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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 -- (Marketwired) -- 02/24/15 -- 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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