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): **July 15, 2015**

Bellerophon Therapeutics, Inc.
(Exact Name of Registrant as Specified in Charter)

Delaware	**001-36845**	**47-3116175**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

53 Frontage Road, Suite 301 **Hampton, New Jersey**	**08827**
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code: **(908) 574-4770**

(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 Instruction A.2. below):

- o Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- o Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- o Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- o Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01. Regulation FD Disclosure.

Information that the management of Bellerophon Therapeutics, Inc. intends to use from time to time during presentations to and discussions with investors, analysts and other interested parties is attached hereto as Exhibit 99.1. The information in Exhibit 99.1 shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits:

Exhibit No.	**Description**
99.1	Bellerophon Therapeutics, Inc. BCM Preview Report and Q&A (furnished and not filed for purposes of Item 7.01)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

BELLEROPHON THERAPEUTICS, INC.

Date: July 15, 2015

By: /s/ Jonathan M. Peacock

Name: Jonathan M. Peacock

Title: Chairman and Chief Executive Officer

EXHIBIT INDEX

Exhibit No.	Description
99.1	Bellerophon Therapeutics, Inc. BCM Preview Report and Q&A (furnished and not filed for purposes of Item 7.01)

BCM Preview Report
July, 2015

This is an update on our PRESERVATION I clinical trial to evaluate the effectiveness of our Bioabsorbable Cardiac Matrix (BCM) in preventing left ventricular remodeling and subsequent progression to congestive heart failure in patients who have had a serious heart attack. We will also, more briefly, provide an update on continued progress in preparing our Phase III INOpulse development program using pulsed nitric oxide to treat pulmonary arterial hypertension in community-based patients.

The PRESERVATION I trial is a double-blind placebo controlled study that has enrolled more than 300 subjects with ST-segment elevation myocardial infarctions or STEMI in the highest quartile for the severity of the heart attack. Subjects were randomized to receive BCM or saline placebo in a 2:1 ratio. Patient enrollment was completed in December of 2014 and the 6-month follow-up visit with echocardiographic examination of the heart was completed in June.

We expect to report topline results from the trial within the next few weeks and to present the findings at the European Society of Cardiology's annual meeting in London on September 1. Based upon discussions with our European notified body, we expect that a statistically significant outcome on the primary endpoint in the PRESERVATION I trial would support the registration of BCM in Europe as soon as mid-2016.

BCM is a medical device deployed as a one-time, 30-to-60 second injection that is intended to prevent left ventricular remodeling, and thereby the occurrence of congestive heart failure after a heart attack. Left ventricular remodeling is a change in the size and shape of the heart that is considered to be the initial stage of congestive heart failure. An increase in the left ventricle's volume is the hallmark sign for left ventricular remodeling.

Clinical studies conducted over the last three decades have repeatedly demonstrated that the presence and severity of increased left ventricular volume at 1-to-6 months after a heart attack is a strong predictor of poor outcomes in future years, such as sudden death due to cardiac arrest and hospitalizations for congestive heart failure. Drugs, such as ACE inhibitors, are now routinely used in the effort to prevent left ventricular enlargement after a heart attack. Reductions in the risks of poor cardiac outcomes that are achieved with these drugs are directly proportional to their mitigation of left ventricle enlargement during the first 6 months after a heart attack. In spite of the widespread use of these drugs, up to 40% of patients who have a heart attack still develop congestive heart failure within 5 years. So, significant medical need remains for new treatments to further reduce the enlargement of the left ventricle after a heart attack.

BCM is a medical device that is metabolically-inert so, if approved, we expect it could be

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combined with any of the current standard-of-care drugs for heart attack patients. It is conveniently administered by an interventional cardiologist as a one-time, 30-to-60 second injection of BCM solution using a procedure routinely performed by interventional cardiologists. A recently published open-label study of 27 heart attack patients that received BCM as well as the ongoing patient safety surveillance in the PRESERVATION I trial by an independent Data Safety Monitoring Board indicated that BCM was safely administered to and generally well tolerated by patients in these studies. Once injected into a coronary artery, BCM is taken up by the infarcted region of the heart where it encounters a high concentration of calcium and forms a hydrogel that resists enlargement of the left ventricular after a heart attack.

In the PRESERVATION I trial, the primary endpoint is enlargement in the end-diastolic volume at 6 months after heart attack in subjects who received BCM compared to those who received saline placebo. Change in end-systolic volume at 6 months after heart attack is another endpoint. Based on the historical clinical studies, we expect that a reduction in the enlargement of the left ventricle at 6 months after heart attack by BCM will translate into improved clinical outcomes in future years. The PRESERVATION I trial is powered to detect a 10 mL difference in the end-diastolic volume of a typical patient. We believe a reduction of this magnitude will translate into improved long-term survival, based on published literature. We also believe a greater reduction than 10 mL in the end-diastolic volume will result in proportionately greater improvement in long-term survival, based on published literature. Other outcomes measures in the PRESERVATION I trial include 6-minute walk distance and Kansas City Cardiomyopathy questionnaire. The sensitivity of these measures to discriminate differences between BCM and placebo in our patient population over a short period like 6 months is not known. However, clinical measures will be repeated at one year and the PRESERVATION I subjects will be followed for 5 years for major cardiac outcomes such as hospitalization for congestive heart failure or death. Based on historical studies indicating that favorable changes in left ventricular volume predict long-term clinical outcomes, we are confident that a positive outcome on the primary endpoint of end-diastolic volume at 6 months should result in positive changes in clinical outcome measures such as death or hospitalization for congestive heart failure over the full course of the trial.

Now, turning briefly to our other late stage program. We are using inhaled nitric oxide delivered using a small, highly portable pulsing device to treat pulmonary arterial hypertension in community-based patients. We have continued to make good progress in preparing a Phase III clinical trial and we are on track to enroll our first patients before the end of this year.

We have discussed with the FDA and European Medicines Agency a protocol for this phase 3 program consisting of two clinical trials with 6-minute walk distance as the primary endpoint and Time to Clinical Worsening as a key secondary. Time to Clinical Worsening data from both trials will be combined for final analysis. We are working on the details of this protocol with the FDA.

We have also completed the first round of site selection with our clinical research organization, PPD and have identified 90 potential sites worldwide for the trial.

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1) What is the natural course of cardiac remodeling in the human heart after a heart attack?

Cardiac remodeling is the result of a relative overload state that is imposed on the surviving left ventricle after a heart attack. The basic process is that the death of a segment of the LV (infarction) results in a reduced ability of the LV to eject blood when it contracts. The extra blood that is left in the LV after contraction causes the end-diastolic volume to increase, the end-diastolic pressure to rise, and the biomechanical stress on the LV wall to increase. The increase in wall stress causes infarct expansion (an increase in the surface area of the infarct) and hypertrophy in

the non-infarcted portion of the LV. Infarct expansion results in a sustained increase in LV volume, which causes the heart to have to pump harder to ejection the same volume of blood. Hypertrophy in the non-infarcted portion of the LV is the heart's attempt to compensate, but hypertrophy can result in arrhythmic foci to arise. Both infarct expansion and hypertrophy can contribute to negative feed-forward cycles ultimately resulting in heart failure.

The pace of cardiac remodeling seems to vary in different patients. In about 1/3rd of patients that suffer cardiac remodeling, most of the remodeling occurs within the first month ("early remodelers"). In another 1/3rd of patients, remodeling is relatively slow initially, but develops steadily between 1 month and 6 month post-MI ("late remodelers"). In the final 1/3rd of patients with cardiac remodeling, the heart steadily expands in volume from the time of the heart attack through 6 months ("progressive remodelers"). Long-term outcome, measured as cardiac event-free survival, is directly related to the extent of remodeling at 6 months, but independent of whether remodeling is early, late, or progressive. [Bolognese, 2002: Circulation 106:2351]

a. What percentage of MI patients does this occur in?

Various papers report that the percentage of patients that develop cardiac remodeling after an anterior AMI ranges from about 30-60% [Bolognese, 2002: Circulation 106:2351; Farah, 2012: Medical Science Monitor 18:CR276]. Overall, 35-40% of patients with AMI develop CHF within 5 years.

The degree of cardiac remodeling that occurs in a susceptible patient correlates with larger infarct size, anterior location of the infarct, and transmurality of the infarct (spanning from inner to outer wall of the heart).

b. How does this impact clinical outcomes?

Poor long-term outcome is directly related the presence and extent of cardiac remodeling after a heart attack. Increased left ventricular volume (the hallmark of cardiac remodeling) measured between 1 month and 6 months after heart attack is a strong, independent predictor of increased risk for death, developing congestive heart

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failure, or having a second heart attack over the next 5-7 years. [White, 1987: Circulation 76:44; Kramer, 2010: JACC 56:392]

c. Is there anything patients are currently treated with to prevent this?

The current standard-of-care to prevent cardiac remodeling after a heart attack is chronic treatment with a beta blocker, ACE inhibitor, or angiotensin receptor blocker (ARB). These drugs reduce the stress in the wall of the left ventricle by lowering the blood pressure. Despite the longstanding standard-of care treatments, incidence of cardiac remodeling, as noted above, remains high.. BCM is deployed as a one-time, 30-to-60 second injection *to prevent cardiac remodeling. PRESERVATION I subjects received standard-of-care treatments in addition to receiving BCM or placebo. Thus, the effects of BCM in the PRESERVATION I study will be on-top of standard-of-care.*

2) Remind us of the scientific rationale of how BCM, sodium alginate and calcium gluconate, could improve post MI

BCM forms a flexible hydrogel within the infarct. The hydrogel has mechanical properties similar to normal cardiac extracellular matrix. This reinforcement of the infarct region occurs at a time when the cardiac myocytes are undergoing necrosis and the extracellular matrix is being liquefied by enzymes activated by the heart attack. Without reinforcement the infarct region would bulge outward during contraction, increasing regional wall stress, and expanding the infarct to involve a larger area of the heart wall. Based on animal studies, BCM decreases the wall stress in the infarct region, indicating that the abnormal mechanical properties of the infarct region are corrected by BCM's reinforcement of the softened infarct region after a heart attack.

BCM forms a hydrogel in the infarct because of the confluence of 3 events: (1) the interventional cardiologist injects BCM into the coronary artery involved in the heart attack once blood flow has been restored; (2) the coronary capillaries are abnormally leaky in the infarct region after the heart attack which allows high molecular weight alginate to pass from the bloodstream into the tissue; (3) the extracellular calcium concentration in the infarct region rises to an abnormally high level after the heart attack so that the alginate molecules in BCM that have been partially crosslinked by calcium gluconate react with the elevated tissue calcium to fully crosslink into a hydrogel.

a. What consistency does the matrix reach?

The hydrogel that forms in the infarct has a mix of stiffness and elasticity that is similar to cardiac extracellular matrix. The stiffness provides reinforcement to the infarct region when the heart contracts. The elasticity allows the heart to relax when it fills with blood between heart beats.

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b. Are there any reasons for pockets of high calcium in the heart that is not related to post-MI tissue damage?

Calcium deposits can occur in atherosclerotic plaque and in the heart wall where old infarcts have occurred. This calcium is NOT in solution and, therefore, not available to react with alginate. The free calcium concentration in the heart tissue is tightly regulated by a system of energy-dependent calcium transporter molecules. Absent hyperparathyroidism, which causes high calcium levels throughout the body, there can be no substantial elevation in free calcium in the heart without an ischemic event, such as a heart attack.

c. Is there a chance the matrix can inappropriately form?

No. BCM forms a hydrogel only in the presence of high calcium levels. Calcium levels in the blood and tissues is tightly regulated except as described above. The combination of an ischemic injury, such as a heart attack, and a high concentration of BCM, such as with intra-coronary artery injection are required for hydrogel formation.

BCM has been safely deployed in GLP animal studies, in a 27-patient pilot study with 5 years of follow-up and in the PRESERVATION I study, where safety surveillance has been performed by an independent Data Safety Monitoring Board — these findings provide robust evidence for the safety of BCM under the conditions of clinical use.

3) Could you review for us the design of the Phase 3a trial underway

a. Number of patients

303 subjects were randomized.

b. Inclusion and exclusion criteria of note?

All *eligibility criteria are described in the study posting on clinicaltrial.gov: "https://clinicaltrials.gov/ct2/show/NCT01226563?term=preservation&recr=Active%2C+not+recruiting&lead=bellerophon&rank=1"*

To be included, subjects had to have a large STEMI, successful PCI with stent within 48hrs of onset of symptoms, and good coronary flow at time of deployment. Subjects were excluded if they experienced shock during deployment, had severe renal insufficiency, presented with inadequate echo windows, or suffered uncontrolled ventricular arrhythmias.

c. When is the BCM administered during the post-MI paradigm?

Study device (BCM or placebo) was deployed 2-5 days after PCI and stenting.

i. Will this be the real world administration?

Yes.

ii. Could the treatment be given during the first admission to the cath lab?

According to the preclinical studies, yes.

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d. What is the primary endpoint of the study?

The anatomic measurements of left ventricular end diastolic volume index (LVEDVI) (Core Lab) at 6 months after injection of BCM will be the primary effectiveness endpoint.

i. How are they measured?

The anatomical endpoints of LVEDVI, left ventricular end-systolic volume index, and left ventricular ejection fraction are measured by echocardiogram. All echocardiograms are centrally read by Duke Clinical Research Institute central echo lab.

ii. How are they adjudicated?

As all echocardiographic measurements are centrally read, no adjudication is required.

iii. What is the powering for the primary endpoint?

80% of power to detect a treatment difference of 5 mL/m^2 under a significance level of 0.05.

b. What are the key secondary endpoints of the study?

i. Patient reported outcomes (PROs) using Kansas City Cardiomyopathy Questionnaire (KCCQ) scores (summary score)

ii. Six minute walk test (6MWT) i.e., distance walked in 6 minutes

iii. New York Heart Association (NYHA) functional classification (Physician reported)

iv. Time to cardiovascular death or non-fatal heart failure events or cardiovascular hospitalizations adjudicated by a Clinical Events Committee (CEC)

v. Time to first re-hospitalization due to any cardiovascular event

vi. What is the most important secondary endpoint in your perspective?

6MWT or NYHA class

c. What were the safety observations seen either in preclinical studies or in the pilot clinical study?

i. BCM was well tolerated by pigs when deployed after myocardial infarction. Of note is the absence of BCM-related histopathological findings at any time point in the acute and chronic studies irrespective of deployment in a first or second procedure.

ii. *During the Pilot Clinical Study, no unexpected adverse events were noticed, and the side effect profile was considered consistent with a post-MI population" (see published literature: Intracoronary Delivery of Injectable Bioabsorbable Scaffold (IK-5001) to Treat Left Ventricular Remodeling After ST-Elevation*

Myocardial Infarction: A First-in-Man Study Norbert Frey, et al. Circ Cardiovasc Intern. published online October 28, 2014; Print ISSN: 1941-7640. Online ISSN: 1941-7632). In addition, no long-term side effects emerged during the 5 year follow-up.

iii. *PRESERVATION I was monitored by an independent Data Safety Monitoring Board and was waived forward to completion of recruitment.*

2) What constitutes success in the primary endpoint of the study?

a. What is the minimal clinically important difference

Increased LV volume is predictive of future major adverse cardiac events with no apparent minimum threshold below which there is no increase in relative risk (White, 1987: Circulation 106:2351). In AMI patients at risk of LV dilation and in chronic heart failure (CHF) patients with established LV dilation, reducing the extent of LV dilation or partially reversing existing LV dilation accrues significant long-term risk reduction for major adverse cardiac events. Risk reduction in these patients is directly related to the magnitude of the reduction in LV dilation. A reduction in LV volume of 10 mL in a typical patient results in a statistically significant risk reduction of 10% in major adverse cardiac events over the next 2-7 years (White, 1987: Circulation 106:2351; Kramer, 2010: JACC 56:392).

b. What clinical outcomes benefit is this thought to translate to?

Avoidance of major adverse cardiac events, defined as sudden death or hospitalization for congestive heart failure or myocardial infarction.

i. What is the extent of the literature that supports this clinical benefit?

- *Three decades of data demonstrate that enlarged left ventricular volume is directly related to increased incidence of adverse cardiovascular outcomes, e.g., heart failure and cardiac death.*
  - *White, 1987; Circulation 76:44*
  - *Bologonese, 2002; Circulation 106:2351*
  - *Konstam, 2011; Journal of the American College of Cardiology: Cardiovascular Imaging 4:98*
- *Left ventricular remodeling remains a problem in the modern era*
  - *White, 2005; Circulation 112:3391*
  - *Savoye, 2006: Journal of Cardiology 98:1144*
  - *Farah, 2012: Medical Science Monitor 18:CR276*
- *Lowering LV dilation by 10 mL in a typical patient predicts a ≈10% risk reduction over the next 2-7 years;*

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greater lowering of LV dilation results in even larger reductions in long-term risks

- *Kramer, 2010: Journal of the American College of Cardiology 56:392*
- *Konstam, 2003; Journal of Cardiac Failure 9:350*

c. Is there any potential responder analysis that would be meaningful?

Based on literature, there is no known cut-off, and there seems to be a continuum of response.

3) If the trial is successful what is timing of next steps:

a. European filing?

i. What are timelines?

We would expect to file in the first half of 2016.

ii. Thoughts on pricing/payor negotiations?

We would expect to start with the bigger European markets where we have strong KOL support (Germany, France, Spain).

b. US Phase 3 trial?

i. Current thoughts on design?

The FDA has indicated that they will be looking for a composite endpoint of an anatomical measure, a functional measure and a patient reported outcome. Depending on the Preservation 1 data we will also explore the newly finalized Early Approval Pathway.

ii. Potential start and time to topline data?

We would expect to start early 2016 and estimate having top line data around mid 2018.

4) If this threshold effect is reached, what are BLPH's current thoughts on market opportunity?

a. Who would be the most appropriate patients?

Patients who have suffered large STEMI's who are at risk of left ventricular remodeling which typically leads to congestive heart failure. Many cardiologists believe that these patients can be recognized within the first few hours after presenting.

b. Where would they be treated with drug?

In the cardiac catheterization laboratories within hospitals.

c. Which doctors are the decision makers for BCM treatment?

Interventional Cardiologists.

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d. What sort of marketing force could address this?

Similar to other cardiac Cath Lab focused therapies e.g., Angiomax.

e. Will the therapy need to US hospital P&T committee approval?

Yes. However available DRG reimbursement codes along with the New Technology Add on Payments and the novel nature of the therapy should provide adequate coverage.

f. What are thoughts on partnership vs self-commercialization?

i. For Europe?

This is a concentrated target physician population. For example,

The German Cardiac Society (DGK) has accredited 136 Chest Pain Units (CPU) which are the key hospitals we would target. These CPUs are accredited based on having a heart catheter lab available in the same hospital 24/7 and an ER lead by a cardiologist. (Source: DGK-accredited CPUs: http://cpu.dgk.org/index.php?id=109). In the larger European markets our plan would be to commercialize independently

ii. For other geographies?

For other ex US geographies we would likely seek a partner.

iii. For US

For the same reasons as outlined above, we would expect to commercialize independently in the US. In the US, there are ~1,500 to 1,750 PCI centers or cardiac Cath Labs which covers the full universe of our potential customers.

(Source: Journal of American Heart Association, 2013 vol. 2; e00370).

5) Quick update on the PAH Phase 3 program

a. Free form update, BLPH prerogative and as time permits.

This is covered in our summary transcript.
