UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, DC 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): February 24, 2015

CymaBay Therapeutics, Inc.
(Exact name of Registrant as specified in its charter)

Delaware	**001-36500**	**94-3103561**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

7999 Gateway Blvd., Suite 130
Newark, CA 94560
(Address of principal executive offices)

(510) 293-8800
(Registrant's telephone number, including area code)

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):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 8.01 Other Events.

On February 24, 2015, CymaBay Therapeutics, Inc. (the "Company") held a conference call to discuss the results of the Company's Phase 2b clinical study of its lead product candidate arhalofenate for the treatment of gout. A copy of the transcript from the conference call (the "Transcript") is attached here as Exhibit 99.1. The Transcript has been selectively edited to facilitate the understanding of the information communicated during the conference call.

In addition, the Company issued a press release announcing the results of its Phase 2b clinical study of its lead product candidate arhalofenate for the treatment of gout. The full text of the press release issued in connection with the announcement is attached as Exhibit 99.2 to this report.

Item 9.01. Financial Statements and Exhibits.

Exhibit	Description
99.1.	Phase 2b Clinical Study of Arhalofenate Transcript, dated February 24, 2015
99.2.	Press Release issued on February 24, 2015

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.

CymaBay Therapeutics, Inc.

By: /s/ Sujal Shah
Name: Sujal Shah
Title: Chief Financial Officer

Dated: March 2, 2015

EXHIBIT INDEX

Exhibit	Description
99.1.	Phase 2b Clinical Study of Arhalofenate Transcript, dated February 24, 2015
99.2.	Press Release issued on February 24, 2015

Exhibit 99.1

Forward Looking Statements

This transcript, including those statements regarding the potential of arhalofenate to treat gout, the therapeutic and commercial potential of arhalofenate and the anticipated timing and therapeutic and commercial potential of other product candidates of CymaBay Therapeutics, Inc., or CymaBay, are forward looking statements that are subject to risks and uncertainties. Actual results and the timing of events regarding the further development of arhalofenate could differ materially from those anticipated in such forward-looking statements as a result of risks and uncertainties, which include, without limitation, risks related to: the possibility that subsequent analyses of the data disclosed below may lead to different (including less favorable) interpretations of the results than the analyses conducted to date or may identify important implications of the Phase 2b study that are not reflected in these statements, or be subject to differing interpretations by any regulatory agency; the success, cost and timing of any of CymaBay's product development activities; any delays or inability to obtain or maintain regulatory approval of CymaBay's product candidates in the United States or worldwide; the ability of CymaBay to attract funding partners or collaborators with development, regulatory and commercialization expertise; the ability of CymaBay to obtain sufficient financing to complete development, regulatory approval and commercialization of its product candidates in the United States and worldwide; and the market potential for CymaBay's product candidates. Additional risks relating to CymaBay are contained in CymaBay's Quarterly Report on Form 10-Q, filed with the Securities and Exchange Commission on November 14, 2014. CymaBay disclaims any obligation to update these forward-looking statements except as required by law.

Mr. Sujal Shah: Thank you and good morning, everyone.

Welcome to CymaBay's conference call to discuss the results of the arhalofenate Phase 2b clinical study.

This morning we issued a press release, which outlines the topline results. The release is available on our website at www.cymabay.com under the Investors tab.

Joining me on the call today is Harold Van Wart, President and Chief Executive Officer of CymaBay; Pol Boudes, Chief Medical Officer; and Chuck McWherter, Chief Scientific Officer.

Before, I turn the call over back to Hal, I would like to remind everyone that statements made during this conference call relating to CymaBay's expected future performance, future business prospects, or future events, or plans may include forward-looking statement as defined under the Private Securities Litigation Reform Act of 1995. All such forward-looking statements are intended to be subject to the Safe Harbor protection provided by the Reform Act. Actual outcomes and results could differ materially from those forecast due to the impact of many factors beyond the control of CymaBay.

CymaBay expressly disclaims any duty to provide updates to its forward-looking statements whether as a result of new information, future events or otherwise. Participants are directed to the risk factors set forth in CymaBay's public SEC filings.

Hal?

Mr. Harold Van Wart: Thank you, Sujal, and good morning, everyone. Thanks for joining us on the call today.

If possible, please refer to the table from our press release during this call to be able to see the numbers that we'll be referring to.

Overall, we are very excited about the results in this study, because they support the dual action of arhalofenate, which both lowers serum uric acid and reduces flares, which we believe constitutes a new class of gout therapy, which we are referring to as Urate Lowering Anti-Flare Therapy, or ULAFT.

Now, there were two key goals for this study, one was to demonstrate that arhalofenate produces statistically significant and medically relevant reductions in gout flares while at the same time lowering serum uric acid. To our knowledge, no other urate-lowering therapy, or ULT, has ever demonstrated both of these properties in the same molecule. Secondly, we wanted to confirm the hypothesis that the anti-flare activity is present when patients are not on background colchicine therapy, since in all of our earlier studies patients had received colchicine prophylaxis.

The Phase 2b study was a randomized, double blind placebo and active controlled 12 week study consisting of five arms; including placebo, arhalofenate at 600 or 800 milligrams, allopurinol 300 milligrams, and finally allopurinol 300 milligrams combined with colchicine. All patients enrolled in the study had hyperuricemia with mean baseline values in the these groups between 9 and 9.2 mgs per deciliter, a diagnosis of gout, and the patients had experienced at least three or more flares in the previous year.

The primary endpoint of the trial was the flare rate, defined as the average number of flares per patient normalized for time on treatment, comparing the arhalofenate 800 milligram group to the allopurinol 300 milligram group in a modified intend to treat population. Patients in the allopurinol 300 mg group experienced an average of 1.24 flares over the 12 week treatment period. The flare rate for the arhalofenate 800 milligram group was 46 percent lower with a p-value of 0.0056. The flare rate for the allopurinol plus colchicine group, the positive control, was reduced by 68 percent relative to allopurinol alone with a p-value less than 0.0001.

A planned secondary analysis compared the flare rate of the arhalofenate groups to placebo. Arhalofenate 800 milligrams also showed a 41 percent lower flare rate than the placebo group with a p-value of 0.049. This improvement compared to placebo is a clear demonstration that arhalofenate lowers the flare rate while lowering serum uric acid at the same time. This study also confirms that arhalofenate produces reductions in flares even when colchicine is not present.

A secondary endpoint was the percent reduction in serum uric acid from baseline. As expected, the serum uric acid levels in the placebo group did not change appreciably, and the allopurinol groups showed the expected 25 to 30 percent decrease observed in prior clinical studies with this agent.

For arhalofenate 800 milligrams, we observed decreases of 20 and 16 percent at weeks eight and 12. While for arhalofenate 600 mgs, we saw 14 and 12 percent respectively. These reductions in serum uric acid for arhalofenate 600 and 800 mgs at 12 weeks versus placebo with statistically significant p-values of 0.0021 and .0059, but did not result in a statistically significant number of patients reaching the goal of less than six.

Now, I'd like to go back and address the issue of the relationship between the flare rate and serum uric acid lowering. A question that we asked ourselves is, "What is the flare reduction observed for the arhalofenate 800 mg group compared with the allopurinol 300 mg group," is effected by the fact there was less serum uric acid lowering. One very interesting finding in this study is that the flare rates for patients on placebo and allopurinol 300 milligrams were almost the same, 1.13 versus 1.24, in spite of the fact that the placebo group had no change in serum uric acid, while the allopurinol 300 mg group had the highest change.

This result is actually consistent with what was reported in Takeda's Apex study, namely that the flare rate only increased at very high levels of serum uric acid reduction above those observed in this study. This suggests that, as in the Apex study, patients were flaring for reasons largely independent of any change in their serum uric acid values. This argues that the 46 percent reduction of flare rate observed for arhalofenate is not strongly dependent on the differences in serum uric acid lowering between groups.

To investigate this further, we performed some post-hoc exploratory analysis. The most straightforward one entails a frequency matching approach that compared the flare rates from all patients in the 800 mg group with the subset of patients in the allopurinol group who had percent changes in serum uric acid in the same range. This approach resulted in matched distributions of mean serum uric acid decreases and distributions with a sample size of 51 in the arhalofenate group and 33 in the allopurinol group.

In this analysis the arhalofenate 800 mg group had a 39 percent lower flare rate than the matched subset. Another analysis of this kind support a similar conclusion. Interestingly, this figure is close to the 41 percent reduction compared to placebo. Thus, we believe that all of this data taken together are consistent with a flare rate reduction for arhalofenate in the 40 to 50 percent range.

The safety and tolerability of arhalofenate continue to be favorable. Arhalofenate was well tolerated and appeared safe. There were no serious adverse events deemed related to arhalofenate. There was one assay documented kidney stone in a patient on allopurinol 300 milligrams.

Eleven patients discontinued their study drug for an AE or laboratory abnormality. Of these, 8 were on allopurinol, one on arhalofenate 600, one on arhalofenate 800, and one on placebo. There were no meaningful differences in a number of patients reporting treatment emergent adverse events and no meaningful TEAE differences between groups. The most frequently reported TEAE during the study were creatine phosphokinase increases of 4.6 percent, upper respiratory tract infections, 3.8 percent, hypertension and headache both 3.3 percent. There were no subjects on arhalofenate 600 or 800 milligrams who developed an abnormal serum creatine value that was more than 1.5 times pretreatment values.

In terms of next steps, we plan to hold an end of Phase 2 meeting with the FDA in the third quarter of 2015 with the goal of starting Phase 3 in early 2016.

We'd now like to open the call up for your questions.

Mr. Brian Klein: Hi. Congrats on completing the study and thanks for taking my questions.

First I wanted to ask about how the drug didn't quite achieve the goal of less than six milligrams per deciliter in either arm. Can you comment as to why you think that happened and if you think potentially longer duration of therapy would achieve that goal?

Mr. Harold Van Wart: Yeah, it's a very good question. If there was anything about this study that was a little disappointing is that we had expected to get uric acid lowering more in the 25 to maybe 30 percent range rather than the close to 20 percent range. And at this time, I can only speculate that that could have been due to some non-compliance.

There was a heavy pill burden in this study. Patients had to take six different pills, one of which was an over capsulated form of colchicine. And one possibility is that there was some non-compliance. We won't know that for sure until, we have a chance to analyze the plasma samples to see how many patients were not compliant. We don't know whether or not the non-compliance might have been distributed differently across the different groups.

What I will say is that I think we hit the hard endpoint in this study, the difficult one, which was the flare rate difference. What we know because we have already read out our study in combination with febuxostat last month is that we would have gotten all of these patients to the serum uric acid goal of less than six if we had done a study in combination with 40 milligrams of febuxostat.

Mr. Brian Klein: Great. Thanks.

Next question is in regards to the 600 versus 800 milligrams. Can you comment on why you think the drop in flare rate was so dramatic when you added the additional 200 milligrams?

Mr. Harold Van Wart: Well, first of all we were very pleased to see that there was dose response because that makes it very believable. I don't have a good answer for why the drop was so different. Right now the data are what they are. It is a relatively bigger factor at 800, and we're pleased with that.

Mr. Brian Klein: Great. Thanks.

Just one final question, in terms of standard of care right now in the community what percentage of patients would you assume are receiving both allopurinol and colchicine simultaneously as opposed to just allopurinol monotherapy?

Unidentified Man: Well, in terms of the recommendations—the treatment guideline recommendations are that all patients should be on colchicine when initiating URAT lowering therapy.

From what we've seen around script data and some reports you know, surveys, it looks to be about half of patients are currently appropriately prophylaxed.

Mr. Brian Klein: Great. Thank you for taking my questions.

Mr. Harold Van Wart: You're welcome, Brian.

Mr. Phil Nadeau: Good morning. Thanks for taking my questions and congratulations on the data also.

Hal, one on the dosing, it does seem like you're not at the flat part of the dose response curve went you went from 600 milligrams to 800 milligrams.

So, is there any thought of perhaps pushing the dose of arhalofenate even higher to get either better flare reductions or a better change in serum uric acid?

Mr. Harold Van Wart: Well, unfortunately the pharmacokinetics start to saturate as you go above 800, Phil. When you go to a thousand you don't get appreciably higher exposures. So, we think 800 milligrams is probably the top dose for this product.

Mr. Phil Nadeau: Okay. And then, second on the Phase 3 program, how do the data today inform the design of the Phase 3 program? And in particular I guess I'm thinking of the serum uric acid reductions. Will you still do a monotherapy study now or is it likely that you'll do mostly studies in combination with febuxostat?

Mr. Harold Van Wart: Yeah. Well, that's a great question, Phil.

So, you know, we've now conducted five Phase 2 studies in gout patients with this product with the goal of trying to understand the best way to position it for patients. So, we've studied it in monotherapy in combination with XO inhibitors, and we've studied it in combination with colchicine and without colchicine.

So, first of all, we believe that there is no really safe uricosuric drug available on the market right now. The only thing that patients have is probenecid, which is not widely used.

So, any kind of a uricosuric drug that gives any reasonable amount of uric acid lowering therapy would be an attractive option for patients who can't take OX inhibitors and there are hundreds of thousands of those according to our market research.

Having said that we know from the study that we read out last month in combination with febuxostat that the combination of arhalofenate 800 with 40 milligrams or 80 milligrams of febuxostat gets virtually everybody below the goals of six and many below the goals of five. So, that would argue that we may shift our focus more toward combination therapy in the future.

Not having matched the allopurinol uric acid lowering would tend to indicate that we would have a hard time competing directly with those patients that are satisfied with their uric acid lowering on allopurinol.

So, worst case scenario is that we shift this—the focus of this program more to combination therapy with febuxostat.

Mr. Phil Nadeau: Okay, that's very helpful.

And then, just one on safety you noted an increase in creatine phosphokinase as one of the more frequently reported treatment emergent adverse events. Could you put that in perspective?

I guess this is probably a naive question, but what's that a marker of and is that something that that you're going to look into further in future trials?

Mr. Harold Van Wart: Well, I'll make a few comments and then ask my CMO to add to them if he wishes.

So, CPK is sort of an indication of muscle, a muscle biomarker. And in this study there was a lot of background therapy on statins, and that was relatively well distributed across the groups. And we believe that those elevations may well be related to that.

I will note that they were asymptomatic. There were no myalgias associated with them, and it's certainly not something that we have any concern about.

Pol, would you like to add to that?

Mr. Pol Boudes: Yeah. Hi, Phil. It's Pol.

The first mention, I want to say that the drug was very well tolerated and appears very safe.

The question of CPK is also a question of preparation, because patients with gout have a background into the incidence of CPK elevation that is around 10 percent. So, the 3.8 percent that we are seeing—or 4.8 percent we are seeing is not that high, and it's not a real difference in incidence between the groups.

So, as Hal mentioned also, as you know, this preparation [unintelligible] around statins, which by themselves elevates CPK. So, we see that. We mention it, but we don't think that it's a real concern.

Mr. Phil Nadeau: Okay. One last question, just a follow-up to my Phase 3 question. What- I guess what will be the goal of the Phase 3s? Will you be looking at flare endpoint or uric acid lowering endpoint or colcrys in the comparator arm or will you be starting this on the background of colchis? Can you maybe give us some idea what your initial thoughts are of the—kind of the overall goal of the Phase 3s?

Mr. Harold Van Wart: Yeah. Well, thanks, Phil. Another good—great question.

So we're just continuing to digest all this data right now, but I think our view is that in our Phase 3s we want to get registration for both serum uric acid lowering and flare as endpoints. And we certainly envision doing an arm of this study that's monotherapy and another arm that would be a combination with febuxostat.

And you know, we may elect to do some studies on a colchicine background and some not on a colchicine background to be able to see whether or not there are advantages to the patient in taking both. Now, that colchicine has gone generic it's not a big price barrier for patients to add that to the regimen.

Mr. Phil Nadeau: Okay, great. Thanks for taking my questions. That's very helpful.

Mr. Ed Arce: Hi, guys. Let me add my congratulations on the data as well.

Most of my questions have been asked and answered. But, I was just curious on the change in serum uric acid lowering from week eight to week 12 it seemed like it was going the other way over the last four weeks. I'm just wondering if you have any insight on why that is or you've seen that in previous trials.

Mr. Harold Van Wart: Yeah. No, we actually did not see that in our one month trial. However, since this was a three month trial one possibility is that we had an increase in non-compliance as the trial went on. And, interestingly we saw the same effect for the allopurinol arms. Between weeks four and 12 we also saw a falloff in the reduction in serum uric acid. And we can only speculate this could have been associated with the pill burden in this study, which, of course, would not be there if this were a product and it was being taken as a single pill.

Because of the fact this was a complicated five arm study patients had to take four different pills containing arhalofenate or arhalofenate placebo. They had to take allopurinol or allopurinol placebo, and they had to take a colchis or colchis placebo.

So, it was a bit of a pill burden problem, and it may well—the falloff may well have been related to that. It's only speculation at this time, but we will be able to figure that out as soon as we analyze the plasma samples.

Mr. Ed Arce: Okay. The other question I had you know relative to the flare rate lowering, obviously statistically significant versus allopurinol but I'm wondering your thoughts on the combination of allopurinol with colchicine you know, given that that's at least half of the use in the marketplace today. I know you said colchicine could be added easily now that it's generic, but the flare rate there is still significantly lower than arhalofenate 800.

Just wanted to get your thoughts on that. Thanks.

Mr. Harold Van Wart: Yeah. So we've always had the view that colchicine is a time honored way of treating and prophylaxing for flares. However, colchicine is not without some tolerability issues, and many patients don't take it or don't respond well to it. It has a lot of GI side effects. And what our data show is that we are an alternative to colchicine for flare reduction. We don't have to take an extra pill. We may even be somewhat additive to it.

So, we don't view that our product will necessarily compete with colchicine. We think it can be used as alternative, as stand alone or in combination with colchicine.

Mr. Ed Arce: Okay, great. Thank you.

Mr. Charles McWherter: This is Chuck. I would just also mention, you know, we're excited when we're going to get the rest of the data to look at other parameters around flares; time to first flare, intensity of flare, duration of flare. There's also—it's a very rich dataset. It was captured with an electronic diary so patient reported outcome just a treasure trove really of information. It hasn't been available in other studies of this type.

So, I think there is a possibility for understanding or comparing and contrasting the anti-flare effects of colchicine versus arhalofenate. That remains to be established, but it's something that we're very eager to get our hands on and dig into.

Mr. Harold Van Wart: I'd just like to conclude by saying that we're extremely excited by this. We really do believe that this is the first entry in a new class of drugs to address gout, which we're calling the ULAFT class. And, I think we- hit the hard endpoint here, which was the flares. We know we can make up for any disappointment in uric acid lowering through a combination with febuxostat, and we're looking forward going to speak to the FDA about this program and getting it into Phase 3 early next year.

So, thank you, everybody, for your attention.

Exhibit 99.2

February 24, 2015

CYMABAY THERAPEUTICS ANNOUNCES POSITIVE RESULTS FROM ITS PHASE 2b CLINICAL STUDY DEMONSTRATING THAT ARHALOFENATE MET THE PRIMARY ENDPOINT OF REDUCTION IN GOUT FLARES

Once daily oral dosing of arhalofenate both reduced gout flares and lowered serum uric acid while demonstrating good safety and tolerability

Newark, CA (February 24, 2015): CymaBay Therapeutics, Inc. (NASDAQ: CBAY) today announced positive preliminary topline results from its Phase 2b clinical study of its lead product candidate, arhalofenate, for the treatment of gout. The study met its primary endpoint of demonstrating a reduction in gout flare rate ($p = .0056$). This is the first study to show that arhalofenate produces reductions in flares without concomitant dosing of colchicine. Arhalofenate was well tolerated and the overall safety profile was favorable and consistent with results of earlier studies.

"We are very excited about these data which demonstrate the dual action of arhalofenate which both lowers serum uric acid and reduces flares constituting what we believe is a new class of gout therapy referred to as Urate Lowering Anti-Flare Therapy (ULAFT)," said Harold Van Wart, Chief Executive Officer of CymaBay. "This combination of activities in a single agent offers a potential new approach for treating gout patients. With five completed studies of arhalofenate in gout, our next step is to hold an end-of-phase 2 meeting with the FDA with the goal of starting Phase 3 in early 2016."

Study details

This randomized, double-blind, placebo- and active-controlled, 12 week study consisted of five arms. All patients had gout with hyperuricemia and experienced three or more flares in the previous twelve months. The flare rates and serum uric acid (sUA) changes for the five groups are shown in the following table along with key safety parameters demonstrating that arhalofenate was well tolerated. The study met its primary endpoint with a reduction of 46% in the flare rate for the arhalofenate 800 mg group compared to the allopurinol 300 mg group ($p = .0056$). In a secondary analysis, arhalofenate 800 mg showed a 41% lower flare rate than placebo ($p = .049$). The reductions in sUA for arhalofenate 600 and 800 mg at 12 weeks vs. placebo were statistically significant ($p = .021$ and ..0059, respectively), but did not result in a statistically significant number of patients reaching the goal of < 6 mg/dL.

		Placebo	Arhalofenate 600 mg	Arhalofenate 800 mg	Allopurinol 300 mg	Allopurinol 300mg + 0.6 mg COL
	N	28	53	51	54	53
Flare rate		**1.13**	**1.04**	**0.66[a]**	**1.24**	**0.40**
Mean % change in sUA from baseline to	**Week 8**	**+1**	**-14**	**-20**	**-30**	**-24**
	Week 12	**-1**	**-12[b]**	**-16[c]**	**-29**	**-25**
Patients discontinued for safety		**1**	**1**	**1**	**3**	**5**

[a] 46% reduction vs. allopurinol 300 mg with p = .0056 and 41% reduction vs. placebo with p= .049
[b] p = .0021 vs.placebo
[c] p = .0059 vs. placebo

The safety and tolerability of arhalofenate continue to appear favorable. Arhalofenate was well tolerated and appeared safe. There were no serious adverse events (SAEs) deemed related to arhalofenate. There was one SAE of a documented kidney stone that occurred in a patient on allopurinol 300 mg.

There were no meaningful differences in the number of patients reporting treatment emergent adverse events (TEAEs) and no relevant TEAE differences between groups. The most frequently reported TEAEs during the study were increases in creatinine phosphokinase (4.6%), upper respiratory tract infections (3.8%), hypertension and headache (both 3.3%). There were no subjects on arhalofenate who developed an abnormal serum creatinine value that was more than 1.5 times above pre-treatment values.

“Results from our clinical program to date suggest that arhalofenate may represent a new paradigm for the treatment of gout,” said Pol Boudes, Chief Medical Officer at CymaBay. “These data highlight the potential for arhalofenate’s use in multiple treatment settings. A more detailed analysis of these new data should be completed in the near future and we expect to present the results at a major scientific meeting.”

CymaBay has now completed five Phase 2 studies in patients with gout. Data from these studies suggest arhalofenate to be a safe and effective uricosuric drug with anti-flare activity. Based on our recently completed combination study of arhalofenate with febuxostat, we expect that the majority of patients taking the combination will achieve the sUA goal of below 6 mg/dL, with many patients achieving below 5 mg/dL. As a monotherapy, arhalofenate may address a need for patients who are intolerant to xanthine oxidase inhibitors or who are moderately hyperuricemic.

Conference Call

CymaBay will host a conference call today, February 24, 2015, at 8:30 a.m. ET / 5:30 a .m. PT to discuss the results of this Phase 2b trial in gout patients. The call can be accessed by dialing 877-407-8913 (domestic) and 201-689-8201 (international) five minutes prior to the start of the call. A live audio webcast of the call can be accessed under the Investors section of CymaBay’s website at http://ir.cymabay.com/events and will be available for 14 days following the call.

About Arhalofenate

Arhalofenate is a potential novel treatment for gout that has a dual mechanism of action. In clinical studies completed to date, arhalofenate has consistently demonstrated the ability to both reduce serum uric acid and reduce gout flares. Arhalofenate lowers serum uric acid by blocking the reabsorption of uric acid in the proximal tubules of the kidney by inhibiting a renal uric acid transporter called URAT1. This leads to the excretion of uric acid into the urine (a uricosuric effect). In addition, arhalofenate has an anti-inflammatory activity that is well suited to treating gout. Data from preclinical models show that it blocks the urate crystal-induced production of IL-1β, explaining its ability to reduce gout flares. This dual mechanism of action differentiates arhalofenate from all currently available treatments for gout. Arhalofenate has established a favorable safety profile in clinical trials involving more than 1,000 patients exposed to date.

About Hyperuricemia and Gout

Gout is a chronic, progressive rheumatic disease, caused by an inflammatory response to urate crystals deposited in joints and soft tissues as a result of excess uric acid in the blood (hyperuricemia). Chronic recurrence of gout flares in joints leads to tissue destruction with loss of function and debilitation. According to the NHANES (2007-2008) study, over 45 million Americans have hyperuricemia and over 8 million have progressed to a diagnosis of gout.

About CymaBay

CymaBay Therapeutics, Inc. (NASDAQ: CBAY) is a clinical-stage biopharmaceutical company developing therapies to treat metabolic diseases with high unmet medical need, including serious rare and orphan disorders. Arhalofenate, the company's lead product candidate, has shown two therapeutic actions in a single drug in multiple Phase 2 gout studies. In gout patients, arhalofenate is intended to prevent painful flares in joints while at the same time promoting excretion of uric acid by the kidney, thereby addressing both the signs and symptoms of gout and the hyperuricemia that is the root cause of the disease. CymaBay's second product candidate, MBX-8025 is a potent, selective, orally active PPAR-δ agonist. A Phase 2 study of MBX-8025 in patients with mixed dyslipidemia established that it has an anti-atherogenic lipid profile. CymaBay is in the process of initiating a pilot study of MBX-8025 in patients with homozygous familial hypercholesterolemia.

Cautionary Statements

The statements in this press release, including those statements regarding the potential of arhalofenate to treat gout, the therapeutic and commercial potential of arhalofenate and the anticipated timing and therapeutic and commercial potential of other product candidates of CymaBay Therapeutics, Inc. are forward looking statements that are subject to risks and uncertainties. Actual results and the timing of events regarding the further development of arhalofenate and other product candidates of CymaBay could differ materially from those

anticipated in such forward-looking statements as a result of risks and uncertainties, which include, without limitation, risks related to: the possibility that subsequent analyses of the data disclosed above may lead to different (including less favorable) interpretations of the results than the analyses conducted to date or may identify important implications of the Phase 2b study that are not reflected in these statements, or be subject to differing interpretations by any regulatory agency; the success, cost and timing of any of CymaBay's product development activities; any delays or inability to obtain or maintain regulatory approval of CymaBay's product candidates in the United States or worldwide; the ability of CymaBay to attract funding partners or collaborators with development, regulatory and commercialization expertise; the ability of CymaBay to obtain sufficient financing to complete development, regulatory approval and commercialization of its product candidates in the United States and worldwide; and the market potential for CymaBay's product candidates. Additional risks relating to CymaBay are contained in CymaBay's Quarterly Report on Form 10-Q, filed with the Securities and Exchange Commission on November 14, 2014. CymaBay disclaims any obligation to update these forward-looking statements except as required by law.

For additional information about CymaBay visit www.cymabay.com.

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