Hengrui Pharma and Braveheart Bio Present Data from 52-Week Open-Label Extension of Phase 2 Study of HRS/BHB-1893 in Obstructive Hypertrophic Cardiomyopathy at American Heart Association Scientific Sessions 2026

November 6, 2026

HRS/BHB-1893 demonstrated meaningful improvements in exercise capacity and patient-reported outcomes, with rapid, deep, and durable left ventricular outflow tract (LVOT) gradient reduction at 52 weeks

Data support a highly simplified dosing regimen, with a majority of patients potentially served by the starting dose

HRS/BHB-1893 data across both forms of hypertrophic cardiomyopathy (HCM) and preclinical models suggest potential to address underlying pathophysiology of disease, findings consistent with reverse-remodeling and diastolic benefits

Braveheart Bio to host Investor Event and Webcast today at 3:35 p.m. CT (4:35 p.m. ET), including discussion of a planned fixed-dose oHCM study, TRUEHEART-HCM

SHANGHAI and SAN FRANCISCO, Nov. 06, 2026 (GLOBE NEWSWIRE) -- Hengrui Pharma (SSE: 600276; HKEX: 1276) and Braveheart Bio (NASDAQ: BRVE) today presented data from a 52-week open-label extension (OLE) of the Phase 2 study of the investigational, next-generation cardiac myosin inhibitor HRS/BHB-1893 in patients with obstructive hypertrophic cardiomyopathy (oHCM). The data were presented at the American Heart Association (AHA) Scientific Sessions 2026.

After the 12-week core period and a washout period, all 42 patients restarted HRS/BHB-1893 for an additional 52 weeks in the OLE. At Week 52, peak oxygen uptake (pVO2) increased by a mean of 2.7 mL/kg/min from OLE baseline; Kansas City Cardiomyopathy Questionnaire Clinical Summary Score (KCCQ-CSS) increased by a mean of 17.3 points over the same period. At Week 52, 79% (33/42) of patients achieved a Valsalva LVOT gradient (LVOT-G) below 30 mmHg, with 90% of patients achieving a Valsalva gradient <30 mmHg at one or more visits during the treatment period. A majority (57%) of patients ended the study on the 40 mg twice-daily (BID) starting dose.

In a separate presentation, Braveheart Bio reported new preclinical mechanistic data and ad hoc analyses of the completed Phase 2 study in non-obstructive HCM (nHCM), suggesting HRS/BHB-1893 improves diastolic function while preserving systolic function.

“These 52-week data suggested the combination of clinical effect, tolerability, and simplicity in dosing of HRS/BHB-1893, which gives us growing confidence to proceed with our clinical development in China,” said Su Zhang, Vice President and Head of Non-Oncology Clinical Development, Hengrui Pharma. “We believe that it indicates HRS/BHB-1893 will be an effective and practical option for HCM patients.”

“These data reinforce the differentiated clinical profile of HRS/BHB-1893, suggesting encouraging efficacy, safety, and ease of use over a longer course of treatment,” said Travis Murdoch, M.D., President and Chief Executive Officer, Braveheart Bio. “The study data also suggest potential reverse-remodeling. Taken together with data presented today on the mechanistic differentiation of HRS/BHB-1893 from preclinical work and the completed nHCM Phase 2 study, we believe that HRS/BHB-1893 could fundamentally address the biology of HCM, including diastolic dysfunction, across both forms of the disease.”

“HCM has long been treated as a disease of obstruction, but the biology is more fundamental, and today's data begin to reflect that reality,” said Theodore Abraham, M.D., Co-director of UCSF HCM Center of Excellence. “These results suggest that it may be possible to engage that biology directly. The evidence of attenuated rebound after 52 weeks points to a treatment that is beginning to modify the structural and biological substrate of the disease. The diastolic findings in non-obstructive patients, consistent across preclinical models and the clinic, show that the same mechanism can improve ventricular relaxation and filling that have historically constrained this class. For patients living with HCM in all its forms, that could represent a meaningfully different kind of progress.”

Long-term efficacy and safety data of HRS/BHB-1893 in patients with obstructive hypertrophic cardiomyopathy: results from the 52-week extension period

The Phase 2 study of HRS/BHB-1893 (NCT06516068) enrolled 42 adults with symptomatic oHCM. During the 12-week core period, patients were randomized across three dosing cohorts. Following the core period and a washout phase, all 42 patients entered the OLE. Patients then resumed treatment with HRS/BHB-1893 at a starting dose of 40 mg BID with opportunity for titration to 60 mg or 80 mg BID based on Valsalva LVOT-G and left ventricular ejection fraction (LVEF) criteria.

Exercise capacity and symptoms improved with extended treatment. Peak oxygen uptake (pVO2) increased by a mean of 2.7 mL/kg/min at Week 52 from OLE baseline, with 70% of patients achieving a ≥1.5 mL/kg/min improvement and 50% achieving a ≥3.0 mL/kg/min improvement. KCCQ-CSS increased by a mean of 17.3 points at Week 52 from OLE baseline, with 45% of

patients achieving a ≥20-point improvement. 60% of patients achieved New York Heart Association (NYHA) Class I after 52 weeks of treatment.

HRS/BHB-1893-treated patients showed rapid, deep, and durable reductions in LVOT-G. Responders were observed as early as Day 1. By Week 1 (before the first titration), 67% of patients achieved complete LVOT-G response (Valsalva gradient <30 mmHg). At Week 52, 79% of patients (33/42) achieved Valsalva LVOT-G <30 mmHg, and 90% of patients achieved a Valsalva gradient <30 mmHg at one or more visits during the treatment period. 57% of patients (24/42) were maintained on the 40 mg BID starting dose and an additional 19% (8/42) were maintained on 60 mg BID at end of study.

The OLE washout design enabled a post hoc analysis comparison of gradient and biomarker rebound following 12 weeks versus 52 weeks of treatment with matched drug exposure. Following the 12-week core period, LVOT-G and N-terminal pro-B-type natriuretic peptide (NT-proBNP) returned to or went above pre-treatment levels after washout. Following 52 weeks of OLE treatment, signs of reduced disease burden were observed with attenuated return of NT-proBNP and LVOT-G.

HRS/BHB-1893 was generally well tolerated across the 52-week OLE period, with no on-treatment severe or serious adverse events. All on-treatment treatment-emergent adverse events (TEAEs) were mild or moderate in severity and no adverse events led to permanent study drug discontinuation. Two patients had transient reductions in LVEF below 50% (48% and 49% site-measured) and both resumed treatment at a lower dose.

The data were presented in a late-breaking oral presentation, “Long-Term Efficacy and Safety of HRS/BHB-1893 in Patients With Obstructive Hypertrophic Cardiomyopathy: Results From the 52-Week Extension Period,” by Xueyi Wu, M.D., Fuwai Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, on Friday, November 6, 2026, at 2:15 p.m. CT.

HRS/BHB-1893 improved indices of diastolic function: evidence from in vitro studies and nHCM patients

In a second presentation, Braveheart Bio reported mechanistic and clinical evidence that HRS/BHB-1893 improves indices of diastolic function with minimal effect on systolic performance, suggesting potential to address a therapeutic gap that has limited existing therapies.

The post hoc pooled-dose analysis evaluated effects of HRS/BHB-1893 on cardiac function in HCM engineered heart tissues (EHTs) and echocardiographic parameters for 84 patients with nHCM from the completed, placebo-controlled Phase 2 HRS-1893-202 study (NCT06816251). In preclinical HCM EHTs carrying the R403Q pathogenic myosin mutation, HRS/BHB-1893 was observed to improve relaxation time well below concentrations required to meaningfully reduce systolic force.

In nHCM patients, post hoc pooled-dose analysis showed significant improvements across multiple established indices of diastolic function after 12 weeks of treatment. For septal e', 59% of treated patients exceeded the normal threshold of 7 cm/s, compared to 20% at baseline and 14% in the control group. For lateral e', 57% of treated patients exceeded the 10 cm/s threshold, compared to 21% at baseline and 18% in the control group. E/e' ratio decreased and left ventricular end-diastolic volume increased, which is consistent with a downward shift of the left ventricular pressure-volume relationship and a more compliant, distensible ventricle. Left atrial volume index decreased, reflecting the downstream benefit of improved diastolic filling. Left ventricular global longitudinal strain (LVGLS) was preserved across the treated group, with 39% of HRS/BHB-1893-treated patients versus 15% of placebo patients demonstrating an improvement in LVGLS by ≥10%.

Because patients with nHCM have no obstruction to relieve, these results point to the proposed mechanism of action of HRS/BHB-1893. Across laboratory models and patients, the molecule appears to act on the excess actomyosin cross-bridge activity that drives diastolic dysfunction in HCM, improving ventricular filling while preserving systolic function. Together, the data support the potential of HRS/BHB-1893 as a differentiated, next-generation cardiac myosin inhibitor.

The data were presented in a moderated poster session, “HRS/BHB-1893, a Next-Generation Cardiac Myosin Inhibitor, Improves Indices of Diastolic Function in Non-Obstructive Hypertrophic Cardiomyopathy: Evidence from In Vitro Studies and Patients,” by Carlos L. del Rio, Ph.D., Senior Vice President, Cardiovascular Research & Translational Science, Braveheart Bio, on Friday, November 6, 2026.

Investor Event and Webcast

Braveheart Bio will host an investor event and webcast today, November 6, 2026, at 3:35 p.m. CT (4:35 p.m. ET) to discuss these data and how they inform future clinical plans, including a planned fixed-dose Phase 2b study in oHCM (TRUEHEART-HCM). A live webcast will be available in the Investors section of the Braveheart Bio website at ir.braveheart.bio, and a replay will be archived following the event.

About Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a chronic disease estimated to affect more than 700,000 individuals in the United States, and remains an area of significant unmet medical need. HCM can lead to heart failure, stroke or sudden cardiac death, including in young adults and athletes who may often live with the disease undetected.

In HCM, overactive myosin can cause excessive contraction of the heart, resulting in the heart muscle becoming abnormally thick, particularly in the left ventricle, the heart's main pumping chamber. This can lead to symptoms like shortness of breath, chest pain, fatigue and fainting, which typically worsen with physical activity and can significantly limit patients' ability to exercise, work or

perform routine tasks.

Approximately two-thirds of patients with HCM have oHCM, where the thickened muscle creates a blockage in the heart's main pumping chamber, making it harder for blood to flow out to the body with each heartbeat. Approximately one-third of patients with HCM have non-obstructive hypertrophic cardiomyopathy (nHCM), where there is no obstruction, but the muscle is thickened, limiting proper heart function.

About HRS/BHB-1893

HRS/BHB-1893 (designated HRS-1893 by Hengrui and BHB-1893 by Braveheart) is an investigational next-generation oral small-molecule cardiac myosin inhibitor engineered for rapid onset of action, minimal impact on left ventricular ejection fraction (LVEF) and ease of use. By modulating cardiac contractility, HRS/BHB-1893 aims to address the underlying pathophysiology of hypertrophic cardiomyopathy (HCM) while maintaining cardiac output.

HRS/BHB-1893 has undergone extensive clinical development, including dose-ranging Phase 2 studies in both symptomatic obstructive HCM (oHCM) and symptomatic non-obstructive HCM (nHCM), multiple clinical pharmacology studies including a bridging study in Australia, and an ongoing Phase 3 study in oHCM in China (NCT07021976). Braveheart Bio entered into an exclusive worldwide license agreement with Hengrui Pharma for the development, manufacture, and commercialization of HRS-1893 outside of Chinese mainland, the Hong Kong SAR, the Macao SAR and Taiwan Region.

About Hengrui Pharma

Hengrui Pharma is an innovation-driven, global pharmaceutical company dedicated to the research, development and commercialization of high-quality medicines to address unmet clinical needs. Its therapeutic areas of focus include oncology, metabolic and cardiovascular diseases, immunological and respiratory diseases, and neuroscience. Driven by a patient-focused philosophy since its founding in 1970, Hengrui Pharma remains committed to advancing human health by striving to conquer diseases, improve health, and extend lives through the power of science and technology.

About Braveheart Bio

Braveheart Bio is a clinical-stage biopharmaceutical company focused on developing therapies for patients with hypertrophic cardiomyopathy (HCM) and other serious cardiovascular diseases. Its lead product candidate, BHB-1893, is a next-generation oral small-molecule cardiac myosin inhibitor (CMI) being developed for the treatment of obstructive HCM (oHCM) and non-obstructive HCM (nHCM). Braveheart's goal is to improve the treatment options for these patients by enhancing speed of onset, depth of gradient response, systolic safety and reversibility, and by reducing prescribing complexity. For more information, visit www.braveheart.bio.

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including, without limitation, statements regarding the therapeutic potential, safety, efficacy, tolerability, and dosing of HRS/BHB-1893, including its potential to support a low-monitoring dosing regime with a majority of patients served by the starting dose and potential reverse-remodeling; the potential of HRS/BHB-1893 to address the underlying biology of HCM, to improve diastolic function or to be a differentiated, next-generation cardiac myosin inhibitor across both forms of HCM; the beneficial characteristics, and the potential therapeutic effects of HRS/BHB-1893; the potential of HRS/BHB-1893 to reach a broad population of patients; and the clinical development of HRS/BHB-1893, including the ongoing Phase 3 study. Words such as “aim,” “anticipate,” “believe,” “expect,” “may,” “potential,” “suggest,” “will” and similar expressions are intended to identify forward-looking statements. These statements are based on current expectations and are subject to risks and uncertainties that could cause actual results to differ materially, including that results of earlier-stage, open-label or nonclinical studies may not be predictive of results of later or controlled clinical trials; risks related to the design, conduct and outcomes of clinical trials; the regulatory review and approval process; dependence on the license arrangement between the companies; and the other risks described in Braveheart’s filings with the U.S. Securities and Exchange Commission, including its most recent Quarterly Report on Form 10-Q and Hengrui’s public disclosures. Forward-looking statements speak only as of the date of this press release, and, except as required by law, Braveheart and Hengrui undertake no obligation to update them.

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