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MYOKARDIA, INC.

FREE WRITING PROSPECTUS

***The following articles were published by the identified publications on August 7 and 8, 2017. Myokardia, Inc. did not prepare or review these articles in advance of their publication. These articles are being filed because of the timing of their appearance and because they contain statements attributed to Tassos Gianakakos, the President and Chief Executive Officer of Myokardia and Marc Semigran, its Chief Medical Officer. Myokardia does not endorse any of the opinions expressed in these articles other than the opinions of Mr. Gianakakos and Dr. Semigran as expressly quoted in the articles. Certain statements contained in the articles are corrected, clarified or caveated by bracketed language that follows each such statement.***

***Some of the statements that the articles attribute to Myokardia are forward-looking statements within the meaning of the safe harbor provisions of Section 27A of the Securities Act of 1933, as amended (the "Securities Act"), and within the meaning of Section 21E of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), that are subject to the "safe harbor" created by those sections. These statements are subject to risks and uncertainties that could cause actual results and events to differ materially from those anticipated, including risks and uncertainties related to the financial markets. Reference should be made to Myokardia's Annual Report on Form 10-K for the year ended December 31, 2016 under the heading "Risk Factors" for a more detailed description of such factors, as well as the Company's subsequent quarterly reports on Form 10-Q. Readers are cautioned not to place undue reliance on these forward-looking statements that speak only as of the date on which such statement was made. Myokardia undertakes no obligation to update publicly any forward-looking statements to reflect new information, events, or circumstances after the date of this release except as required by law.***

The following is a reproduction of the original articles:

**Article 1 – Published by BioWorld on August 8, 2017**

'Opportunity for real disease interruption'

Myokardia blasts through ceiling with mavacamten findings in oHCM.

*By Marie Powers, New Editor*

For decades, the biopharma industry has struggled to develop "game-changing" therapies in heart failure, according to Tassos Gianakakos, CEO of Myokardia Inc.

"We've not been able to get to what's causing the disease," Gianakakos told BioWorld. Instead, "we get the therapies that are palliative in nature, dealing with compensatory effects. It's time for us to start making some of the breakthroughs in cardiovascular disease that we're seeing in other disease areas, like oncology."

Launched by Third Rock Ventures in 2012, Myokardia has been plying a precision medicine approach to advance targeted therapies in cardiovascular disease, beginning with rare heritable cardiomyopathies. On Monday, the South San Francisco-based company showed in resounding fashion that it's on the right track, reporting top-line data from the first patient cohort of the ongoing phase II PIONEER-HCM study of lead candidate, mavacamten (formerly MYK-461), in the lead indication of symptomatic, obstructive hypertrophic cardiomyopathy (oHCM).

The database was tiny – only 10 evaluable patients – but the study in the orphan indication met the primary endpoint of change in post-exercise peak left ventricular outflow tract (LVOT) gradient along with key secondary endpoints, including peak oxygen consumption (pVO2), with a safety profile deemed acceptable by the company. The findings, if borne out in additional studies, could put Myokardia in the driver's seat in bringing to market the first therapy to target cardiac hypercontractility, the cause of oHCM.

***[CAUTIONARY STATEMENT: The preceding paragraph contains forward-looking statements.]***

"We believe we're getting at the underlying cause, with the opportunity for real disease interruption and potential reversal," Gianakakos said.

The open-label PIONEER-HCM study was designed to investigate the efficacy, safety, pharmacokinetics, pharmacodynamics and tolerability, enrolling oHCM patients with left ventricular ejection fraction (LVEF) □ 55 percent, LVOT gradient (resting gradient □ 30 mmHg, post-exercise peak LVOT gradient □ 50 mmHg) and New York Heart Association (NYHA) class □ II. Participants were treated with mavacamten for 12 weeks, followed by a four-week washout phase. In addition to the primary endpoint of change in post-exercise peak LVOT gradient from baseline to week 12, additional endpoints include change from baseline to week 12 in pVO2; VE/VCO2; NYHA class; N-terminal pro b-type natriuretic peptide, or NTproBNP; rest and exercise LVEF; and dyspnea score. Safety endpoints include treatment-related adverse events (AEs) and serious AEs and changes from baseline in laboratory test results, vital signs and electrocardiograms.

Eleven patients enrolled in the first cohort, and 10 completed the study. A statistically significant improvement was observed in LVOT gradient (p=0.002), with the 10 participants achieving a reduction in post-exercise peak LVOT gradient from a baseline mean of 125 mmHg. In eight of the 10, the post-exercise peak LVOT gradient was reduced below the diagnostic threshold for oHCM (≤30 mmHg); measurements for the other two patients fell below 50 mmHg.

Clinically meaningful improvements (≤30 mmHg) in resting LVOT gradient were observed as early as week two in nine of the 10 patients. Clinically and statistically significant improvements also were observed in pVO2 (p=0.004).

The study also included an exploratory endpoint of improvements from baseline with respect to NYHA functional classification, the most commonly cited classification system for

patients with heart failure, based on clinical severity and prognosis. In PIONEER-HCM, improvements from baseline by at least one class were observed at week 12 (p=0.016) in seven patients, with two of the patients improving by two classes. Mavacamten, an oral allosteric modulator of cardiac myosin, was generally well-tolerated, according to Myokardia. The single patient who discontinued the study had a history of paroxysmal atrial fibrillation and experienced a serious AE. As a condition to participate in the study, the patient had discontinued background beta blocker and disopyramide therapy, both indicated to manage atrial fibrillation. During the study, the patient had a recurrent episode of atrial fibrillation, and cardioversion was used to reset the heart's rhythm. The patient subsequently had another episode of atrial fibrillation, was hospitalized and was successfully treated with antiarrhythmic therapy. However, the patient then elected to stop study drug at week four.

Although Myokardia did not disclose details, the company said other AEs were mild to moderate. Most were deemed unrelated to study drug.

After reviewing safety data, an independent data monitoring committee recommended continuation of the study, which has fully enrolled the low-dose patient cohort. The company added the second cohort to explore lower daily doses of mavacamten, given marked improvement seen within two weeks of dosing in the initial cohort. Additionally, the second group of patients was not required to discontinue beta blocker therapy prior to enrollment. Myokardia did not disclose the number of patients remaining in the trial but expects to report top-line data from the second patient cohort in the first quarter of 2018. [***CAUTIONARY STATEMENT: The preceding sentence contains forward-looking statements.]***

'We're gaining more and more confidence'

Lead investigator Stephen Heitner, director of the HCM Clinic at Oregon Health and Science University's Knight Cardiovascular Institute, plans to present additional details from the first cohort next month at the Heart Failure Society of America's annual scientific meeting in Dallas.

But company officials could barely contain their excitement. Marc Semigran, who joined Myokardia in December as chief medical officer, said the findings "were more than I could have imagined" in a relatively young patient population that he called "severely compromised." With mavacamten, "we have the possibility of offering them a therapy that will really help to improve their lives," he told BioWorld. ***[CAUTIONARY STATEMENT: The data from the trial is from a small sample of 10 patients, and is not indicative of future results in ongoing or planned trials. The preceding paragraph contains forward-looking statements.]***

By "following the science" and using a genetic underpinning to develop a small molecule that targets the hypercontractility abnormality of oHCM, Myokardia's thesis "is bearing out in the physiologies and the functional and exercise capacities of patients," Semigran added.

Myokardia already requested an end-of-phase II meeting with the FDA and plans to ask for its next study, EXPLORER-HCM, to be designated as pivotal, using pVO2 as the primary endpoint. The trial design is expected to use similar inclusion and exclusion criteria to those for PIONEER-HCM, with planned enrollment of 200 to 250 patients expected to initiate by year-end. ***[CAUTIONARY STATEMENT: The preceding paragraph contains forward-looking statements.]***

The potential import of the data wasn't lost on investors, who powered the company's shares (NASDAQ:MYOK) to a historic high of $32.30. With nearly 8.7 million shares traded, or 52 times the company's three-month moving average, the stock closed at $31.45 for a gain of $14.30, or 83.4 percent. Following its IPO in October 2015, in which the company raised $50.4 million, the previous high mark for the shares was $22.83 on Aug. 24, 2016. (See BioWorld Today, Sept. 30, 2015.)

Although the company reported cash and equivalents of $117.3 million at June 30 – projected to see it through 2018 ***[CLARIFICATION: Based on its current operating plans, the Company expects that its cash, cash equivalents and investments as of June 30, 2017, together with anticipated payments its collaboration agreement with Sanofi, will enable the Company to fund its anticipated operating expenses and capital expenditure requirements at least into 2019.]*** – Myokardia disclosed late Monday that it will pursue an underwritten public offering of 3.5 million shares that, at Monday's closing price, would generate approximately $110 million. The company plans to grant underwriters a 30-day option to purchase another 525,000 common shares. J.P. Morgan Securities LLC and Cowen and Co. LLC are joint bookrunners, with BMO Capital Markets Corp. as an additional bookrunner.

Myokardia also has a partnership with Sanofi SA, inked in 2014, that called for the Paris-based pharma to pay $200 million to advance Myokardia's work in HCM and in dilated cardiomyopathy (DCM) in the form of equity investments, milestone payments and R&D fees, including $45 million delivered as an up-front licensing fee and initial equity investment. (See BioWorld Today, Sept. 18, 2014.)

Myokardia is leading initial research and global development activities of the partnered assets and will direct global development and U.S. commercial activities for mavacamten in oHCM and a follow-on program in non-obstructive HCM – an indication where it will begin a phase II study by year-end. Sanofi, which made the final $70 million payment at the end of 2016 and opted to remain in the partnership ***[CORRECTION: Sanofi made a $25.0 million milestone payment for the submission of an investigational new drug application ("IND") for MYK-491 with the FDA in November 2016, and a $45.0 million continuation payment in January 2017 in connection with its decision to continue the collaboration.]***, Gianakakos said, is funding half of the remaining R&D expenses. Following phase IIa, the pharma will pick up 100 percent of expenses for mavacamten, where it also holds ex-U.S. regulatory and commercial rights for the two HCM programs. ***[CORRECTION: Sanofi and MyoKardia will split expenses for mavacamten 50/50 after Phase IIa. Sanofi will fund 100% of expenses for MYK-491 after Phase IIa, for which it has worldwide regulatory and commercial rights in DCM.]***

Sanofi also has the option to co-promote in the U.S. for potential expanded cardiovascular indications outside the genetically targeted indications in either HCM program. At the time the partnership was formed, Myokardia hadn't even disclosed a lead compound, let alone a pipeline, but the company now has a handful of assets – three as part of the Sanofi research collaboration and two unpartnered programs. Also in the clinic is the myosin modulator MYK-491, targeting DCM. A phase I study in healthy volunteers is expected to read out in the

second half of this year. ***[CLARIFICATION: Topline data from this study is expected to be announced in the third quarter of 2017.]*** Sanofi holds global development and commercialization rights for that product, with Myokardia retaining the option to co-promote in the U.S.

HCM and DCM are types of heritable heart diseases caused by mutations in the genes of the proteins that are primarily responsible for the contraction of the heart muscle. HCM, which results in abnormal thickening of the left ventricle, is the leading cause of sudden cardiac death in young adults. DCM, which results in thinning of the heart muscle and weakened output, is the leading genetic illness leading to heart transplantation. Together, the two cardiomyopathies are believed to affect about 1 million children and adults in the U.S., according to Gianakakos.

"Myokardia's approach has been to dive deep into the disease biology and try to understand, in subgroups of these patients with heart failure, where we can address the underlying causes," he explained. With no approved drugs in HCM or DCM, patients with the conditions tend to progress until surgery or transplant become their only treatment options.

In oHCM, "we're showing, for the first time ever, that with a pill you can actually alleviate this obstruction," Gianakakos said. "Clinicians will tell you that if you can alleviate that obstruction, you can help these folks feel and function better. You've got lower pressures in the heart, you've got more blood flowing to the rest of the body and all of these good things start to happen." The company's entire pipeline is informed by the findings in PIONEER-HCM, Gianakakos said.

"We are now starting to see this body of evidence around our particular approach – this targeting approach to offset the effect of these mutations – so we're gaining more and more confidence in that," he said. "In that context, we're learning a lot about the basics of targeting the heart muscle in a very specific way and tuning the drugs to offset the negative effects of these mutations."

Like investors, analysts were wowed. In her earnings update, Cowen and Co. analyst Ritu Baral said the PIONEER-HCM "completely" surpassed expectations by meeting not only the primary endpoint but also the pVO2 secondary endpoint – the FDA's desired pivotal endpoint – with statistical significance. Baral doubled her price target on the company's shares, to $56, based on increased probabilities of approval for mavacamten, citing updated peak sales estimates of $1.2 billion and $793 million, respectively, for oHCM in the U.S. and EU.

Credit Suisse analyst Vamil Divan also hiked his price target on Myokardia to $45 from $25. Despite the smallish study, Divan was assuaged by the consistency of the findings, noting that the top-line data offered "added comfort on the emerging profile of mavacamten (MYK-461) and helps to de-risk the overall MYOK story. We raise our probability of success on mavacamten to 75 percent (from 40 percent)."

**Article 2 – Published by Endpoints on August 7, 2017**

A small, groundbreaking cardio study pushes MyoKardia to a pivotal showdown — shares soar

*By John Carroll*

MyoKardia $MYOK has come up with the proof-of-concept efficacy data that the South San Francisco-based biotech was looking for in its small but potentially groundbreaking cardio study. And now it will explore bypassing one element in its development plan and see whether the FDA will sign up on a straight shot at a pivotal study that could point the company down a straight path to the marketplace. ***[CAUTIONARY STATEMENT: The preceding paragraph contains forward-looking statements.]***

MyoKardia's shares rocketed up 81% as investors took a look at the numbers and its plans for its drug mavacamten.

The field is currently dominated by a few major drugs looking to influence the course of heart disease for large numbers of patients. While raising the bar on safety and efficacy in heart disease is an obstacle course rarely navigated by anyone outside of giant pharma, this biotech is following a game-changing molecular strategy with a rifle-shot approach at disease.

MyoKardia's initial focus on heart disease centers on an ailment called obstructive hypertrophic cardiomyopathy, where a mutation in heart proteins forces the heart to squeeze more, thickening heart muscles and creating a cascade of effects and symptoms that can lead to afibrillation and death.

"By targeting the motor protein in the heart, offsetting the mutation, you're bringing the force of the squeeze back to normal," MyoKardia CEO Tassos Gianakakos tells me. "The heart relaxes, that obstruction moves out of the way… By dampening the mutational effect, the heart remodels. We'd love to see that heart look like a normal heart."

Outcomes in the field take a fortune to nail down, but MyoKardia's concentrated on post-exercise peak left ventricular outflow tract, or LVOT, gradient from baseline to week 12, in 11 patients who were recruited for the study. One of those patients was forced out due to episodes of atrial fibrillation possibly linked to the requirement that the patients in the first cohort wash out beta blockers that are used to keep the condition

But for the 10 who completed the dosing, the baseline measure of 124.9 plunged to 18.9 — below the diagnostic threshold for the disease in 8 of 10 patients — while peak VO2 dropped more than the 10% to 15% level than the biotech had told analysts to watch out for.

"While it is 10 patients," notes the CEO, "the treatment is massive." ***[CAUTIONARY STATEMENT: The data from the trial is from a small sample of 10 patients, and is not indicative of future results in ongoing or planned trials.]***

As a result of the data, and the efficacy on peak VO2, which is the planned pivotal endpoint, Gianakakos is now planning to meet with the FDA and see if he can bypass a Phase IIb and go straight to a registration study, cutting as much as 18 months out of the development timeline. ***[CAUTIONARY STATEMENT: The preceding paragraph contains forward-looking statements.]***

I've covered MyoKardia right from the Series A with an idea of starting a biotech that would break things down in cardio R&D on a genetic basis. Gianakakos estimates that there are some 400,000 patients with this particular condition, about 65,000 of whom are symptomatic of the disease. That's the kind of target that a small, focused biotech company can hit with a small sales force. And he believes that if the FDA signs off on his pivotal plans, he can get started later this year on a 2-year effort. ***[CAUTIONARY STATEMENT: The preceding paragraph contains forward-looking statements.]***

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**Article 3 – Published by STAT on August 7, 2017**

MyoKardia drug improves blood flow in patients with serious, inherited form of heart disease

*By Adam Feuerstein*

MyoKardia announced Monday that its experimental drug mavacamten demonstrated statistically significant improvements in blood flow and aerobic capacity in patients with an inherited form of heart disease in a small, mid-stage clinical trial.

Eight of the 10 patients who completed the study achieved improvement in blood flow to a level where they no longer met the threshold for being diagnosed with their disease — obstructive hypertrophic cardiomyopathy, or HCM.

Based on these results, MyoKardia plans to start a phase 3 clinical trial of mavacamtem by the end of the year, pending discussions with the Food and Drug Administration. ***[CAUTIONARY STATEMENT: The preceding paragraph contains forward-looking statements.]***

HCM is a chronic, genetically driven disease that causes the muscle cells in the heart's left pumping chamber to contract excessively, thicken, and obstruct blood flow. Most patients progress slowly towards disability and heart failure.

HCM is also the most common cause of sudden cardiac death in young people, particularly athletes. In 1993, Boston Celtics star Reggie Lewis, 27, collapsed and died from sudden cardiac arrest that was later attributed to HCM.

MyoKardia estimates there are approximately 630,000 patients in the U.S. who carry the genetic mutation that causes HCM. Initially, mavacamten is being targeted at approximately 66,000 patients with the most severe, symptomatic form of obstructive HCM. The company is also studying the drug to treat patients with less advanced forms of HCM.

The phase 2 study enrolled 11 patients with symptomatic, obstructive HCM. All patients were treated with a once-daily mavacamten pill for 12 weeks. Ten of the 11 patients completed the study. After 12 weeks, the mean change in post-exercise left ventricular outflow tract gradient, a measure of obstruction in blood flow pumped out of the heart, fell by over 80 percent from baseline. This was the primary endpoint of the study, and the change from baseline to 12 weeks was statistically significant.

"This is a most gratifying result in what is a really difficult disease," said MyoKardia CEO Tassos Gianakakos. "Every patient responded with a massive drop in obstruction and eight of the 10 patients were subclinical, meaning their disease was essentially completely gone. The remaining two patients still had strong reductions below a level where they're often referred to surgery."

The patients treated with mavacamten also showed a mean 3.5-unit increase — nearly 19 percent — in peak V02, a measure of improved aerobic efficiency. This is an important secondary and functional endpoint in the phase 2 study because MyoKardia expects it to serve as the primary endpoint of the planned phase 3 study. ***[CAUTIONARY STATEMENT: The preceding paragraph contains forward-looking statements.]***

The one patient who did not complete the study had a history of serious heart palpitations which returned part way through the study, requiring treatment in the hospital. As a result, the patient dropped out of the mavacamten study after four weeks. All other adverse events reported in the study were considered mild to moderate and none was attributed to mavacamten, MyoKardia said.

MyoKardia, which is based in the Bay Area, is developing mavacamten under a partnership with Sanofi. MyoKardia retains full development and commercialization rights to the drug in the U.S. Sanofi is in charge of getting the drug approved outside the U.S and will market it there, paying a royalty on sales back to MyoKardia.

A full presentation of the mavacamten phase 2 study will be made at the Heart Failure Society's annual meeting on Sept. 18.

MyoKardia shares closed Friday at $17.15, up 32 percent for the year.