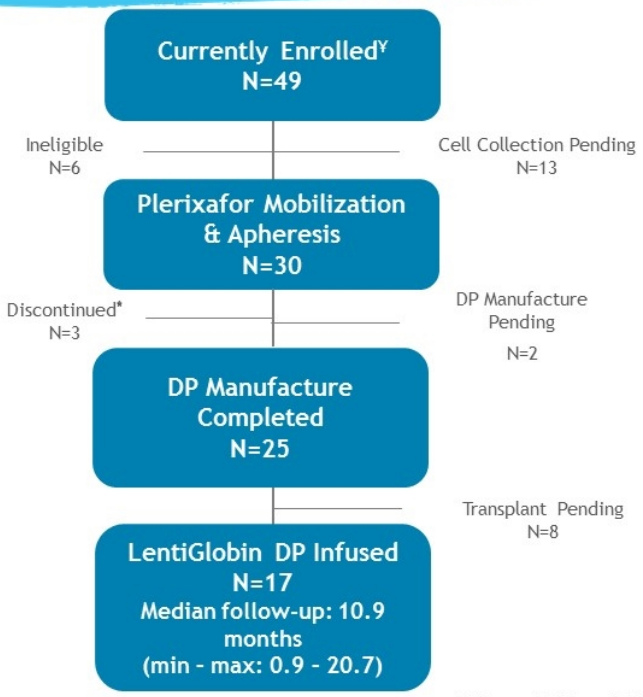
Expanding development program to evaluate LentiGlobin across SCD patient types and ages

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

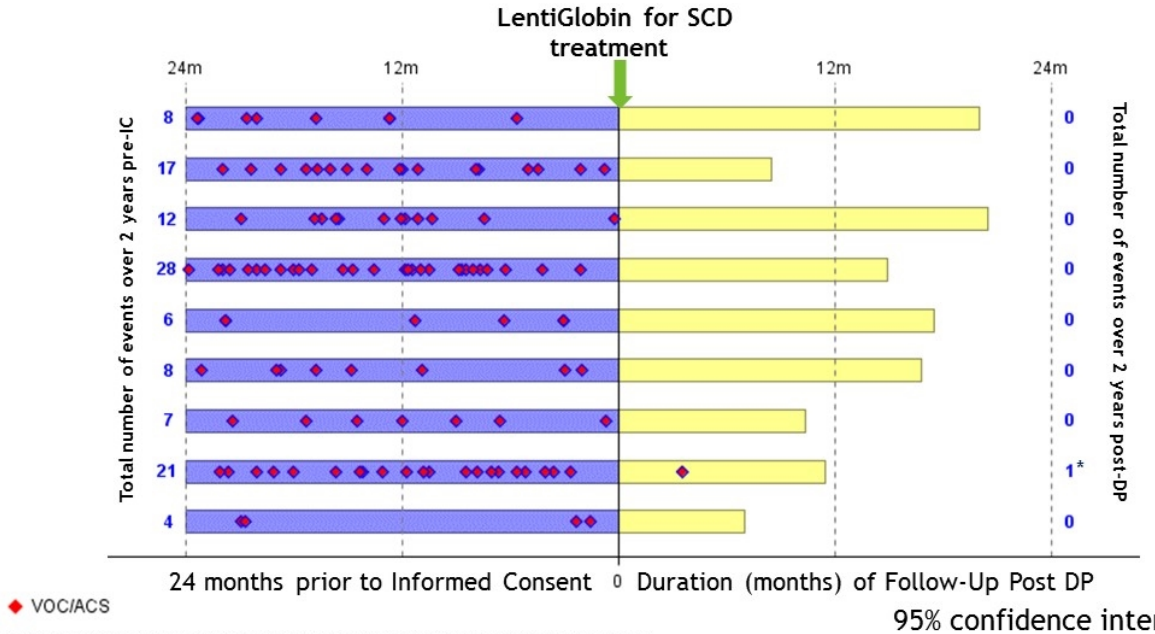


[Y] Currently active, not recruiting

*1 withdrew consent, 1 discontinued due to investigator discretion, 1 mobilization failure; DP, drug product

Data as of 26 August 2019

99% reduction in annualized rate of VOC + ACS in HGB-206 Group C patients with history of VOCs and ACS who had ≥ 6 months of follow-up



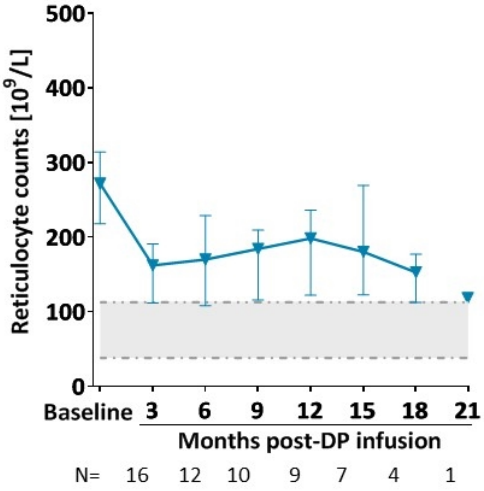
95% confidence interval: 92.5-100%

*As previously reported, 1 non-serious Grade 2 VOC was observed in 1 patient ~3.5 months post-LentiGlobin treatment
Investigator-reported adverse events of VOC or ACS are shown; *Patients with ≥ 4 VOC/ACS at baseline before Informed Consent and with ~ ≥ 6 months of follow-up post-DP infusion
ACS, acute chest syndrome; VOCs, vaso-occlusive crises; DP, drug product

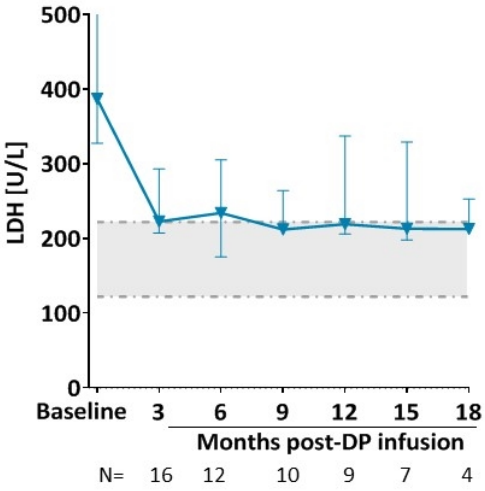
Data as of 26 August 2019

Improving key markers of hemolysis in HGB-206 Group C patients following treatment with LentiGlobin for SCD

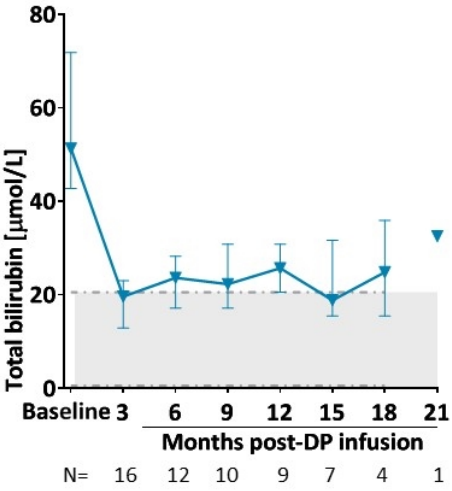
Reticulocyte Counts



Lactate Dehydrogenase



Total Bilirubin

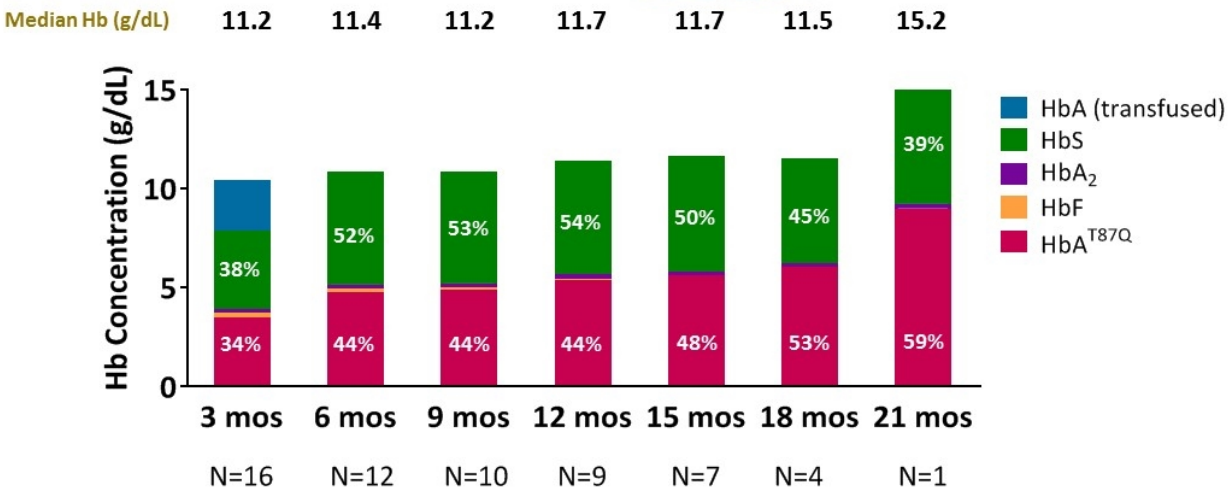


Median (Q1, Q3) depicted; Dot-dash lines denote lower and upper limits of normal values; *Number of patients with data available; †Total bilirubin at last follow-up remains > 2-fold lower than at screening

DP, drug product; LDH, lactate dehydrogenase

Data as of 26 August 2019

HGB-206 Group C patients at 6 months post-treatment produced consistent median levels of anti-sickling hemoglobin ranging from 44% - 59% (Month 6-21)



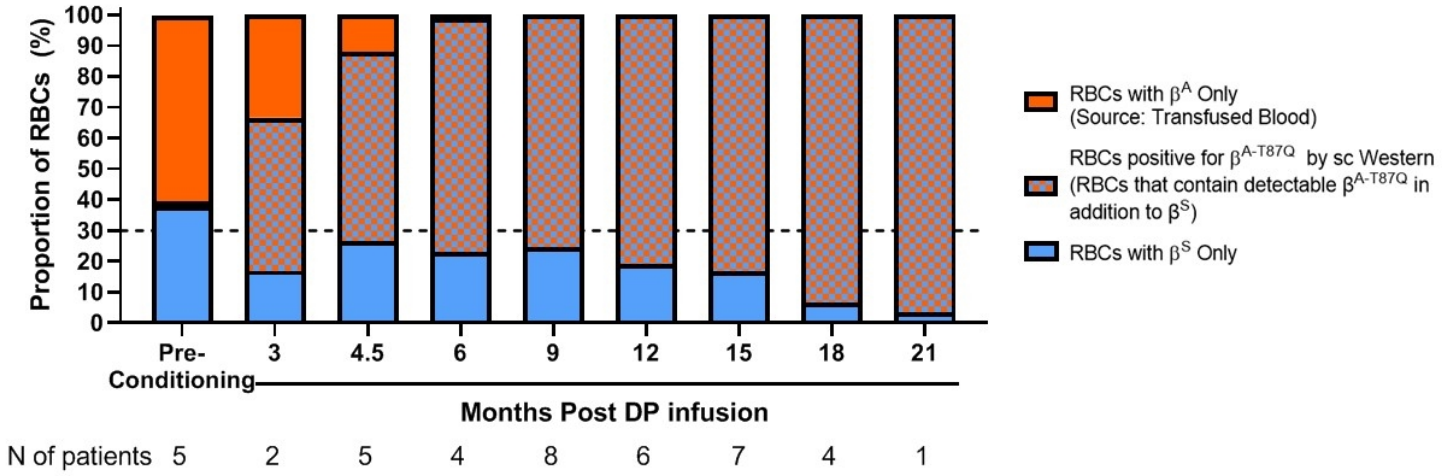
- Median HbS ≤ 60% at ≥ 6 months post-LentiGlobin for SCD treatment
- Total Hb and HbA^{T87Q} ranged from 9.3 - 15.2 g/dL and 2.7 - 9.0 g/dL, respectively, at last visit in patients with ≥ 6 months of follow-up

% represents median Hb fraction as % of total Hb; Hb, hemoglobin

Data as of 26 August 2019

On average, ≥ 70% of RBCs from patients treated with LentiGlobin for SCD contain $\beta^{A\text{-}T87Q}$ by month 6

- Exploratory single RBC western assay performed on samples from 12 patients in Group C



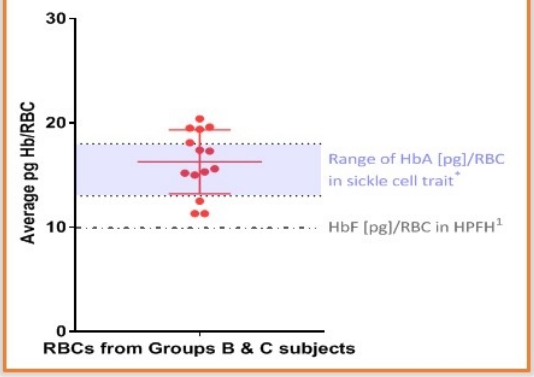
Mean is depicted - if N=1, data show technical replicates; ˚Pre-conditioning sample does not contain any $\beta^{A\text{-}T87Q}$, signal is due to error rate of multiples
DP, drug product; RBCs, red blood cells; sc, single cell

Data as of 26 August 2019

Exploratory assay: high concentrations of T87Q achieved at the cellular level

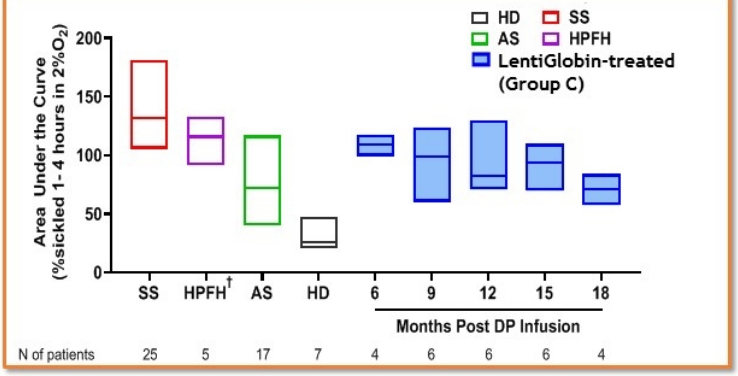
Propensity to sickle decreases over time post-gene therapy with LentiGlobin for SCD; Group C similar to trait

HbA^{T87Q} pg/RBC comparable to HbA pg/RBC in trait & HbF pg/RBC in HPFH



Average pg Hb/RBC = (% HbA^{T87Q} of total Hb/% RBCs containing $\beta^{A\text{-}T87Q}$) x MCH

% sickling for LentiGlobin-treated patients with ≥ 6M follow-up Group C only



*Calculated using 50% HbA/RBC for the lower end of the range and 60% HbA/RBC for the upper end of the range ¨Group C only; †HbF contribution to total Hb in these samples ranged from 28% - 42%

1. Steinberg MH et al., Blood. 2014;123(4):481-5.

DP, drug product; Hb, hemoglobin; HPFH, hereditary persistence of fetal hemoglobin; MCH, mean corpuscular hemoglobin; RBC, red blood cells AS, sickle cell trait; HD, healthy donor; SS, sickle mutation on both *HBB* alleles

Data as of 26 August 2019

HGB-206 Group C: safety profile post-DP infusion generally consistent with myeloablative single-agent busulfan conditioning

Non-hematologic Grade ≥ 3 AEs *Post-DP infusion in ≥ 2 patients**	**N = 17** n (%)
Febrile neutropenia	10 (58.8)
Stomatitis	9 (52.9)
Increased blood bilirubin	3 (17.6)
Upper abdominal pain	2 (11.8)
Increased alanine aminotransferase	2 (11.8)
Increased aspartate aminotransferase	2 (11.8)
Nausea	2 (11.8)
Premature menopause	2 (11.8)
Serious AEs *Post-DP infusion in ≥ 2 patients*	**N = 17** n (%)
Nausea	2 (11.8)
Vomiting	2 (11.8)

- Safety profile post-DP infusion is generally consistent with myeloablative single-agent busulfan conditioning
- No DP-related adverse events
- No cases of veno-occlusive liver disease
- No graft failure or deaths reported
- No vector-mediated RCL
- No evidence of clonal dominance
- No further cases of MDS have been observed across studies of LentiGlobin†▪

*Hematologic AEs commonly observed post-transplantation have been excluded

†As of June 2019 (HGB-205); 12 Jun 2019 (HGB-204, HGB-207), and 30 Sep 2019 (HGB-212)

▪One patient in Group A was reported to have MDS at ASH 2018. There was no evidence of LVV-mediated oncogenesis and the MDS SAE was considered unlikely related to LentiGlobin gene therapy.

AE, adverse event; DP, drug product; RCL, replication competent lentivirus

Notable impact on underlying pathophysiology of SCD

- 99% reduction in annualized rate of VOC + ACS in Group C patients with history of VOCs and ACS who had ≥ 6 months of follow-up, with no reports of ACS or serious VOCs at up to 21 months post-treatment
- Continued improvement in key markers of hemolysis in Group C patients as of the data cut-off date
- Group C patients at 6 months post-treatment produced consistent median levels of anti-sickling hemoglobin ranging from 44% - 59%
- Continue to pursue an accelerated development path based on hematological primary endpoint

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ACS, acute chest syndrome; VOCs, vaso-occlusive crises

multiple myeloma

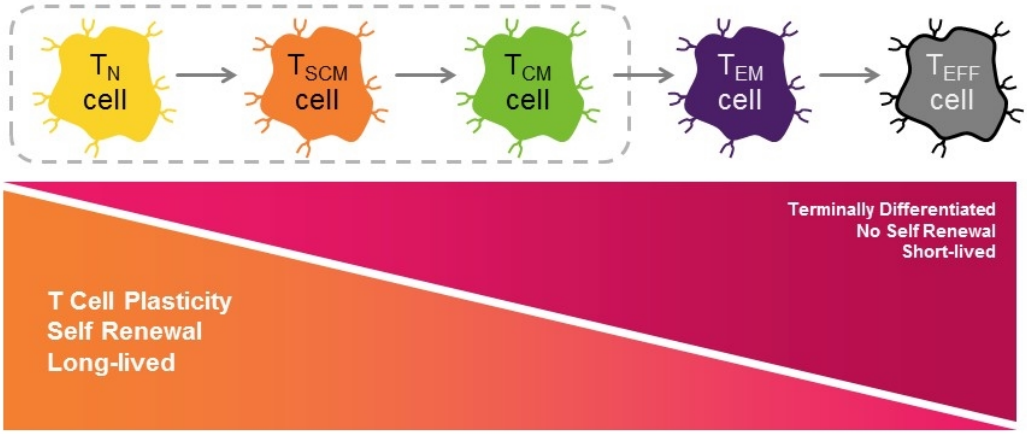
- An incurable type of blood cancer that arises from antibody producing cells in the bone marrow, resulting in anemia, kidney failure, infections and skeletal fractures.
- Second most common hematologic cancer[1,2]
- In 2018, MM was diagnosed in nearly 160,000 patients worldwide and over 31,000 patients in the US. It is estimated that over 130,000 patients in the US are living with this disease.

BCMA program overview

- **ide-cel (bb2121):**
  - U.S. BLA submission planned for 1H:2020
  - KarMMa-2 and KarMMa-3 studies in earlier lines of therapy open and enrolling; Phase 1 study in newly-diagnosed multiple myeloma set for 2019
- **bb21217 CRB-402** Phase 1 study underway

[1]NCI SEER. https://seer.cancer.gov/statfacts/html/mulmy.html. Accessed June 5, 2019.
[2] Bray F, et al. *CA Cancer J Clin*. 2018;68(6):394-424

bb21217: PI3K inhibition during manufacturing drives increase in long-lived, memory-like T cells



Hypothesis: Increasing long-lived, memory-like T Cell subsets in the drug product may result in enhanced persistence of functional anti-BCMA CAR T cells in vivo

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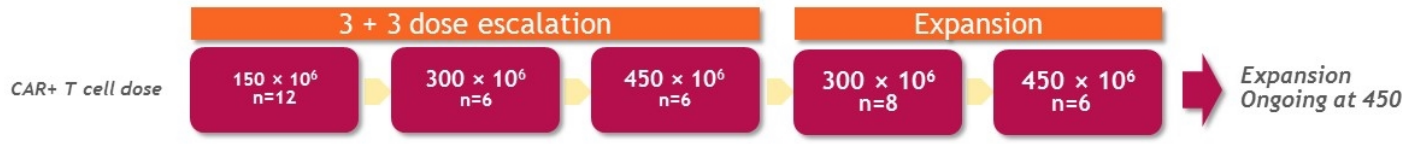
Phase 1 dose-escalation study in heavily pretreated and refractory patient population continues to enroll



N ≈ 74

- R/R MM
- ≥3 prior regimens
- Prior IMiD and PI required
- In escalation only ≥50% BCMA expression required
- In expansion only αCD38 exposure and refractory to last line required

- **Primary endpoints:** AEs, DLTs
- **Other endpoints:** Response[c], PFS, OS, MRD, CAR+ T cell expansion and persistence

Manufacturing Success Rate 100%*

*3 patients required
1 re-manufacturing run

BCMA, B-cell maturation antigen; IMiD, immunomodulatory imide drugs; MRD, minimal residual disease; OS, overall survival; PFS, progression-free survival; PI, proteasome inhibitor; R/R, relapsed/refractory. [c]Per International Myeloma Working Group Criteria.

Data as of 4 September 2019

Baseline patient characteristics and treatment history

Characteristic	bb21217-Treated (N=38)
Median (min, max) age, y	62 (33, 74)
Male, n (%)	21 (55)
Time since initial diagnosis, y	
Median (min, max)	5.5 (1.0, 13.5)
ECOG PS, n (%)	
0	12 (32)
1	24 (63)
2	2 (5)
ISS stage[a], n (%)	
I	11 (29)
II	7 (18)
III	10 (26)
Unavailable	10 (26)
High-risk cytogenetics, n (%)	
del(17p), t(4;14), t(14;16)	13 (34)
Unknown	1 (3)

Characteristic		bb21217-Treated (N=38)	
Median (min, max) no. prior regimens[b]		6 (3, 17)	
Prior autologous SCT, n (%)			
0		7 (18)	
1		22 (58)	
>1		9 (24)	
Prior therapies, n (%)		Exposed	Refractory
IMiD agent	Any	38 (100)	30 (79)
	Lenalidomide	38 (100)	30 (79)
	Pomalidomide	35 (92)	22 (58)
PI	Any	38 (100)	33 (89)
	Bortezomib	36 (95)	21 (55)
	Carfilzomib	32 (84)	25 (66)
αCD38 antibodies	Any	36 (95)	29 (76)
	Daratumumab	35 (92)	28 (74)
Cumulative	PI/IMiD	38 (100)	29 (76)
	PI/IMiD/αCD38 antibodies	36 (95)	24 (63)

ECOG PS, Eastern Cooperative Oncology Groups performance status; IMiD, immunomodulatory imide drugs; ISS, International Staging System; mAb, monoclonal antibody; PI, proteasome inhibitor; SCT, stem cell transplantation.
[b]Number of antimyeloma regimens, including autologous SCT.

Data as of 4 September 2019

Safety profile consistent with CAR T experience

Grade 3/4 AEs in >2 Patients[a], n (%)	Grade 3/4 (N=38)
Neutropenia	31 (82)
Leukopenia	21 (55)
Thrombocytopenia	21 (55)
Anemia	19 (50)
Lymphopenia	13 (34)
Hypophosphatemia	8 (21)
Infection[b]	7 (18)
Hyponatremia	5 (13)
Febrile neutropenia	4 (11)

	N	Grade, n (%) 1	2	3	4	5	Total all grades
CRS							
150×10^6	12	4 (33)	3 (25)	1 (8)	0	0	8 (67)
300×10^6	14	4 (29)	3 (21)	0	0	0	7 (50)
450×10^6	12	4 (33)	5 (42)	0	0	1 (8)	10 (83)
Neurotoxicity							
150×10^6	12	1 (8)	1 (8)	0	1 (8)	0	3 (25)
300×10^6	14	1 (7)	2 (14)	1 (7)	0	0	4 (29)
450×10^6	12	1 (8)	0	1 (8)	0	0	2 (17)

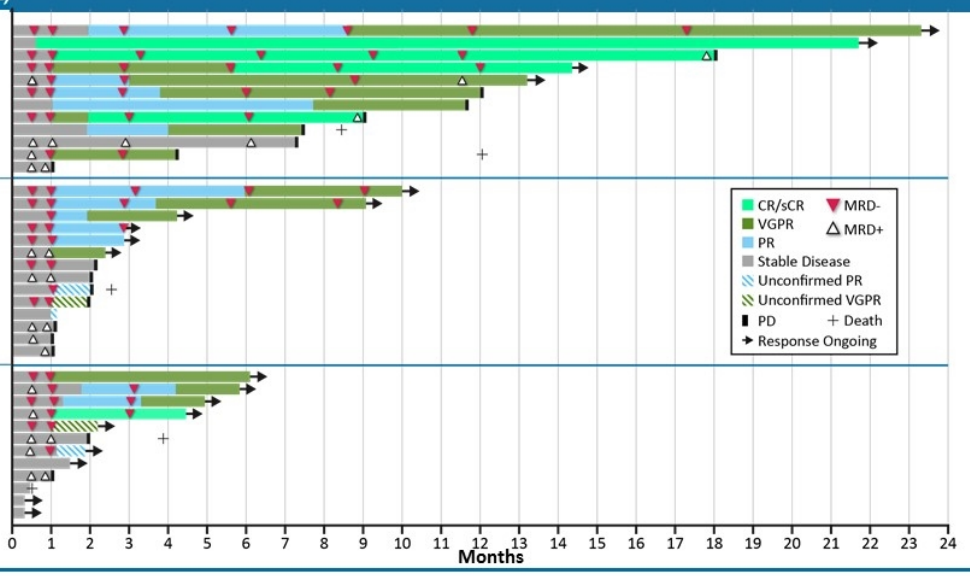
- CRS[c] occurred in 25 patients (66%)
  - Median (min, max) time to onset was 3 d (1, 20)
  - Generally adequately managed with tocilizumab (n=10) and tocilizumab plus corticosteroids (n=4)
  - 1 fatal CRS event associated with grade 3 neurotoxicity at the 450×10^6 dose occurred after 15 days of follow-up
- Neurotoxicity[d] of grade 3 or higher occurred in 3 patients
  - 2 grade 3 events and 1 previously reported grade 4 event
  - Median (min, max) time to onset of neurotoxicity was 7 d (3, 24)
- 7 grade 3/4 infections reported
- 19 patients (50%) experienced ≥1 SAE

AE, adverse event, SAE, serious AE, CMV, cytomegalovirus
[a]AEs and SAEs after first documented progression are excluded
[b]Includes SOC infections and infestations, one case each of anal abscess, bacteraemia, CMV colitis, device related infection, escherichiabacteraemia, pneumococcal bacteraemia, pneumococcal sepsis and pneumonia; CRS, cytokine release syndrome;. [c]CRSuniformly graded according to Lee et al., *Blood*2014;124:188-195 occurring after bb21217 infusion and before disease progression. [d]Events selected as CAR T neurotoxicity on the case report form occurring within 90 days after bb21217 infusion.

Data as of 4 September 2019

To date, no progression in patients with confirmed response at the 300 x 10^6 and 450 x 10^6 dose cohorts; mDOR of 11.1 months at 150 x 10^6 dose

CAR+ T Cells	N	Median follow up (min, max)
150 × 10^6	12	17.6 mo (12, 23)
300 × 10^6	14	4.0 mo (2, 10)
450 × 10^6	12[a]	3.3 mo (<1, 6)



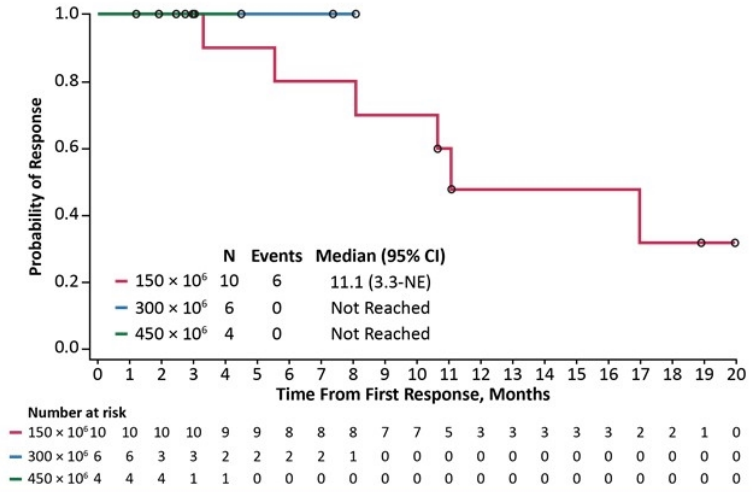
CR, complete response; MRD, minimal residual disease; PD, progressive disease; sCR, stringent complete response; VGPR, very good partial response.
[a] One patient ongoing at the time of the data extraction missed their 2-month visit and another was in VGPR but is reported as a PR owing to a missed assessment.

Data as of 4 September 2019

Confirmed responses across dose cohorts

CAR+ T Cells:	150 × 10^6 (n=12)	300 × 10^6 (n=14)	450 × 10^6 (n=7)
Median follow-up (min, max)	17.6 mo (12, 23)	4.0 mo (2, 10)	3.3 mo (<1, 6)
Confirmed response[a], n (%)			
sCR/CR	4 (33)	0	1 (14)
VGPR	6 (50)	4 (29)	2 (29)
PR	0	2 (14)	1 (14)
Total	**10 (83)**	**6 (43)**	**4 (57)**
Median time to first response (min, max), mo	1.0 (1.0, 1.9)	1.0 (1.0, 1.0)	1.2 (1.0, 1.8)
MRD status in bone marrow[b]			
Evaluable responders, n	7	6	4
MRD negative, n	7	5	4

Confirmed Response Duration by Dose[a]

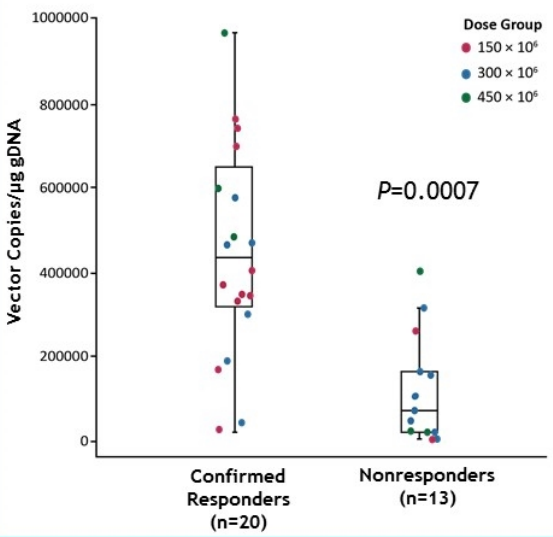


DOR, duration of response; MRD, minimal residual disease; NE, not estimable; PD, progressive disease; PR, partial response; sCR/CR, stringent complete response/complete response; VGPR, very good partial response.
[a]Patients with ≥2 months of follow up or PD/death within 2 months. Response confirmed by a consecutive response of the same category or better.
[b]Patients with ≥PR and ≥1 valid post-baseline MRD assessment by Adaptive next-generation sequencing. 150x10^6 dose 6 neg at 10^{-6} and 1 neg at 10^{-5}, 300x10^6 dose 4 neg at 10^{-6} and 1 at 10^{-5}, 450x10^6 2 neg at 10^{-6} and 2 at 10^{-5}.

Data as of 4 September 2019

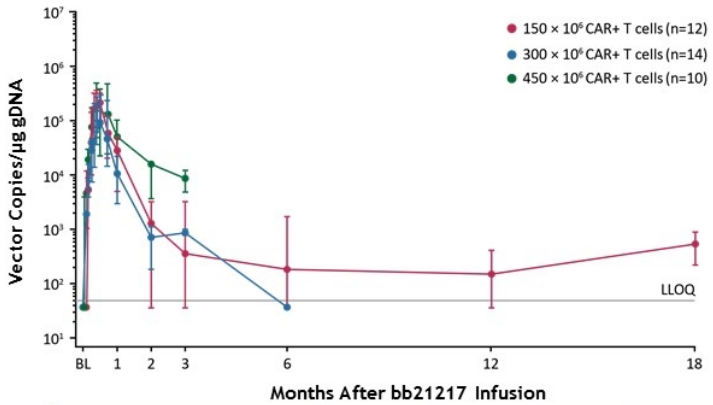
Confirmed responders show increased CAR T cell expansion and durable persistence

Peak VCN by Response



P value based on a 2 sided Wilcoxon rank sum test.

Median VCN Over Time by Dose



Month	3	6	12	18
Detectable vector[a], n	13	8	2	2
No. at risk[b]	15	10	4	2

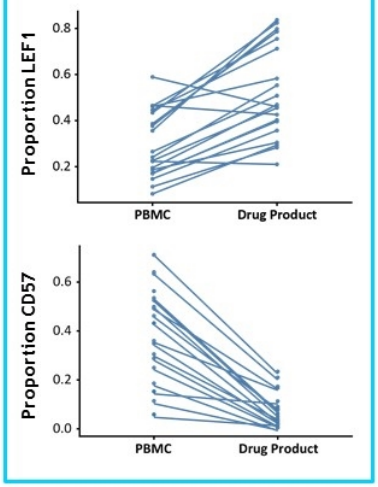
BL, baseline; gDNA, genomic DNA; IQR, interquartile range; LLOQ, lower limit of quantification; VCN, vector copy number.
[a] includes detectable but not measurable. [b] includes VCN data for patients until PD,includes 1 patient who received subsequent chemotherapy before progression. Error bars for median VCN represent IQR

Data as of 4 September 2019

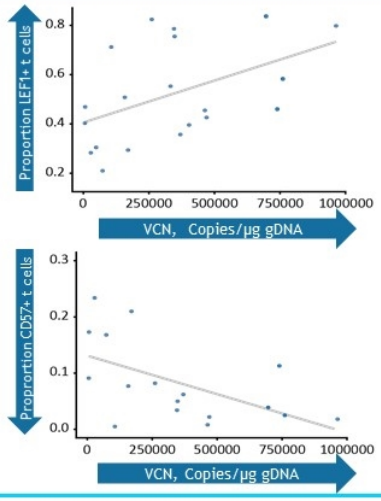
Enrichment for memory-like T cells is associated with robust CAR T expansion and lack of progression by month 6

- Patients with a higher proportion of memory-like T cells in bb21217 drug product have significantly better peak expansion
- A higher proportion of memory-like T cells is associated with numerically less progression by M6

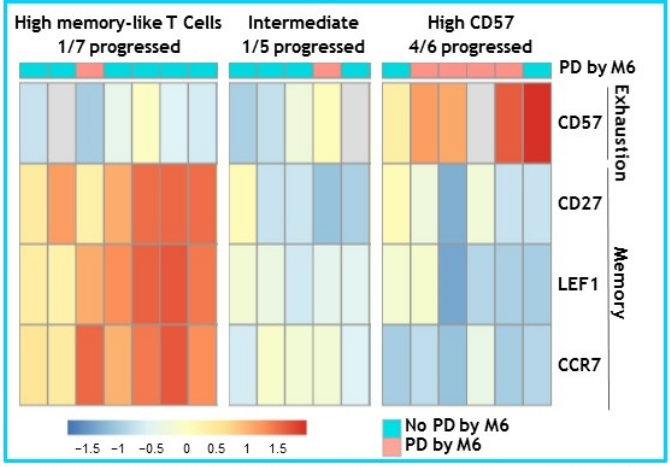
bb21217 Drug Product Enriched For Memory-Like T Cells



Memory-Like T Cells Associated With Peak CAR T Expansion



Memory-Like T Cells in Drug Product Associated With Lack of Progression at M6[a]



M6, month 6; PBMC, peripheral blood mononuclear cell in apheresis product; PD, progressive disease; VCN, vector copy number. [A]Responding Patients with biomarker data who had either PD or a month 6 visit confirming absence of PD, 1 patient with continued stable disease at M6 included.

Data as of 4 September 2019

Emerging data supports memory T cell hypothesis

- Safety profile consistent with known toxicities of CAR T-cell therapies
- Confirmed responses achieved across all doses
- Detectable CAR-T cells at 18 months for patients remaining in response with greater than 20 months follow up
- Demonstrated association between enrichment in 21217 manufacturing process and robust CAR-T cell expansion
- Dose escalation is complete. Continue to evaluate safety and efficacy at recommended phase 2 dose of 450 x 10^6 dose

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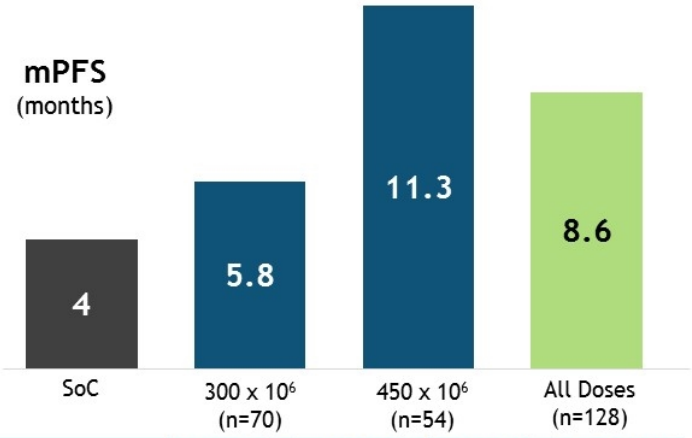
recode for life

KarMMa topline data readout

ide-cel (bb2121): Positive Pivotal Data



	150 x 10^6 CAR+ T cells (N=4)	300 x 10^6 CAR+ T cells (N=70)	450 x 10^6 CAR+ T cells (N=54)	All Doses CAR+ T cells (N=128)
ORR, n (%)	2 (50.0)	48 (68.6)	44 (81.5)	94 (73.4)
CR/sCR, n (%)	1 (25.0)	20 (28.6)	19 (35.2)	40 (31.3)
Median DoR, mo	---	9.9	11.3	10.6

- **Heavily pretreated population**
  - 94% refractory to anti-CD38, 84% triple refractory
  - All patients were refractory to their last treatment (progression during or within 60 days of last therapy)
- **Deep and durable responses across dose levels**
  - mPFS of >11mo at the 450 x 10^6 dose
  - Durability is consistent across doses
- **Safety consistent with the Ph1 data**
  - Gr ≥ 3 CRS and iiNT were reported in <6% of subjects at each target dose
  - CRS and iiNT of any grade occurred in 83.6% and 18% of patients, respectively

iiNT: investigator identified neurotoxicity
Ide-cel is being developed in collaboration with Bristol-Myers Squibb

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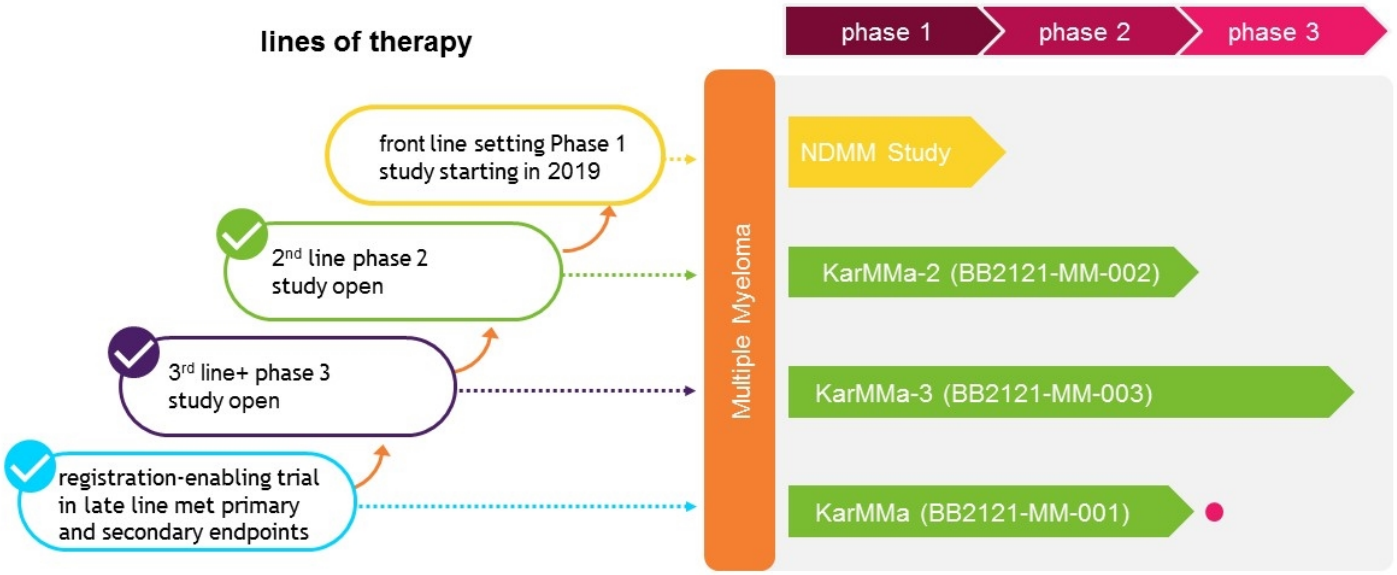
Advancing ide-cel (bb2121) into earlier lines of multiple myeloma



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● Planned 1H:2020 regulatory submission

closing

bbb at ASH 2019:

LentiGlobin TDT	• Patients achieving and maintaining TI across genotypes • Launch progressing in EU
LentiGlobin SCD	• Clinical impact underscored by impact on underlying disease • Program gradually expanding
bb21217	• Indication of hypothesis bearing out • Further investigation underway
bb2121	• Topline data support 1H:2020 regulatory submission • Development path in earlier lines of therapy progressing


thank you
LET'S RECODE
THE STORY

Exhibit 99.2

bluebird bio Presents New Data Demonstrating Long-Term Transfusion Independence and Safety for LentiGlobin™ Gene Therapy for β-thalassemia (betibeglogene autotemcel) at 61st ASH Annual Meeting and Exposition

More than four years of durable transfusion independence (TI), stable total hemoglobin (Hb) levels and reduced liver iron concentrations in completed Phase 1/2 Northstar (HGB-204) study in patients who do not have a β^0/β^0 genotype

Ninety percent of evaluable patients who do not have a β^0/β^0 genotype achieved TI, with median average total Hb levels of 12.2 g/dL in Phase 3 Northstar-2 (HGB-207) study

In ongoing Phase 3 Northstar-3 (HGB-212) study in patients with β^0/β^0 genotype or IVS-I-110 mutation, the two patients evaluable for TI achieved it with Hb levels of 13.2 g/dL and 10.4 g/dL at last visit

Nine of 11 patients with at least six months of follow-up in HGB-212 have not had a transfusion for at least three months

CAMBRIDGE, Mass.— (BUSINESS WIRE)— December 9, 2019 - bluebird bio, Inc. (Nasdaq: BLUE) announced new data from ongoing studies of LentiGlobin™ gene therapy for β-thalassemia (betibeglogene autotemcel) in pediatric, adolescent and adult patients who have transfusion-dependent β-thalassemia (TDT), including results from the Phase 3 Northstar-3 (HGB-212) study in patients with a β^0/β^0 genotype or IVS-I-110 mutation, and the Phase 3 Northstar-2 (HGB-207) study in patients who do not have a β^0/β^0 genotype. These data, as well as updated results reflecting up to five years of follow-up from the completed Phase 1/2 Northstar (HGB-204) study, were presented at the 61st American Society of Hematology (ASH) Annual Meeting and Exposition in Orlando, Florida.

As of the data cutoff presented today, 52 pediatric, adolescent and adult patients with TDT who do not have a β^0/β^0 genotype or have a β^0/β^0 genotype have been treated with LentiGlobin for β-thalassemia in the Northstar, Northstar-2 and Northstar-3 studies.

“The results from our clinical studies of LentiGlobin for β-thalassemia support its potential benefits and consistent safety profile across a broad range of TDT genotypes and patient populations, including pediatric patients, with the longest duration of follow-up now extending beyond five years,” said David Davidson, M.D., chief medical officer, bluebird bio. “Importantly, patients have achieved and maintained transfusion independence, with improvements in multiple markers of bone marrow red blood cell production, as well as reductions in iron overload. These outcomes demonstrate the long-term disease-modifying potential of LentiGlobin for people living with TDT.”

TDT is a severe genetic disease caused by mutations in the β-globin gene that result in reduced or significantly reduced 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 was designed to address 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). bluebird bio's clinical development program for LentiGlobin for β-thalassemia 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.

"Blood transfusions, while necessary, result in unavoidable iron overload, which can cause multi-organ damage. These infusions also have an impact on patients and their families' lives," said Ashutosh Lal, M.D., UCSF Benioff Children's Hospital, Oakland, Calif. "It's encouraging to see that adult, adolescent and pediatric patients with various genotypes in the LentiGlobin for β-thalassemia clinical trials have achieved and continue to maintain transfusion independence."

Northstar (HGB-204) Efficacy
As of June 12, 2019, data from up to five years (median 44.9; min–max: 34.8–61.3 months) of follow-up from the completed Phase 1/2 Northstar (HGB-204) study show durable transfusion independence (TI) and stable HbA^{T87Q} levels in patients who achieved TI. TI is defined as weighted average Hb ≥ 9 g/dL without red blood cell (RBC) transfusions for more than 12 months.

Eight of 10 treated patients who did not have a β^0/β^0 genotype achieved and continued to maintain TI for up to 51.3 months as of the data cutoff, with a median weighted average Hb during TI of 10.3 g/dL. Transfusion volumes were reduced by 79% and 52% in the two patients who did not achieve TI.

In patients who have a β^0/β^0 genotype (n=8), three of eight achieved and continued to maintain TI with a current duration up to 30.4 months as of the data cutoff, and a median weighted average Hb during TI of 9.9 g/dL.

Among patients who achieved TI, a decrease in markers of iron burden, including liver iron concentration, serum ferritin and transferrin saturation, were observed over time. Liver iron concentrations began to decrease in eight of the 11 patients who achieved TI, with the largest decrease observed in patients who had 48 months of data available (n=7). A median 44% reduction (min–max: 17%–83%) was reported in these seven patients.

Northstar-2 (HGB-207) Efficacy
As of June 12, 2019, 21 of 23 patients were treated and have been followed for a median of 11.6 months. These patients ranged in age from 8 to 34 years, including six pediatric (<12 years) and 15 adolescent/adult (≥12 years) patients. Three patients with more than 24 months of follow-up are enrolled in the long-term follow-up study LTF-303.

Ninety percent (9/10) of patients evaluable for TI had achieved it, with median weighted average Hb levels of 12.2 g/dL (min–max: 11.4–12.8 g/dL) during TI. All nine patients continued to maintain TI for a median duration of 15.2 months (min–max: 12.1–21.3 months) as of the data cutoff.

Ninety percent (18/20) of patients with at least five months of follow-up had not received a transfusion for at least 3.5 months and total Hb was near normal in most, with the median total Hb at Months 6, 12 and 18 at 11.5 (n=17), 12.3 (n=11) and 12.2 g/dL (n=8), respectively. HbA^{T87Q} levels were stable over time: 8.7 g/dL at Month 6; 9.3 g/dL at Month 12; and 9.4 g/dL at Month 18.

In an exploratory analysis, bone marrow from nine patients who had reached 12 months of follow-up and were transfusion independent was evaluated for myeloid to erythroid ratio. A low myeloid to erythroid ratio is a key feature of abnormal bone marrow RBC production that is characteristic of patients with TDT.

In all nine patients, an increase in the myeloid to erythroid ratio was observed, suggesting improvement in bone marrow RBC production. A trend toward normalization of soluble transferrin receptor, a marker of RBC production, and reticulocyte counts, a marker of hemolysis or RBC destruction, was also observed. The trend toward normalization in RBC production supports the disease-modifying potential of LentiGlobin for β-thalassemia for patients with TDT.

Northstar-3 (HGB-212) Efficacy
As of September 30, 2019, 13 patients (eight β_0/β_0, two β_0/IVS-I-110, three homozygous IVS-I-110 genotypes) were treated and had a median follow-up of 8.8 months (min–max: 2.5–20.0 months). Median age at enrollment was 17 years of age (min–max: 7–33 years); four patients were under 12 years of age.

Two patients had at least 12 months of follow-up and were evaluable for TI. Both patients, one patient with a β_0/β_0 genotype and one pediatric patient with a β_0/IVS-I-110 genotype, achieved TI, and continued to maintain it with Hb levels of 13.2 g/dL and 10.4 g/dL, respectively, at last visit.

In addition, nine of 11 patients with at least six months of follow-up did not receive a transfusion for more than three months as of last follow-up. In these patients, total Hb levels ranged from 8.3–14.2 g/dL at last visit.

LentiGlobin for β-thalassemia Safety
Non-serious adverse events (AEs) observed during the HGB-204, HGB-207 and HGB-212 clinical studies that were attributed to LentiGlobin for β-thalassemia were hot flush, dyspnoea, abdominal pain, pain in extremities, thrombocytopenia, leukopenia, neutropenia and non-cardiac chest pain. One serious adverse event (SAE) of thrombocytopenia was considered possibly related to LentiGlobin for β-thalassemia 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.

With more than five years of follow-up to date, there have been no new unexpected safety events, no deaths, no graft failure and no cases of vector-mediated replication competent lentivirus or clonal dominance. In addition, there have been no new reports of veno-occlusive liver disease (VOD) as of the data cutoff presented at ASH.

About LentiGlobin for β-Thalassemia (betibeglogene autotemcel)
The European Commission granted conditional marketing authorization for LentiGlobin for β-thalassemia, 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.

TDT is a severe genetic disease caused by mutations in the β-globin gene that result in reduced or significantly reduced 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 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 may eliminate or significantly reduce the need for transfusions.

The conditional marketing authorization for ZYNTEGLO is only valid in the 28 member states of the EU as well as Iceland, Liechtenstein and Norway. For details, please see the Summary of Product Characteristics (SmPC).
The U.S. Food and Drug Administration granted LentiGlobin for β-thalassemia Orphan Drug status and Breakthrough Therapy designation for the treatment of TDT.

By the end of 2019, bluebird bio plans to initiate rolling submission of a Biologics Licensing Application (BLA) of LentiGlobin for β-thalassemia in the U.S. for the treatment of patients with TDT who do not have a β^0/β^0 genotype. bluebird bio is engaged with the FDA in discussions regarding the requirements and timing of the various components of the rolling BLA submission and, subject to these ongoing discussions, the company is currently planning to complete the BLA submission in the first half of 2020.

LentiGlobin for β-thalassemia continues to be evaluated in the ongoing Phase 3 Northstar-2 and Northstar-3 studies. For more information about the ongoing clinical studies, visit **www.northstarclinicalstudies.com** or **clinicaltrials.gov** and use identifier NCT02906202 for Northstar-2 (HGB-207), NCT03207009 for Northstar-3 (HGB-212).

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 β-thalassemia. For more information visit: **https://www.bluebirdbio.com/our-science/clinical-trials** or clinicaltrials.gov and use identifier NCT02633943 for LTF-303.

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 including cerebral adrenoleukodystrophy, sickle cell disease, 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, LentiGlobin, and bluebird bio are trademarks of bluebird bio, Inc.

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, including statements regarding the Company's views with respect to the potential for LentiGlobin to treat transfusion-dependent β-thalassemia (TDT). 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 risks that the preliminary positive efficacy and safety results from our prior and ongoing clinical trials of LentiGlobin to treat TDT will not continue or be repeated in our ongoing or planned clinical trials or in the commercial context, risks that the current or planned clinical trials of our product candidates will be insufficient to support future regulatory submissions or to support marketing approval in the US, the risk that the ability to submit and obtain approval of a BLA is ultimately an FDA review decision and the FDA may impose additional requirements before we can submit or obtain approval of a BLA, or before we are able to treat patients in the commercial context, and the risk that any one or more of our product candidates, will not be successfully developed, approved or commercialized. 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 and Bristol-Myers Squibb Present Updated Data from Ongoing Phase 1 Study of BCMA-Targeted CAR T Cell Therapy bb21217 in Relapsed/Refractory Multiple Myeloma at 61st ASH Annual Meeting and Exposition

Safety profile consistent with known toxicities of CAR T therapies

CAR T persistence observed in 8/10 evaluable responders at Month 6 and 2/2 evaluable responders at Month 18

CAMBRIDGE, Mass., & PRINCETON, NJ – December 9, 2019 – bluebird bio, Inc. (Nasdaq: BLUE) and Bristol-Myers Squibb (NYSE: BMY) announced updated safety and efficacy results from the ongoing Phase 1 study (CRB-402) of bb21217, an investigational BCMA-targeted chimeric antigen receptor (CAR) T cell therapy being studied in patients with relapsed/refractory multiple myeloma (R/RMM). The data were presented at the 61st American Society of Hematology (ASH) Annual Meeting and Exposition in Orlando, Florida.

bb21217 is an investigational BCMA-targeted CAR T cell therapy that uses the idecabtagene vicleucel (ide-cel; bb2121) CAR molecule and is cultured with the PI3 kinase inhibitor (bb007) to enrich for T cells displaying a memory-like phenotype with the intention to increase the *in vivo* persistence of CAR T cells.

“Early data from the CRB-402 study in heavily pre-treated patients (median of six prior lines) with relapsed/refractory multiple myeloma demonstrate the potential for durable responses following bb21217 CAR T cell treatment, with a median duration of response of 11.1 months at the 150 x 10^6 CAR+ T cell dose level,” said David Davidson, M.D., chief medical officer, bluebird bio. “Consistent with the hypothesis underlying the bb21217 program that memory-like phenotype T cells may survive longer *in vivo*, we have observed durable CAR T cell persistence in evaluable patients (n=2/2) with ongoing response at up to 18 months following treatment. We are continuing to recruit additional patients in the study and performing ongoing assessments of the functional persistence of bb21217, as well as its potential correlation to durability of response.”

CRB-402, the first in-human study of bb21217 in patients with R/RMM, is designed to assess the primary endpoint of safety as well as other pre-defined endpoints including efficacy and pharmacokinetics measurements. CRB-402 is a two-part, open-label, multi-site Phase 1 study of bb21217 in adults with R/RMM with a projected final enrollment of 74 patients. The dose escalation part of CRB-402 is complete, and the dose expansion part of the study is ongoing.

“The data of CRB-402 provide additional support that targeting BCMA with a CAR T therapy could be beneficial in treating relapsed/refractory multiple myeloma, particularly for heavily pre-treated patients,” said Kristen Hege, M.D., Senior Vice President, Hematology/Oncology and Cell Therapy, Early Clinical Development for Bristol-Myers Squibb. “We have observed durable responses with bb21217 in this study and look forward to further results.”

“One of the challenges in treating patients with relapsed/refractory multiple myeloma is that they often become resistant to currently available therapies and response durations generally shorten with each subsequent therapy,” said presenting author Jesus G. Berdeja, M.D., Sarah Cannon Center for Blood Cancers, Nashville, Tennessee. “In this heavily-treated patient population, we are encouraged by the results with bb21217 treatment in this ongoing study.”

As of the September 4, 2019 cutoff date, data include results for 38 treated patients. Twenty-four patients received bb21217 in the dose escalation cohort at three dose levels (12 at 150 x 10^6 CAR+ T cells; six at 300 x 10^6 CAR+ T cells; and six at 450 x 10^6 CAR+ T cells). Fourteen additional patients received bb21217 in the dose expansion cohort at two dose levels (8 at 300 x 10^6 CAR+ T cells and 6 at 450 x 10^6 CAR+ T cells). The patients had a median of six prior lines of therapy (min – max; 3 – 17 lines) and 82% had at least one prior autologous stem cell transplant. High-risk cytogenetics were reported in 34% of patients and 95% of patients received prior treatment with an anti-CD38 antibody. All patients treated in CRB-402 (n=38) had previously received at least three prior lines of therapy, including an immunomodulatory agent and proteasome inhibitor. The enrollment criteria for the dose expansion cohort required all enrolled patients (n=14) to be refractory to their last prior line of therapy and have previously received an anti-CD38 antibody.

Safety Results
As of the data cutoff, the adverse events observed with bb21217 were consistent with known toxicities of CAR T therapies, regardless of dose level.

Of the 38 treated patients, the most common Grade 3/4 toxicities include neutropenia (82%), leukopenia (55%), thrombocytopenia (55%), anemia (50%), lymphopenia (34%), hypophosphatemia (21%), hyponatremia (13%) and febrile neutropenia (11%). Grade 3/4 infections were reported in seven patients (18%).

Twenty-five of 38 patients (66%) developed bb21217-related cytokine release syndrome (CRS); 12 Grade 1, 11 Grade 2, one Grade 3 and one Grade 5 (death). The fatal CRS event occurred at the 450 x 10^6 CAR+ T cells dose level, after 15 days of follow-up. Nine of 38 (24%) patients developed neurotoxicity; three Grade 1, three Grade 2, two Grade 3 (one with vertigo/dizziness and one with encephalopathy) and one Grade 4 (encephalopathy, previously reported). For the one patient previously reported with Grade 4 neurotoxicity, Grade 3 CRS was also reported, and both have resolved.

Efficacy Results
As of the data cutoff, 33 of the 38 patients were evaluable for clinical response as defined per the International Myeloma Working Group Uniform Response Criteria for multiple myeloma.

Twelve patients were evaluable in the 150 x 10^6 CAR+ T cells cohort, with a median follow-up of 17.6 months (min – max; 12 – 23 months). Ten of 12 (83%) evaluable patients (defined as treated patients with ≥ two months of response data or progressive disease/death/lost to follow-up within <=2 months) in the 150 x 10^6 CAR+ T cells cohort demonstrated clinical response, including four with a stringent complete response (sCR) or complete response (CR), and six with a very good partial response (VGPR). Among the ten confirmed responders, the median duration of response was 11.1 months (95% Confidence Interval (CI); 3.3 – not estimable).

As of the data cutoff, follow-up within the two higher dose cohorts (300 x 10^6 and 450 x 10^6 CAR+ T cells) remains early and none of the confirmed responders have experienced disease progression. In the 300 x

10^6 CAR+ T cells cohort, 14 patients were evaluable for response and six of the 14 (43%) evaluable patients demonstrated clinical response, including four with a VGPR and two with a partial response (PR), with a median follow-up of four months (min – max; 2 – 10 months). In the 450 x 10^6 CAR+ T cells cohort, seven patients were evaluable for response and four of the seven (57%) evaluable patients demonstrated clinical response, including one with a sCR, two with a VGPR and one with a PR, with a median follow-up of 3.3 months (min – max; <1 – 6 months).

Evidence of myeloma in the bone marrow, known as minimal residual disease (MRD), was undetectable by next-generation sequencing at a sensitivity level of 10^{-5} in 94% (n=16/17) of all confirmed responders who had evaluable bone marrow samples (patients with $\geq$ PR and $\geq$ 1 valid post-baseline MRD assessment).

As of the data cutoff, CAR T cell persistence was observed in eight of ten patients with ongoing response and evaluable at six months, and two out of two patients with ongoing response and evaluable at 18 months.

The dose expansion part of the CRB-402 study is ongoing to further recruit patients and explore bb21217 at the 450 x 10^6 CAR+ T cells dose cohort, assess functional persistence of bb21217 and durability of response.

About bb21217 for Multiple Myeloma
bb21217 is an investigational BCMA-targeted CAR T cell therapy that uses the ide-cel CAR molecule and is cultured with the PI3 kinase inhibitor (bb007) to enrich for T cells displaying a memory-like phenotype with the intention to increase the *in vivo* persistence of CAR T cells bb21217 is being developed in partnership between bluebird bio and Bristol-Myers Squibb.

The clinical development program for bb21217 includes the ongoing Phase 1 CRB-402 study. CRB-402 is the first-in-human study of bb21217 in patients with R/RMM, designed to assess safety, pharmacokinetics, efficacy and duration of effect. CRB-402 is a two-part (completed dose escalation and ongoing dose expansion), open-label, multi-site Phase 1 study of bb21217 in adults with R/RMM with a projected final enrollment of 74 patients. For more information visit: **clinicaltrials.gov** using identifier NCT03274219.

bb21217 is not approved for any indication in any geography.

About Multiple Myeloma
Multiple myeloma is a cancer of certain cells in the blood, called plasma cells. The cause of multiple myeloma is not known, and currently there is no cure. However, there are a number of treatment options available that can lead to response. For some people with multiple myeloma, response can last many years. Patients who have already been treated with some available therapies but continue to have progression of their disease have “relapsed” and “refractory” multiple myeloma, meaning their cancer has reoccurred after they have received initial treatments. Patients with relapsed and refractory multiple myeloma have fewer treatment options.

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 including cerebral adrenoleukodystrophy, sickle cell disease, 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**.
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About Bristol-Myers Squibb

Bristol-Myers Squibb is a global biopharmaceutical company whose mission is to discover, develop and deliver innovative medicines that help patients prevail over serious diseases. For more information about Bristol-Myers Squibb, visit us at BMS.com or follow us on LinkedIn, Twitter, YouTube, Facebook and Instagram.

Bristol-Myers Squibb: Advancing Cancer Research

At Bristol-Myers Squibb, patients are at the center of everything we do. The goal of our cancer research is to increase quality, long-term survival and make cure a possibility. We harness our deep scientific experience, cutting-edge technologies and discovery platforms to discover, develop and deliver novel treatments for patients.

Building upon our transformative work and legacy in hematology and Immuno-Oncology that has changed survival expectations for many cancers, our researchers are advancing a deep and diverse pipeline across multiple modalities. In the field of immune cell therapy, this includes registrational chimeric antigen receptor (CAR) T-cell agents for numerous diseases, and a growing early-stage pipeline that expands cell and gene therapy targets, and technologies. We are developing cancer treatments directed at key biological pathways using our protein homeostasis platform, a research capability that has been the basis of our approved therapies for multiple myeloma and several promising compounds in early to mid-stage development. Our scientists are targeting different immune system pathways to address interactions between tumors, the microenvironment and the immune system to further expand upon the progress we have made and help more patients respond to treatment. Combining these approaches is key to delivering new options for the treatment of cancer and addressing the growing issue of resistance to immunotherapy. We source innovation internally, and in collaboration with academia, government, advocacy groups and biotechnology companies, to help make the promise of transformational medicines a reality for patients.

Bristol-Myers Squibb Cautionary Statement Regarding Forward-Looking Statements

This press release contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995 regarding, among other things, the research, development and commercialization of pharmaceutical products. All statements that are not statements of historical facts are, or may be deemed to be, forward-looking statements. Such forward-looking statements are based on historical performance and current expectations and projections about our future financial results,

goals, plans and objectives and involve inherent risks, assumptions and uncertainties, including internal or external factors that could delay, divert or change any of them in the next several years, that are difficult to predict, may be beyond our control and could cause our future financial results, goals, plans and objectives to differ materially from those expressed in, or implied by, the statements. These risks, assumptions, uncertainties and other factors include, among others, that future study results will be consistent with the results to date, that bb21217 may not achieve its primary study endpoints or receive regulatory approval for the indication described in this release in the currently anticipated timeline or at all and, if approved, whether such product candidate for such indication described in this release will be commercially successful. No forward-looking statement can be guaranteed. Forward-looking statements in this press release should be evaluated together with the many risks and uncertainties that affect Bristol-Myers Squibb's business and market, particularly those identified in the cautionary statement and risk factors discussion in Bristol-Myers Squibb's Annual Report on Form 10-K for the year ended December 31, 2018, as updated by our subsequent Quarterly Reports on Form 10-Q, Current Reports on Form 8-K and other filings with the Securities and Exchange Commission. The forward-looking statements included in this document are made only as of the date of this document and except as otherwise required by applicable law, Bristol-Myers Squibb undertakes no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events, changed circumstances or otherwise.

Bluebird bio Cautionary Statement Regarding Forward-Looking Statements

This press release contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995 regarding, among other things, the research, development and commercialization of pharmaceutical products. All statements that are not statements of historical facts are, or may be deemed to be, forward-looking statements. Such forward-looking statements are based on historical performance and current expectations and projections about our future financial results, goals, plans and objectives and involve inherent risks, assumptions and uncertainties, including internal or external factors that could delay, divert or change any of them in the next several years, that are difficult to predict, may be beyond our control and could cause our future financial results, goals, plans and objectives to differ materially from those expressed in, or implied by, the statements. These risks, assumptions, uncertainties and other factors include, among others, that future study results will be consistent with the results to date, that bb21217 may not achieve its primary study endpoints or receive regulatory approval for the indication described in this release in the currently anticipated timeline or at all and, if approved, whether such product candidate for such indication described in this release will be commercially successful, and that the collaboration with Bristol-Myers Squibb may not continue or be successful. No forward-looking statement can be guaranteed. Forward-looking statements in this press release should be evaluated together with the many risks and uncertainties that affect bluebird bio's business, particularly those identified in the risk factors discussion in bluebird bio's Annual Report on Form 10-K for the year ended December 31, 2018, as updated by our subsequent Quarterly Reports on Form 10-Q, Current Reports on Form 8-K and other filings with the Securities and Exchange Commission. The forward-looking statements included in this document are made only as of the date of this document and except as otherwise required by applicable law, bluebird bio undertakes no obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events, changed circumstances or otherwise.

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