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): January 12, 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 7.01 Regulation FD Disclosure.

Attached as Exhibit 99.1 to this report, and incorporated herein by reference, is a slide presentation (the "Corporate Presentation"), which will be presented by CymaBay Therapeutics, Inc. at management presentations beginning Monday, January 12, 2015, to be held in San Francisco, California.

In accordance with General Instruction B.2. of Form 8-K, the information contained above in this Item 7.01 and the Corporate Presentation shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall the information or Corporate Presentation be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, except as shall be expressly set forth by specific reference in such a filing. This Item 7.01 will not be deemed a determination or an admission as to the materiality of any information in the Corporate Presentation that is required to be disclosed by Regulation FD.

Item 8.01 Other Events.

On January 12, 2015, the Company issued a press release announcing positive preliminary results from its Phase 2 clinical study of arhalofenate in combination with febuxostat. The full text of the press release issued in connection with the announcement is attached as Exhibit 99.2 to this report, which is incorporated by reference.

Item 9.01 Financial Statements and Exhibits.

Exhibit	Description
99.1	Arhalofenate Febuxostat Phase 2 PK/PD Study Presentation
99.2	Press Release issued on January 12, 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: January 12, 2015

EXHIBIT INDEX

Exhibit	Description
99.1	Arhalofenate Febuxostat Phase 2 PK/PD Study Presentation
99.2	Press Release issued on January 12, 2015

Exhibit 99.1

CymaBay Therapeutics Arhalofenate Febuxostat Phase 2 PK/PD Study Preliminary Top Line Data

Safe Harbor Statement

This presentation contains "forward-looking" statements that involve risks, uncertainties and assumptions, and actual results may differ substantially from those projected or expected in the forward-looking statements. Forward-looking statements include, but are not limited to: any projections of financial information; any statements about future development, clinical or regulatory events; any statements concerning CymaBay's plans, strategies or objectives; and any other statements of expectation or belief regarding future events. These statements are based on estimates and information available to CymaBay at the time of this presentation and are not guarantees of future performance. Actual results could differ materially from CymaBay's current expectations as a result of many factors including, but not limited to: CymaBay's ability to obtain additional financing to fund its operations; unexpected delays or results in clinical trials; uncertainties regarding obtaining regulatory approvals; uncertainties regarding the ability to protect CymaBay's intellectual property; uncertainties regarding market acceptance of any products for which CymaBay is able to obtain regulatory approval; the effects of competition; and other market and general economic conditions. You should read CymaBay's Quarterly Report on Form 10-Q filed with the SEC on November 14, 2014, especially under the caption "Risk Factors," which is available on the SEC web site at http://www.sec.gov, for a fuller discussion of these and other risks relating to an investment in CymaBay's common stock. CymaBay assumes no obligation for and does not intend to update these forward-looking statements, except as required by law.

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Arhalofenate Febuxostat Phase 2 PK/PD Study

- **Objectives**
  - Assess sUA reductions of different dose combinations
  - Measure the inter- and intraday fractional excretion of UA (FEUA)
  - Assess if there is a drug-drug interaction
  - Additional safety data for arhalofenate/febuxostat combination

	Weeks 1-2	Week 3	Week 4	Weeks 5-6
Cohort 1	Arhalofenate 600	Febuxostat 80 + Arhalofenate 600	Febuxostat 40 + Arhalofenate 600	Febuxostat 40
Cohort 2	Arhalofenate 800	Febuxostat 40 + Arhalofenate 800	Febuxostat 80 + Arhalofenate 800	Febuxostat 80

N = 16 per cohort ; PK from cohort 2 at Weeks 2, 4 and 6
All patients received colchicine for flare prophylaxis

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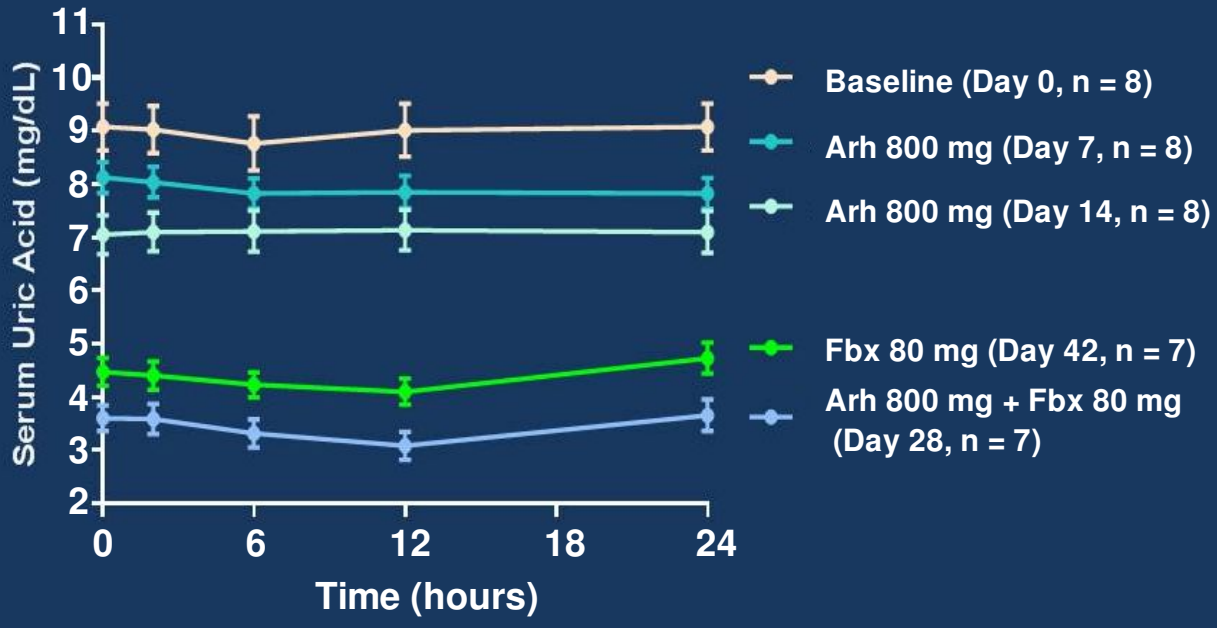
Arhalofenate Febuxostat Phase 2 PK/PD Study
Intraday variation of sUA for selected dose combinations



Mean ± SE

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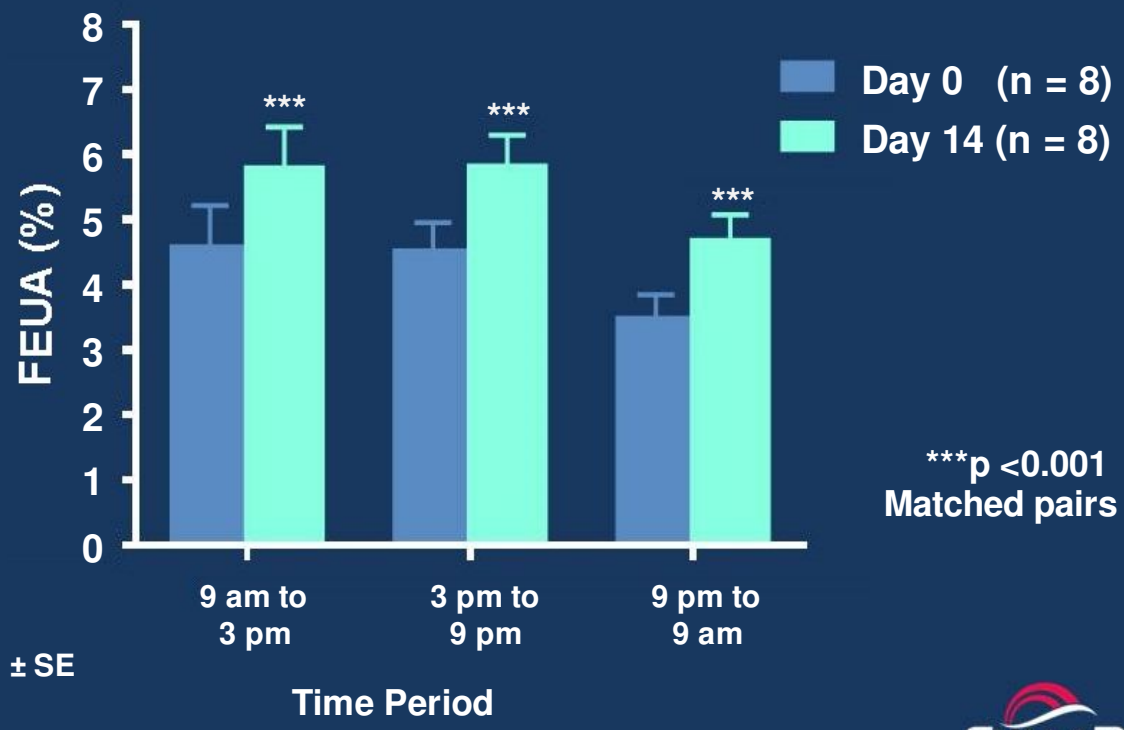
Arhalofenate Febuxostat Phase 2 PK/PD Study

Intra- and interday variation in FEUA for arhalofenate (800 mg



***p <0.001
Matched pairs t-test

Mean ± SE

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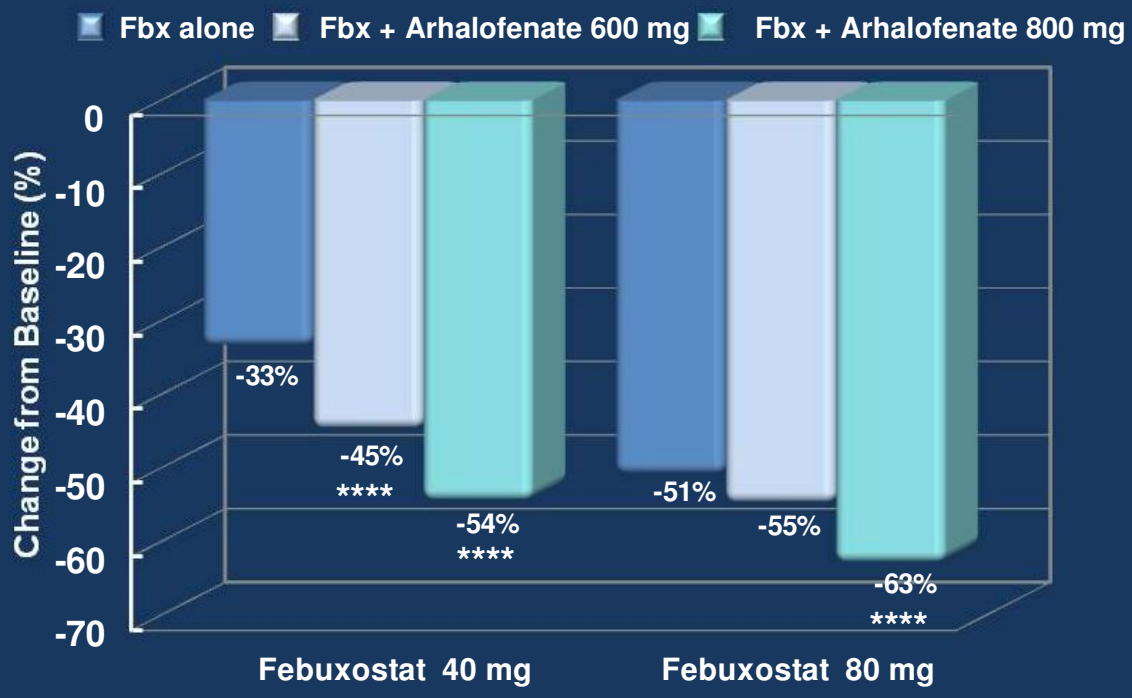
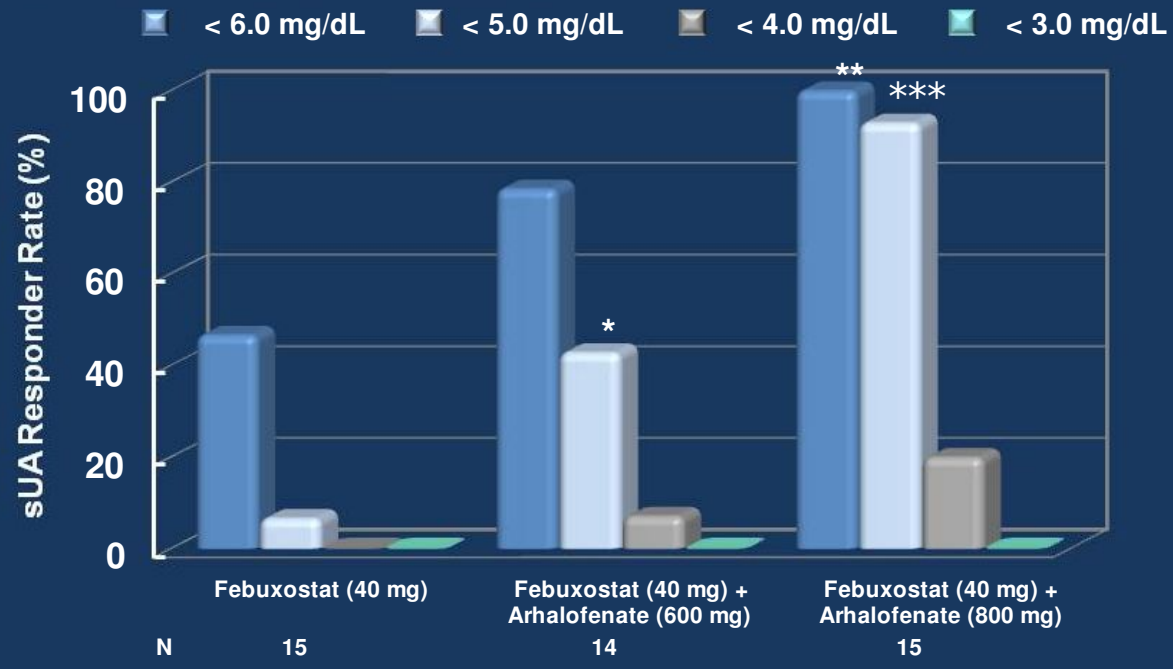
Arhalofenate Febuxostat Phase 2 PK/PD Study
Mean lowering of serum uric acid



**** p < .0001 vs. febuxostat alone groups

Arhalofenate Febuxostat Phase 2 PK/PD Study
sUA responder rate for febuxostat 40 mg treatments



p-values reflect comparisons vs. febuxostat 40 mg. McNemar's exact test and Fischer's exact test were used for comparisons within and between cohorts, respectively.
* p < .05 ** p < .01 *** p < .001

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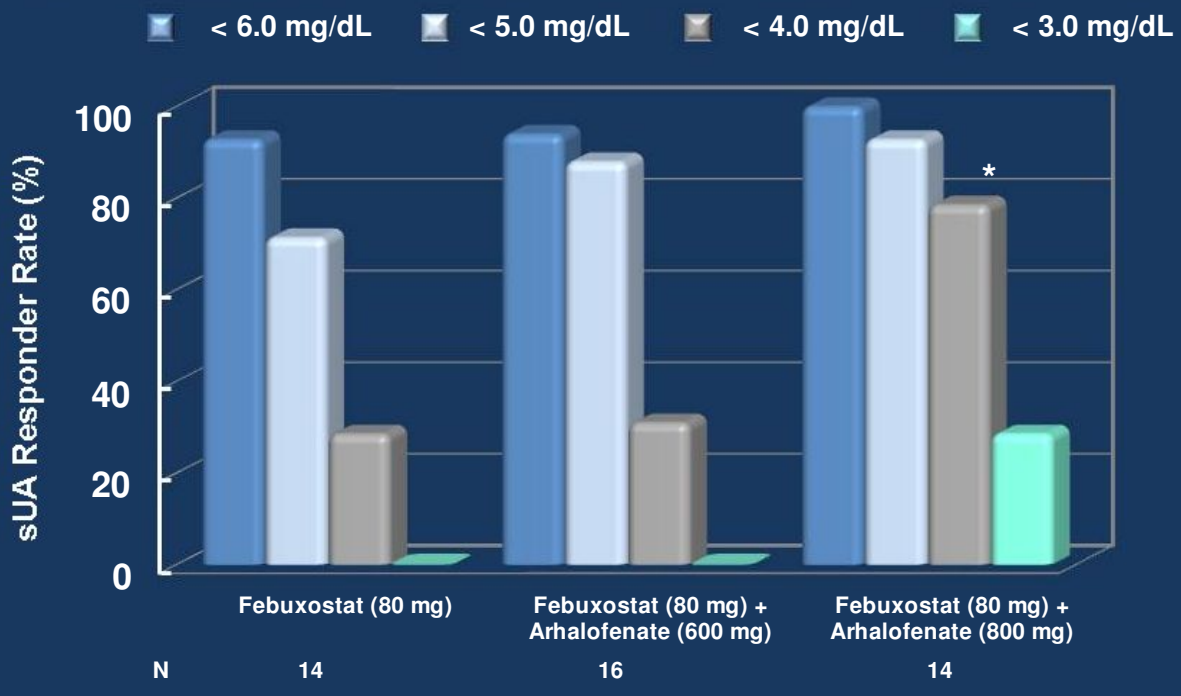
Arhalofenate Febuxostat Phase 2 PK/PD Study
sUA responder rate for febuxostat 80 mg treatments



p-values reflect comparisons vs. febuxostat 80 mg. McNemar's exact test and Fischer's exact test were used for comparisons within and between cohorts, respectively.
* p < .05

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Arhalofenate Febuxostat Phase 2 PK/PD Study
Safety overview

- **Noncompleters**
  - Uncontrolled hypertension deemed unrelated to study drugs by PI
  - GI symptoms, myalgia, headache attributed to colchicine by PI
  - One patient terminated due to protocol non-compliance

- **Adverse events**
  - No serious adverse events
  - 1 Severe AE (uncontrolled hypertension)
  - 1 flare during the run-in and 3 during the treatment phase

- **Laboratory findings**
  - One case of transaminase elevation that emerged after initiation of febuxostat
  - No patient had a creatinine increase of >1.5X or a creatinine value greater than the upper limit of normal

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Exhibit 99.2

January 12, 2015

CYMABAY THERAPEUTICS ANNOUNCES POSITIVE RESULTS FROM ITS PHASE 2 CLINICAL STUDY OF ARHALOFENATE IN COMBINATION WITH FEBUXOSTAT

Results indicate that arhalofenate increases the fractional excretion of uric acid with low intraday variations and increases the serum uric acid responder rate in combination with febuxostat

Newark, CA (January 12, 2015): CymaBay Therapeutics Inc. (NASDAQ: CBAY) today announced positive preliminary results from its clinical study of arhalofenate administered in combination with febuxostat (Uloric™, Takeda Pharmaceutical Company Limited). Arhalofenate is a once-daily, oral candidate for the treatment of gout with a unique dual mechanism of action which lowers serum uric acid (sUA) while also reducing the occurrence of gout flares.

Current treatment guidelines for gout recommend the use of urate lowering drugs to reverse hyperuricemia in order to remove deposits of proinflammatory urate crystals. The minimal goal of this treatment is to reduce sUA levels to below 6 mg/dL; reducing sUA values to below 5 or 4 mg/dL is particularly desirable for patients with advanced disease in order to dissolve urate deposits (known as tophi) within a practical timeframe. Many patients treated with currently marketed xanthine oxidase inhibitors (allopurinol or febuxostat) alone do not reach these goals.

Arhalofenate blocks 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) that could provide additional sUA lowering when used in combination with xanthine oxidase inhibitors. In an earlier study, CymaBay showed that the combination of arhalofenate (400 and 600 mg) and febuxostat (80 mg) markedly lowered sUA in gout patients.

In the present Phase 2 clinical study, the sUA lowering of additional combinations of arhalofenate (600 and 800 mg) and febuxostat (40 and 80 mg) were evaluated. In addition, data have been collected to understand the time course of the uricosuric effect. Arhalofenate has a long serum half-life (~50 hours) and serum levels reach steady state gradually. The time course of changes in sUA, urinary uric acid (uUA) and the fractional excretion of uric acid (FEUA) as monotherapy have been examined over the first 2 weeks. Analysis of arhalofenate and febuxostat drug levels to assess for a potential drug-drug interaction is underway and will be reported in a subsequent communication.

Clinical Study of Co-administration of Arhalofenate and Febuxostat

This study was an open label Phase 2 study carried out at a single center on two separate cohorts (n = 16 each) of gout patients with baseline sUA levels of 9.4 and 9.2 mg/dL, respectively (clinicaltrials.gov NCT02252835). The patients were either treatment naïve or willing to discontinue uric acid lowering therapy. All dosing was once daily oral and the patients received colchicine for flare prophylaxis. One cohort received arhalofenate 600 mg for 2 weeks followed by sequential one week periods of co-administration of

febuxostat 80 mg followed by 40 mg. During the final two weeks, febuxostat 40 mg was administered as monotherapy. The second cohort had a similar design in which patients received arhalofenate 800 mg for 2 weeks, followed sequentially by one week of co-administration of 40 mg followed by 80 mg of febuxostat. Dosing with febuxostat at 80 mg was then continued for two additional weeks. sUA was assessed at multiple time points including at the end of each treatment period for both cohorts. uUA was measured for 8 patients in the cohort receiving arhalofenate 800 mg monotherapy on selected days over three time intervals (9 am to 3 pm, 3 pm to 9 pm and 9 pm to 9 am), allowing FEUA values to be assessed.

The sUA levels for patients receiving arhalofenate 800 mg decreased gradually over 14 days with approximately half of the decrease by day 7. Intraday variations in mean sUA were small (<10%) on all days. In contrast, uUA increased over the 14 day treatment period. Correspondingly, FEUA values increased over baseline for each day assessed during the 14 day treatment period and did not vary appreciably during the day. FEUA values ranged from ~4.6 (morning) to ~3.5% (night) at baseline and from ~5.8% (morning) to ~4.7% (night) on day 14, respectively. The increases from baseline to day 14 were significant ($p < .001$). These data are consistent with the view that serum levels of arhalofenate slowly (>2 weeks) equilibrate to steady state producing gradual decreases in sUA, increases in uUA and increases in FEUA. The small intraday variations in arhalofenate levels likewise produce small intraday variation in sUA, uUA and FEUA. This slow, natural equilibration eliminates the need for dose titration of arhalofenate.

The responder rates (percentage of patients reaching goal) for the sUA targets of <6, <5 and <4 mg/dL for both doses of febuxostat and all four combinations of febuxostat and arhalofenate are shown in the table below. The addition of arhalofenate to either dose of febuxostat increases the responder rates for all sUA targets. The combination of febuxostat (80 mg) and arhalofenate (800 mg) is particularly effective with 79% of patients achieving the target of <4 mg/dL ($p < .05$).

Responder Rate (% patients with sUA < Target)

		Target (mg/dL)		
Febuxostat (mg)	Arhalofenate (mg)	<6	<5	<4
40	0	47	7	0
40[a]	600	79	43*	7
40[a]	800	100**	93***	20
80	0	93	71	29
80[b]	600	94	88	31
80[b]	800	100	93	79*

*p < .05 ** p <.01 *** p < .001
(a) Comparison vs. 40 mg and (b) 80 mg febuxostat monotherapy

The combination of arhalofenate and febuxostat was well tolerated. There were no serious adverse events and only one severe adverse event of uncontrolled hypertension not deemed to be related to the study drugs. There was one case of elevated liver transaminases that emerged after the initiation of febuxostat in the second cohort. No patient in the study experienced a >1.5X elevation in creatinine or had a value greater than the upper limit of normal.

“These results indicate that arhalofenate has very attractive characteristics for a uricosuric drug and that it has the potential to be used in combination with febuxostat to provide clinically meaningful lowering of serum uric acid for patients with gout” said Pol Boudes, MD, Chief Medical Officer of CymaBay. “This combination of oral agents has the potential to lower serum uric acid levels into the range needed to promote dissolution of debilitating uric acid crystals, thereby providing a potential treatment alternative for gout patients.”

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 inherent 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 nearly 1,000 patients exposed to date.

About Hyperuricemia and Gout

Gout is a chronic, progressive rheumatic disease, caused by an inflammatory response to uric acid 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, the incidence of hyperuricemia in the US is over 45 million 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 Phase 2a gout studies. In gout patients, arhalofenate is intended to prevent painful flares in joints while at the same time promoting excretion of serum uric acid (sUA) by the kidney, thereby addressing both the signs and symptoms of gout and the hyperuricemia that is the root cause of the disease. In addition to the study described above, CymaBay has a second ongoing 12-week Phase 2b clinical trial in patients with gout which is powered to detect statistically significant reductions in gout flares. 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. We are in the process of initiating a pilot study in patients with homozygous familial hypercholesterolemia.

Cautionary Statements

The statements in this press release regarding the potential of arhalofenate in combination with febuxostat to treat gout are forward looking statements that are subject to risks and uncertainties. Risks that could cause actual results to differ from these statements include: the study was a small study of two cohorts with only 16 patients in each cohort and therefore the statistical significance of positive results is not as great as would be obtained in a trial with significantly more patients, and therefore different results may be obtained in a larger clinical trial; CymaBay may experience a number of unforeseen events during further and larger clinical trials for arhalofenate that could delay or prevent the commencement and/or completion of clinical trials, which may prevent the commercialization of arhalofenate; arhalofenate has not been approved by the FDA, and obtaining regulatory approval is difficult and may never be obtained. 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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