Hengrui Pharma and Braveheart Bio Announce Positive Phase 2 Results with HRS/BHB-1893 in Non-Obstructive Hypertrophic Cardiomyopathy

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Results with next-generation cardiac myosin inhibitor suggest a potentially disease-modifying therapy in non-obstructive hypertrophic cardiomyopathy

HRS/BHB-1893 treatment resulted in rapid and sustained reductions in key biomarkers, structural remodeling and meaningful improvements in symptoms

Safety and tolerability profile was favorable

Data highlighted in a late-breaking featured clinical research presentation at Heart Failure 2026, the annual congress of the Heart Failure Association of the European Society of Cardiology

SHANGHAI, China, and SAN FRANCISCO, May 11, 2026 — Hengrui Pharma (Hengrui) and Braveheart Bio (Braveheart) today announced results from a multi-center, randomized, double-blind, placebo-controlled Phase 2 study evaluating HRS-1893 (also known as BHB-1893), an investigational next-generation cardiac myosin inhibitor (CMI), in patients with non-obstructive hypertrophic cardiomyopathy (nHCM). HRS/BHB-1893 treatment resulted in improvements across biomarkers of cardiac wall stress and tissue injury, echocardiographic measures of diastolic function and cardiac structure, and patient-reported symptoms and exercise capacity, with a favorable tolerability profile.

“In obstructive HCM, it has been difficult to separate the direct biological effect of a therapeutic candidate from the mechanical consequence of relieving the gradient,” said Anjali Owens, M.D., Professor of Medicine at the Hospital of the University of Pennsylvania and member of Braveheart’s Clinical Advisory Board. “In non-obstructive HCM, where there is no left ventricular outflow tract gradient (LVOT-G), that confounding variable does not exist. The structural reverse remodeling toward normal observed here is a compelling early signal that HRS/BHB-1893 may be modifying the underlying disease in nHCM, in addition to helping manage the condition.”

“Non-obstructive HCM has long represented one of the most difficult challenges in cardiovascular medicine, a disease with clear pathophysiology and significant patient burden, but limited treatment options,” said Sheng Qi, M.D., Executive Director and Head of Cardiovascular, Hengrui Pharma. “The breadth of improvement we have observed here across biomarkers, cardiac structure and patient-reported outcomes, in a population with no gradient to relieve, points to a mechanism that may act directly on the disease. We look forward to advancing this program and continuing our partnership with Braveheart to bring a meaningful treatment option to patients.”

“These results offer promise thatHRS/BHB-1893 could potentially offer a meaningful therapy for nHCM patients,”said Travis Murdoch, M.D., Chief Executive Officer and President, Braveheart Bio. “The rapid and meaningful responses across diastolic function, cardiac structure, biomarkers and clinical endpoints in this placebo-controlled study suggest that we may be engaging the underlying pathophysiology in a unique and direct way. We plan to evaluate this approach at scale in an upcoming global registrational study.”

The multi-center, randomized, double-blind, placebo-controlled Phase 2 study (NCT06816251) enrolled 84adults with symptomatic nHCM. Key inclusion criteria for the study were left ventricular ejection fraction (LVEF) ≥60%, New York Heart Association (NYHA)Class II–III, maximal wall thickness of ≥15 mm (≥13 mm with family history of HCM),LVOT-G <30 mmHg, NT-proBNP >300 pg/mL and KCCQ-CSS ≥30 and ≤85.

The primary endpoint was the safety and tolerability of multiple oral doses of the intervention in patients, including the incidence and severity of adverse events and the incidence of LVEF <50%. Key secondary endpoints included biomarkers (NT-proBNP, a marker of diastolic wall stress, and cardiac troponin I, a marker of myocardial injury), patient-reported symptoms and exercise capacity (KCCQ-CSS, peak oxygen consumption [pVO_2]) and echocardiographic measures of diastolic function, cardiac structure and change from baseline in LVEF.

Patients treated with HRS/BHB-1893 reported improvements in symptoms and exercise capacity. The high-dose group showed a placebo-adjusted improvement of +5.5 points in KCCQ-CSS. 52% of high-dose patients achieved a ≥20-point improvement, the threshold for a large to very large improvement, compared to 21% in placebo. Among patients titrated to 60 mg twice daily, absolute pVO_2 change was +2.1 mL/kg/min, representing a placebo-adjusted gain of +0.9 mL/kg/min. 55%of high-dose patients achieved a ≥1.5 mL/kg/min increase from baseline.

HRS/BHB-1893 also produced improvements across important echocardiographic domains. Early myocardial relaxation velocity improved, with septal and lateral e' values moving toward normal range in the high-dose group. Left atrial volume index fell by

7.1mL/m² from baseline versus essentially no change in placebo, consistent with normalization of left atrial pressure overload. Left ventricular mass index decreased by 17.4 g/m² in the high-dose group versus an increase of 3.2 g/m² in placebo, and left ventricular wall thickness decreased by 3.3 mm versus 0.1 mm in placebo, consistent with structural reverse remodeling of the left ventricle.

HRS/BHB-1893 treatment resulted in rapid and sustained improvement in clinically relevant cardiac biomarkers: NT-proBNP geometric mean reduction (68–69%) and cardiac troponin I geometric mean reduction (55–60%), with p values less than 0.0001 versus placebo for both.

HRS/BHB-1893 was generally well tolerated. Treatment-emergent adverse events (TEAEs) were mild or moderate in severity, with no severe adverse events and no adverse events leading to temporary interruption or permanent discontinuation of HRS/BHB-1893. Serious adverse events, all assessed as unrelated to the study drug, were reported in four patients, two in the low-dose group (cellulitis, otolithiasis), one in the high-dose group (atrial fibrillation in a patient with a history of atrial fibrillation) and one in the placebo group (atrial tachycardia). Four patients (7% of those treated) had a temporary drop in LVEF (nadir between 46% and 49%) and required a dose reduction. All four returned to an LVEF above 50% after dose reduction.

Results of the study were highlighted in a late-breaking featured clinical research presentation at Heart Failure 2026, the annual congress of the Heart Failure Association of the European Society of Cardiology.

About Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a rare disease, affecting 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.

Based on recent published estimates, approximately one-half of patients with HCM have non-obstructive HCM (nHCM), where there is no obstruction but the muscle is thickened, limiting proper heart function. In nHCM, the absence of left ventricular outflow tract gradient (LVOT-G) means there is no mechanical target to relieve, making drug development in this population particularly challenging and leaving patients with limited pharmacological treatment options.

About HRS-1893/BHB-1893

HRS-1893 (also known as BHB-1893) 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-1893 aims to address the underlying pathophysiology of hypertrophic cardiomyopathy (HCM) while maintaining cardiac output.

HRS-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 Mainland China, the Hong Kong SAR, the Macao SAR and Taiwan Region.

About Hengrui Pharma

Hengrui Pharma is an innovative, 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 in1970, 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, reversibility and reducing prescribing complexity. For more information, visit www.braveheart.bio.

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