bridgebio

hope through rigorous science





JP Morgan Presentation

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BridgeBio Pharma: Hope through rigorous science

We have created a sustainable engine for the systematic discovery, development, and commercialization of medicines for genetic diseases



Our R³ 5-year vision: We are building a distinctive product-focused genetic disease company



1-2 INDs per year on average, technology platforms spanning 4 modalities

Leadership team of world-renowned drug hunters

Scientific insight and judgment from industry leaders with a proven track record



Experienced team of R&D operators responsible for 100+ INDs and 20+ approved products



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Late-stage pipeline is poised to deliver significant risk-adjusted revenue and diversified top-line growth over following decade

Risk-adjusted revenue, \$mn



BBIO's catalyst map for 2023 – large potential value inflection points in the first 6 months of the year

Program	Stage	2023	
		1H	2H
Acoramidis for ATTR-CM	Phase 3		ATTRibute Ph3 topline data (July)
Low-dose infigratinib for achon. and hypochon.	Phase 2	Achon. Ph2 Cohort topline data (March	5 1)
Encaleret for ADH1	Phase 3		CALIBRATE Ph3 topline data (YE23)
AAV5 gene therapy for CAH (BBP-631)	Phase 2	Update: To date BBP-631 has been well-tolerated in 4 p first two dose levels. We plan to collect data from addit at the highest dose (cohort 3) which we expect will be a	patients treated at the cional patients including available in 2H23 Ph2 data readout (2H23)
Ribitol for LGMD2i	Phase 3-ready		Ph3 initiation (2023)
Next-gen G12Ci (BBO-8520)	Pre-IND		🗙 IND (2023)
RAS:PI3K α breaker	Lead-opt	Clinical cano selection (1	didate H23)
	ProgramAcoramidis for ATTR-CMLow-dose infigratinib for achon. and hypochon.Encaleret for ADH1AAV5 gene therapy for CAH (BBP-631)Ribitol for LGMD2iNext-gen G12Ci (BBO-8520) RAS:PI3Kα breaker	ProgramStageAcoramidis for ATTR-CMPhase 3Low-dose infigratinib for achon. and hypochon.Phase 2Encaleret for ADH1Phase 3AAV5 gene therapy for CAH (BBP-631)Phase 2Aibitol for LGMD2iPhase 3-readyNext-gen G12Ci (BBO-8520)Pre-IND Lead-opt	ProgramStage20Acoramidis for ATTR-CMPhase 3Low-dose infigratinib for achon. and hypochon.Phase 2Encaleret for ADH1Phase 3AAV5 gene therapy for CAH (BBP-631)Phase 2Vipdate: To date BBP-631 has been well-tolerated in 4 g first two dose levels. We plan to collect data from addit at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at the highest dose (cohort 3) which we expect will be at

We are well-capitalized into 2024

Financial position as of 3Q22 report Cash¹ Shares outstanding² ~\$558 Mn ~149 Mn **Anticipated burn trajectory Runway into 2024** with cash on hand to read out: Achon cohort 5 data, ATTR Ph3 data, ADH1 Ph3 data, CAH POC, KRAS Clinical Data Anticipate OpEx and cash burn will continue to decline in Q4 Potential levers to further extend runway, given our 5 owned late-stage clinical assets

□ Non-dilutive financings (i.e., royalty sales on late-stage assets)

Upfronts and burn mitigation through BD deals

OpEx, \$mn

26% decrease in QoQ OPEX less SBC this year



Acoramidis for transthyretin (TTR) amyloidosis (ATTR)

- Prevalence (US/EU): 400K
- Stage: Phase 3
- Next Catalyst: Phase 3 30-month allcause mortality & CV hospitalization data expected mid-2023



Acoramidis is well-positioned for success in the ATTR-CM market

- ATTR is a \$3B global market today, growing to \$10-\$15B
- Acoramidis is a 2nd generation TTR stabilizer that has demonstrated a superior stabilization profile ex vivo and in vitro
 - Superior stabilization has been demonstrated to improve outcomes 20 mg v 80 mg in ATTR-ACT
- Our Phase 3 patient population appears to be left-shifted in time. Nevertheless, with the absolute magnitude of mortality being large and a more potent stabilizer, the trial remains wellpowered
- In the contemporary ATTR-CM clinical context, we believe best-in-class data is represented by patients living longer and going to the hospital less:
 - A better win ratio
 - Improved survival rates at 30 months
 - A meaningful separation in CV outcomes at 30 months
 - Superior treatment-related biomarkers as measured by serum NT-proBNP and TTR concentrations

ATTR-CM has grown into a multi-billion dollar global market in 3 years and is primed for continued expansion



Drivers of market growth include:

- Increased adoption of non-invasive diagnostic tools
- Earlier detection of disease
- Growing market familiarity with oral TTR stabilizers
- Tailwinds from the Inflation
 Reduction Act reducing patient
 out-of-pocket expenditure
- Durable market growth with
 Vyndamax polymorph patent
 protection through 2035⁵
- Precedent of persistent brand sales amidst 1st generation generic entry

¹ATTR market includes all approved drugs for ATTR-PN and ATTR-CM

²First ATTR-CM sales occurred in Q2 2019

³Assumes linear annualization of Q4 2022 sales.

⁴Consensus estimates of \$10B+ ATTR-CM market. Projection conducted prior to announcement of IRA market expansion opportunity for stabilizers. ⁵Orange Book

Acoramidis was designed to achieve maximal stabilization and preserve native TTR



We plan to enter the ATTR-CM market with acoramidis, a second generation, more potent TTR stabilizer

TTR stabilization by acoramidis is *two-fold more potent* than tafamidis



Source: Ji, A.X., et al. American Heart Association Scientific Sessions 2019;140(1):13847.

¹Western Blot quantitation of tetrameric TTR in plasma samples incubated with stabilizers. Each bar represents the percentage of tetrameric TTR remaining after 72 hr incubation at pH 3.8, n = 3 independent experiments. DMSO = control.

²FPE characterization of TTR binding site occupancy in serum incubated with stabilizer, n = 12 for each condition tested.

Acoramidis increases serum TTR significantly more than tafamidis

Serum TTR (mean % change from baseline)



Note: Direct cross-study comparisons may suggest misleading similarities or differences. The values shown are directional and do not report robust comparative analysis. ¹Estimated from Damy, T., et al., Eur J Heart Fail. 2021;23(2):277-285; ²BridgeBio Part A press release, December 27, 2021. ³Heitner SB, et al. J Am Coll Cardiol. 2019, 73(9):660. Oral presentation at ACC 2019.

Acoramidis improves median NT-proBNP at 30 months



Median percent change from baseline in NT-proBNP^{1,2}

NT-proBNP is emerging as a leading predictive biomarker of mortality

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Note: Direct cross-study comparisons may suggest misleading similarities or differences. The values shown are directional and do not report robust comparative analysis. ¹Masri A, et al. J Am Coll Cardiol, 2022;79(9):227. Oral presentation at ACC 2022. ²NT-proBNP was a reported laboratory parameter, not a pre-specified safety endpoint. Baseline defined as the date of the first dose of acoramidis. ³Represents all evaluable data from participants who continued in the study. ⁴Hanna M, et al. J Am Coll Cardiol. Adv. 2022;1(5):100148.

ATTRibute-CM is on track to provide Part B 30-month mortality and CV hospitalization results in mid 2023^{1,2}



6MWD = Six-minute walk distance KCCQ = Kansas City Cardiomyopathy Questionnaire NYHA = New York Heart Association

^{99m}Tc = Technetium labeled pyrophosphate (PYP) or bisphosphonate (e.g., DPD) CV = cardiovascular-related

¹ClinicalTrials.gov identifier: NCT03860935 ²Gillmore JD et al. Circulation 2019;140(1):14214. Oral poster presented at AHA. ³Primary analysis will use the Finkelstein-Schoenfeld method

Confidence in positive ATTRibute-CM Month 30 outcomes data based on Month 12 data

Month 12 acoramidis vs. placebo		Month 30 What gives us confidence		
NT-proBNP Cardiac biomarker associated with survival in ATTR-CM	+0.6% vs. +24.3% on placebo ¹	Acoramidis nominally improved quality-of-life and stabilized NT-proBNP vs. placebo		
Serum TTR Measure of TTR stabilization	+38.5% vs. -0.7% on placebo ¹	Acoramidis was generally well-tolerated and demonstrated potent, sustained TTR stabilization		
KCCQ Quality-of-life measurement	Observed improvement ²	27% fewer treatment emergent adverse events leading to death occurred in patients on acoramidis vs. placebo ³		

Source: BridgeBio Part A press release, December 27, 2021

¹Nominal p<0.05 vs. placebo based on absolute change from baseline between groups

²Nominal P<0.05, mixed model repeated measures without imputation

³To protect the integrity of Part B, Sponsor's access to adverse event data for Part A excludes AEs leading to a cardiovascular hospitalization excepting events with the outcome of death

Patient standard of care has changed: Diagnosis, management, and prognosis of ATTR-CM have improved

Elevated diagnostic suspicion & disease awareness

Rapid rise of ATTR-CM diagnoses, particularly wildtype disease in recent years¹



Increased adoption of non-invasive diagnostic modality³

 Grade 2/3 finding on bone scintigraphy was >99% sensitive & in the absence of a monoclonal protein was 100% specific in detecting ATTR-CM²



Evolving management with standard heart failure interventions⁴

- ATTR-CM patients generally have a **narrow therapeutic window for volume optimization with loop diuretics** due to stiff ventricles with limited stroke volume
- Guideline-directed medical therapy for heart failure can be poorly tolerated due to restrictive physiology of ATTR-CM

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¹Witteles RM, et al. JACC Heart Fail. 2019;7(8)709-716. ²Gillmore JD, et al. Circulation. 2016;133(24):2404-2412. ³Bourque J., et al., American Journal of Cardiology 2021; 167:98-103. ⁴Stern L. et al., Methodist DeBakey Cardiovasc. Jour. 2022;18(2):59-72.

Bar for demonstrating efficacy has increased as patients are 'leftshifted' in time as compared with ATTR-ACT



Implications:

- Potential for later separation on survival
- Potential for smaller risk reduction
- Opportunity for more effective interventions given earlier presentation and residual unmet need

Duration of ATTR Cardiac Amyloidosis

ATTRibute-CM trial continues to have high operating fidelity as Part B of the study matures to 30-month outcomes readout

Ongoing fidelity of ATTRibute-CM remains high

✓ Low tafamidis drop-in rate in study participants

✓ Maximum 18-month duration on concomitant tafamidis allowed by protocol

✓ Low study discontinuation rate for reasons outside of mortality

✓ ATTRibute-CM was adequately powered to enroll 510 participants, and over-enrolled by 24% with 632 participants randomized

ATTRibute-CM Part B topline data are planned to be announced in mid-2023

Market research shows we have many ways to win

How will prescribers select which drug to use?¹



2

3

Totality of benefit across CV outcomes (as measured by Win Ratio)



- Absolute mortality rate on intervention 80+% survival
- Cardiovascular hospitalization on intervention



Favorable safety & tolerability profile



Best in class stabilizer adoption potential²

74% of cardiologists surveyed would adopt a best-in-class stabilizer **within 1 year of approval** for newly diagnosed patients

¹BridgeBio market research, December 2022 (n=90) ²BridgeBio market research, May 2021 (n=141)

Low-dose oral FGFR inhibitor (infigratinib) for achondroplasia

- Prevalence (US & EU): 55k¹, ~10k
 treatable
- Stage: Phase 2
- Next Catalyst: Phase 2 update from cohort 5 (March 2023)



Executive summary

- Achondroplasia, hypochondroplasia, and other FGFR3-driven skeletal dysplasias represent a large, durable market
 - Total addressable market greater than \$5Bn
 - Vosoritide, the first approved treatment for Achondroplasia, is delivering strong sales and has seen increased guidance every quarter since launch
- To address this market, BridgeBio is developing infigratinib as a potentially best in class product, focusing on two design principles:
 - Target the condition at the source to maximize efficacy
 - Provide a safe oral option to avoid the need for injections
- In preclinical mouse models infigratinib was shown to drive 3-5x greater skeletal growth than other potential options
 - Infigratinib also demonstrated preclinical benefit in widening the foramen magnum
- In clinical studies we have observed a clean safety profile and a +1.52 cm increase in annualized height velocity (AHV) for cohort 4 compared to baseline
- We expect to report on cohort 5 in mid-March or earlier
 - Cohort 5 is twice the dose of cohort 4 (0.25 mg/kg/day vs 0.128 mg/kg/day)
 - We expect efficacy to be in line with or greater than cohort 4, with a continued clean safety profile

Achondroplasia and related FGFR-driven skeletal dysplasias represents a large durable total addressable market for infigratinib, with IP to at least 2041

Expansion of the achondroplasia opportunity into hypochondroplasia and other skeletal dysplasias could result in a total treatable population on par with other large genetic medicine franchises



The achondroplasia market today is attractive and growing quickly



Drivers of market growth include:

- First available treatment increasing number of children seeking treatment
- Anticipated expansion of treatable population to include youngest children
- High enthusiasm for treatment among physicians and families has seen BioMarin increase guidance every quarter since launch
- Growing awareness among pediatric endocrinologists referred from geneticists

Against this large market and unmet need, we are developing infigratinib based on two key design principles

Objectives

Design principles

Maximize efficacy

For all the manifestations of ACH—not just height—which matter for families and physicians

Target the condition directly at the source (FGFR3)

Avoid injections

For children and families, to reduce burden and pain of treatment

Provide a safe oral treatment option

Infigratinib is the only treatment option in development that incorporates both design principles

Infigratinib addresses the first design principle by directly targeting the underlying cause of achondroplasia, FGFR3 overactivity



Slight gain-of-function mutation in FGFR3 leads to disordered chondrocyte proliferation and clear genotype:phenotype

- >95% of cases due to Gly380Arg substitution
- Infigratinib blocks FGFR signaling from inside the cell
- Infigratinib inhibits both pathways responsible for the clinical phenotype associated with achondroplasia
- Clear PD effects in growth plate suggest efficient penetration into target tissue

SOURCE: Ornitz DM et al. Developmental Dynamic 2017; Richette Joint Bone Spine 2007; Unger Curr Osteoporos Rep 2017, Hoover-Fong Am J Gen Med 2017 Confidential and proprietary. Do not duplicate or distribute without written permission from QED Therapeutics, Inc.

By targeting the disease at its source, infigratinib has achieved impressive efficacy in mouse models

Infigratinib 2 mg/kg/day preclinical efficacy

Increase in length compared to non-treated mouse

Asset	Company	Mouse model	Tibia length	Femur length	L4-L6
Infigratinib (BGJ398) 2 mg/kg/day	bridgebio	FGFR3 ^{Y367C/+}	32.6%	20.9%	12.1%
Vosoritide (BMN111) 800 µg/kg/day	BOMARIN	FGFR3 ^{Y367C/+}	6.6%	5.2%	3.3%
TransCon CNP 5.6 mg/kg/day	ascendis pharma	WT	12.3%		
Recifercept (TA-46) 2.5 mg/kg twice weekly	Pfizer	FGFR3 ^{ach/+}	8.6%	6.2%	

Preclinically, infigratinib has shown remarkable efficacy in bone length and a foramen magnum effect

SOURCE: Komla-Ebri J et al. Clin Inv 2016, Lorget et al. Am J Hum Genet 2012, Garcia et al. Science Trans Med 2013, Breinholt ENDO 2017. NOTE: subcutaneous doses, percent increase compared to vehicle treated FGFR3^{Y367C/+}, FGFR3^{ACH/+} mouse unless otherwise noted. Highest dose available for each compound is reported, this does not necessarily translate into the clinical doses

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The PROPEL clinical program to evaluate infigratinib is approaching completion of Phase 2 and initiation of Phase 3



Interim Phase 2 clinical data from Cohort 4 suggests encouraging tolerability profile and potential for best-in-class efficacy at the Cohort 5 dose level

+1.52 cm/yr mean increase in annualized height velocity (AHV) across all children 5 years or older in cohort 4 0.61 cm/yr mean increase in AHV across all children 3 to <5 years of age Baseline AHV of 4.01 cm/yr across all children

 Baseline AHV of 4.01 cm/yr across all children in cohort 4

Well-tolerated with 0 SAEs

- No discontinuations due to AEs
- No dose dependent phosphate elevation
- No ocular AEs

64% responder rate

across all children 5 years or older in cohort 4

 Responses were consistently seen irrespective of baseline AHV

Statistically significant increase in biomarker

- Collagen X is an accepted biomarker for collagenous growth
- No previous cohort has demonstrated significant increases

What are we looking for from our achondroplasia cohort 5 update?

Mean change from baseline in annual height velocity, cm/yr



We are measuring and looking for:

- Clean safety, in-line with cohort 4 and prior disclosure on cohort 5 (no SAEs, no AEs requiring dose modification including hyperphosphatemia)
- Efficacy across 3 measures: change from baseline AHV, responder rate, and % of children getting to 7 cm/yr
- We believe it's essential to have clear baselines for growth and a range of ages to demonstrate compelling efficacy
 - A baseline is critical: Inter-patient variability is greater than intra-patient variability

Market research validates the importance of infigratinib's oral route of administration, suggesting majority share even without an efficacy edge

HCP survey of vosoritide vs. infigratinib showing equivalent efficacy

% of children with achondroplasia seeking treatment who would potentially receive each product

 Blinded survey of a representative sample of 54 HCPs who treat ACH (50/50 bridge academic/community) We tested preference for a blinded **68%** infigratinib TPP vs blinded vosoritide TPP with equivalent efficacy 45% of children on vosoritide would be The blinded infigratinib TPP closely switched to matches the Cohort 4 profile, with upside infigratinib once potential for cohort 5 33% DAILY 17% of survey available, in our ORAL Oral ROA is a major draw vs injectables population would survey expect to not treat at **INJECTION** all, in line with "Our daughter was in the BMRN trial, but we had to discontinue because of the expectations trauma we experienced as a family due to Blinded Blinded the daily injections. How can I learn about vosoritide infigratinib enrolling in QED's trial?"

We see room for significant market share upside with a best-in-class efficacy profile

Quick hits on other value drivers – all \$1bn+ markets

Program	Stage	Next update	Timing guidance
AAV5 gene therapy for CAH	Ph1/2	Ph2 data	2H23
Encaleret (CaSRi) for ADH1	Ph3	Ph3 topline data	YE23/1H24
Ribitol for LGMD2i	Ph3-ready	Ph3 initiation and FDA Feedback	2023
Next gen dual G12Ci (BBO-8520)	IND-enabling	Ph1 initiation	2023 (clinical data 2024)