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): March 3, 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 March 3, 2015, CymaBay Therapeutics, Inc. (the "Company") issued the slide presentation (the "Corporate Presentation"), attached here as Exhibit 99.1 to this report, which will be presented by the Company at management presentations beginning on March 4, 2015, to be held in Boston, Massachusetts.

Item 9.01. Financial Statements and Exhibits.

Exhibit	Description
99.1.	Corporate Presentation

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 3, 2015

EXHIBIT INDEX

Exhibit	Description
99.1.	Corporate Presentation

Exhibit 99.1

Cowen Health Care Conference
March 4, 2015
Boston, MA

Harold Van Wart, Ph.D.
President and CEO

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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CymaBay Highlights

- **Arhalofenate is the first drug in the Urate Lowering Anti-Flare Therapy (ULAFT) class for the treatment of gout**
  - Reduces gout flares and lowers serum uric acid (sUA)
  - Refined product profile established in recently completed Phase 2 studies
  - Good overall and renal safety data in over 1,000 subjects

- **MBX-8025 is a potential novel treatment for rare or serious lipid and liver disorders, including homozygous familial hypercholesterolemia (HoFH)**
  - Positive effects on lipids and markers of liver health demonstrated in mixed dyslipidemia Phase 2 trial
  - Focusing further development in rare or orphan diseases

- **Near-term, value-driving catalysts**
  - Arhalofenate End-of-Phase 2 meeting with FDA in 3Q 2015
  - Phase 2 trial initiation for MBX-8025 in HoFH expected in 1H 2015

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Key Features of Gout
Hyperuricemia, urate crystal deposits and flares

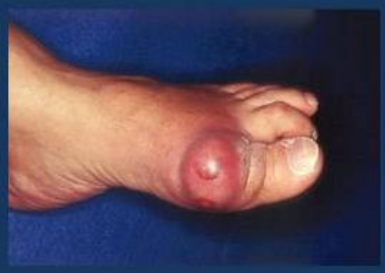
Mono Sodium Urate (MSU) crystal deposits

Inflammatory response
↑ IL-1β

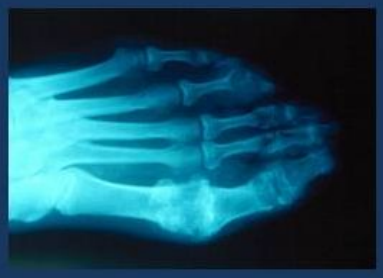
Therapeutic targets

Hyperuricemia
↑ Serum Uric Acid (sUA)

Painful flare



Joint erosion



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Current Treatment of Gout
Uric acid Lowering Therapies (ULTs) and anti-inflammatories

- **Treatment paradigm (ACR Guidelines)**
  - Anti-inflammatory to treat the flare
  - ULT is initiated to address the hyperuricemia with a goal of sUA < 6 mg/dL to debulk offending MSU burden
  - Initiation of ULT increases flare risk, requiring dose titration and colchicine prophylaxis

- **Anti-inflammatory drugs**
  - Colchicine (Colcrys)
  - NSAIDs, steroids
  - Ilaris (anti-IL-1β biologic) approved in EU

- **ULTs**
  - Xanthine oxidase (XO) inhibitors (allopurinol, febuxostat)
  - Uricosurics (probenecid, lesinurad in development)
  - Pegloticase (for severe treatment failure gout)

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Gout Patients Are Poorly Served by Available Drugs
Need for more sUA lowering and better control of flares

- **More sUA lowering**
  - ~2 million patients do not reach their sUA goal on their current ULT
    - <6 mg/dL for patients without tophi
    - <5 mg/dL for patients with tophi
  - ~300 thousand patients are allopurinol intolerant
  - Inadequate responders to ULT continue to increase their MSU burden and the disease continues to progress

- **Better flare control**
  - ~1 million patients experience≥ 3 flares/year
  - Patients care about the pain, suffering and medical costs of flares
  - Colchicine, NSAIDs and steroids are "strongly contraindicated" in >40% of gout patients due to their comorbidities
  - Colchicine non-compliance is estimated to be ~40%

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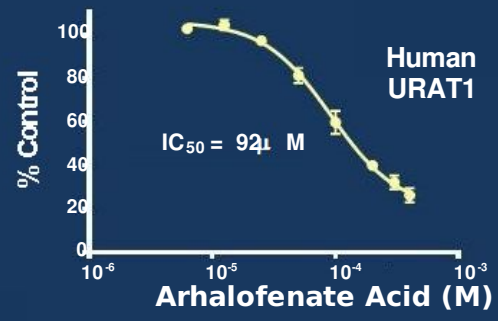
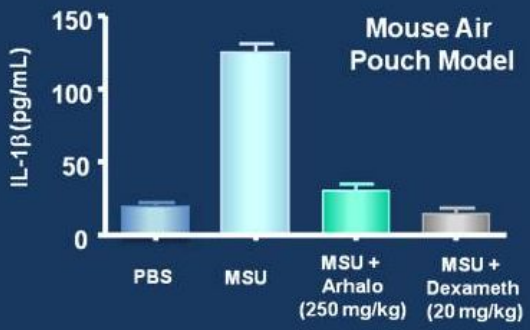
Arhalofenate
The First Urate Lowering Anti-Flare Therapy (ULAFT)

- **Lowers sUA by improving uric acid excretion in the kidney (uricosuric effect)**
  - Blocks urate reabsorption by URAT1
  - Same target as lesinurad
  - Very favorable PK for a uricosuric drug
    - 50 hour half-life produces gradual changes in serum and urinary UA with small intraday fluctuations
    - Avoids "hyperuricosuria"
- **Reduces flares through anti-inflammatory properties and long plasma half-life**
  - Suppresses MSU crystal-induced IL-1β in gouty joints
  - No systemic suppression of IL-1β and no infection risk





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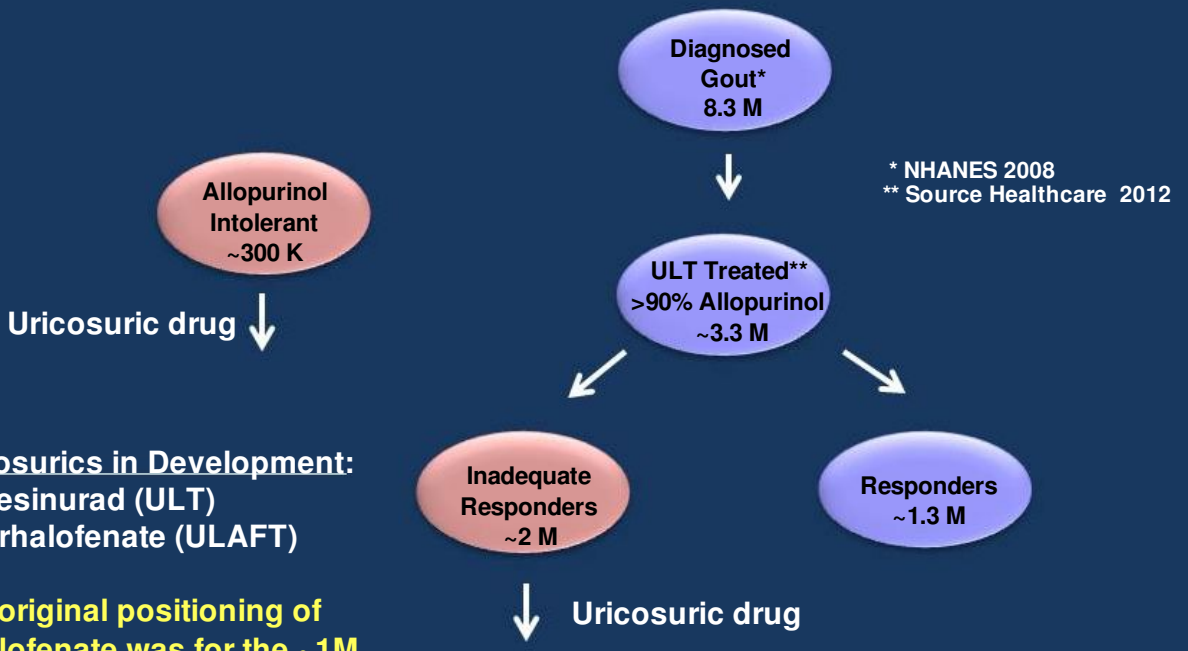
Gout Population Segregated by sUA Status
Treating inadequate responders with a uricosuric drug



Uricosurics in Development:
- Lesinurad (ULT)
- Arhalofenate (ULAFT)

The original positioning of arhalofenate was for the ~1M patients flaring >3 times/year

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Arhalofenate Phase 2 Clinical Program for Gout

- **Five Phase 2 studies completed**
  - Monotherapy with and without colchicine
  - Combination with febuxostat and allopurinol
  - Arhalofenate doses of 400, 600 and 800 mg
- **Summary of results**
  - Very effective sUA lowering in combination with febuxostat (40 or 80 mg)
  - Arhalofenate monotherapy is an alternative for the XOI intolerant patients
  - Arhalofenate has anti-flare activity
    - Provides the symptomatic relief that patients really want
    - Helps patients in which colchicine is contraindicated or not tolerated
  - Does not require dose titration
- **Refined product positioning**
  - Recent arhalofenate Phase 2 data and lesinurad news has shifted positioning to address the larger sUA inadequate responder segment

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Arhalofenate Febuxostat Phase 2 Study

- **Objectives**
  - Assess sUA reductions of different dose combinations
  - Measure the interday 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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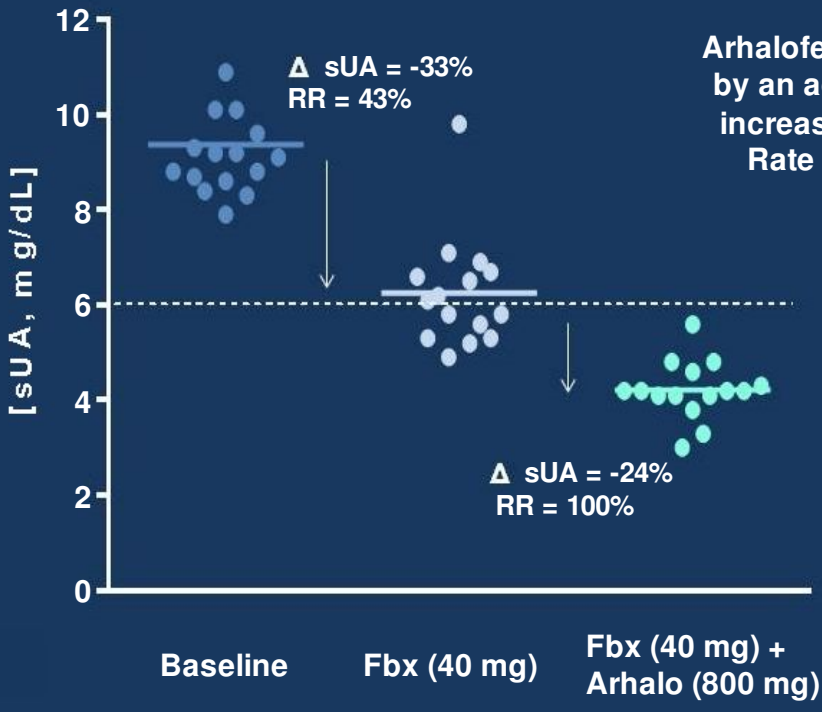
Arhalofenate Febuxostat Phase 2 Study
Changes in sUA for treatment phases



Arhalofenate decrease sUA by an additional 24% and increases the Responder Rate from 43 to 100%

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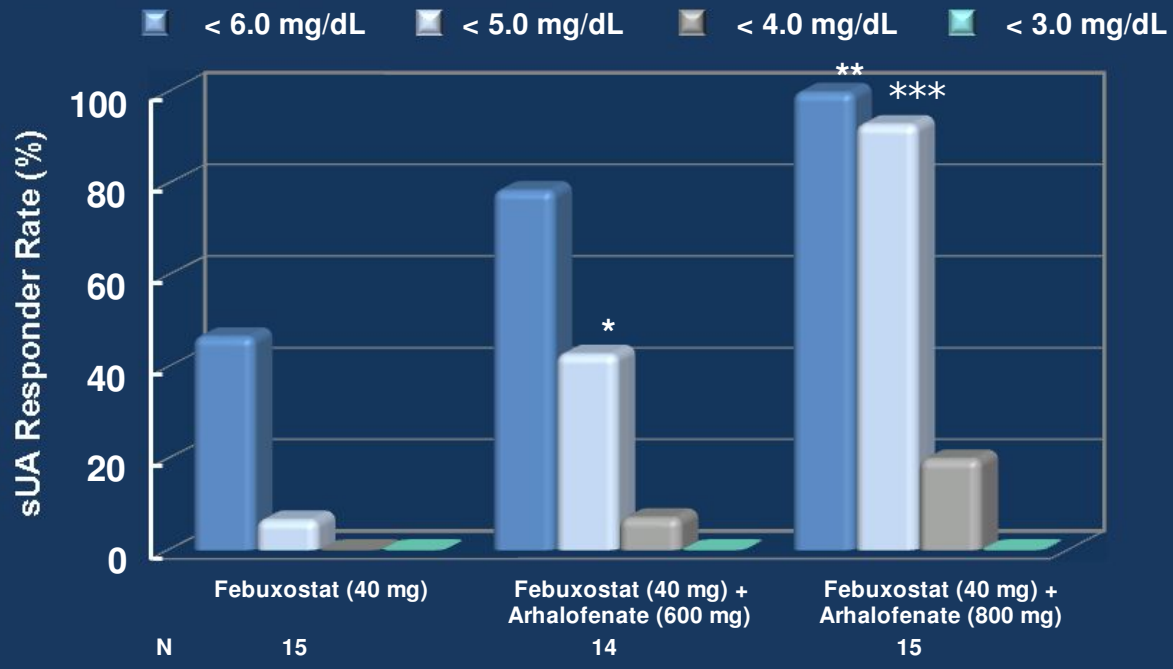
Arhalofenate Febuxostat Phase 2 PK/PD Study
sUA responder rate for febuxostat 40 mg treatments



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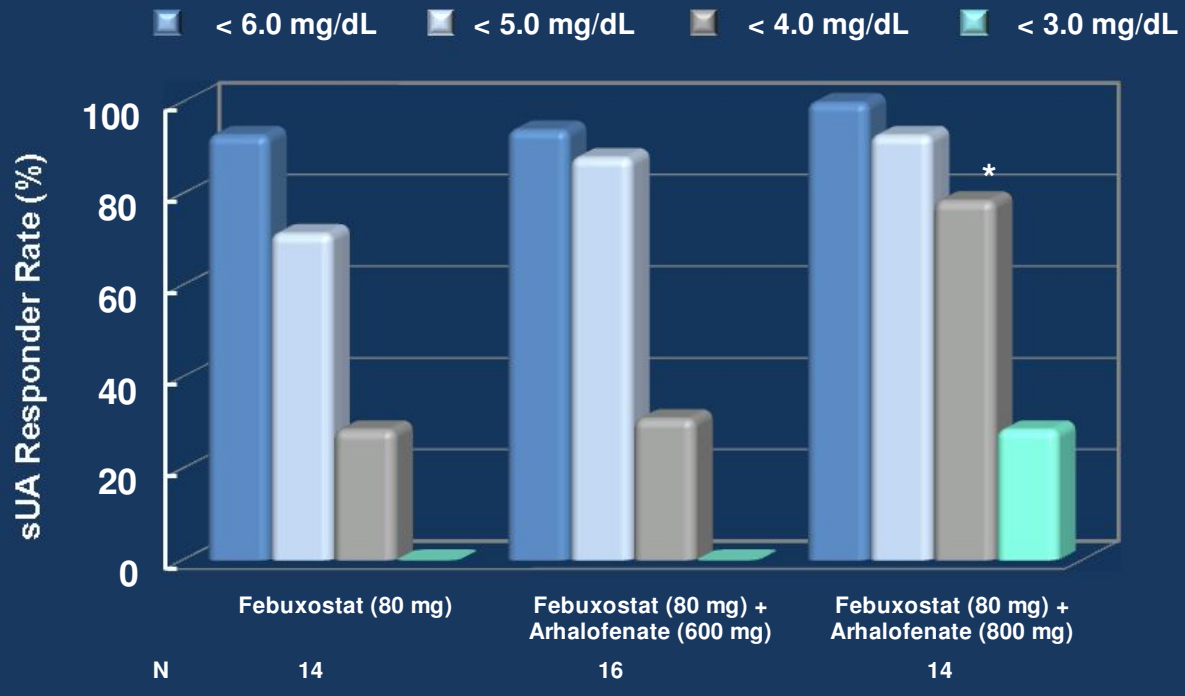
Arhalofenate Febuxostat Phase 2 PK/PD Study
sUA responder rate for febuxostat 80 mg treatments



* p < .05 for comparison vs. febuxostat 80 mg; McNemar's exact test

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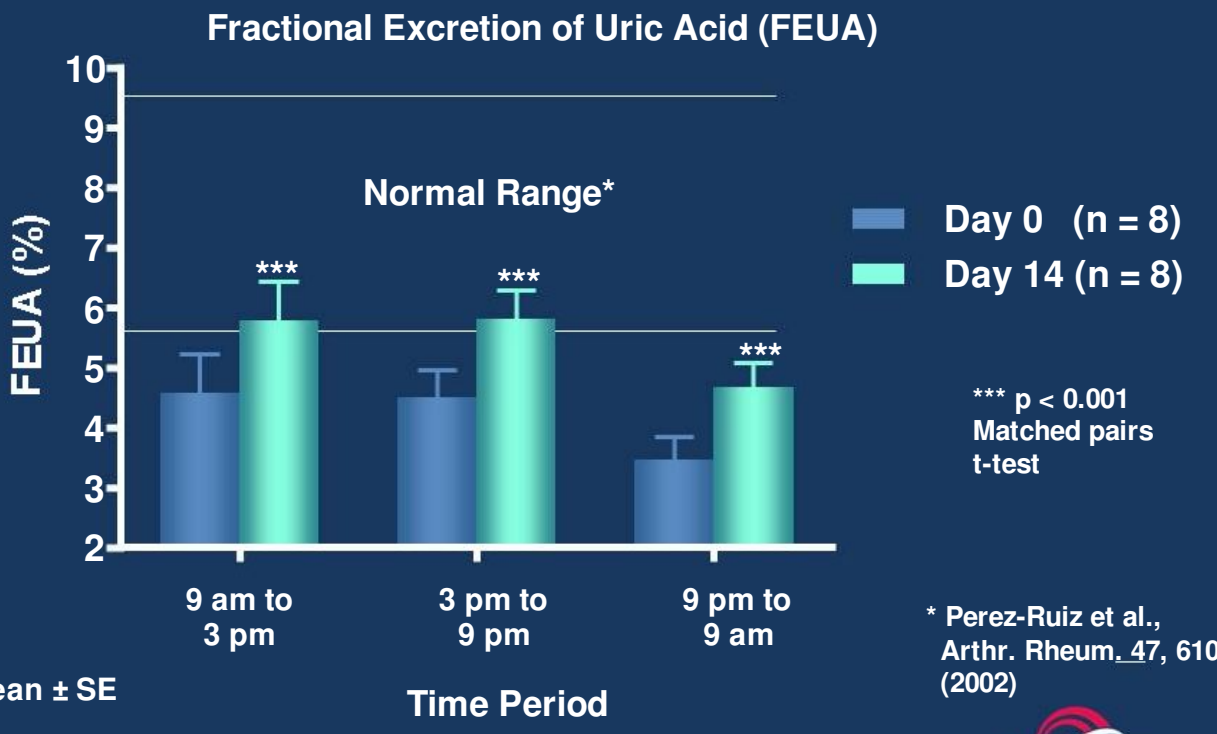
Arhalofenate Febuxostat Phase 2 PK/PD Study

Intraday and interday variation in FEUA for arhalofenate (800 mg)



*** p < 0.001 Matched pairs t-test

Mean ± SE

* Perez-Ruiz et al., Arthr. Rheum. 47, 610 (2002)

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Arhalofenate Phase 2b Flare Study
Arhalofenate flare study

- **Design**
  - Randomized double blind placebo- and active-controlled study
  - 12-week duration
  - 248 gout patients who had ≥3 flares in prior year
- **Goal for arhalofenate**
  - Establish statistically significant reduction in gout flares
  - Show anti-flare effect in the absence of colchicine
- **Primary end point**
  - Mean flares/patient
- **Secondary end point**
  - Reduction in sUA

Electronic diary for patient flare reporting

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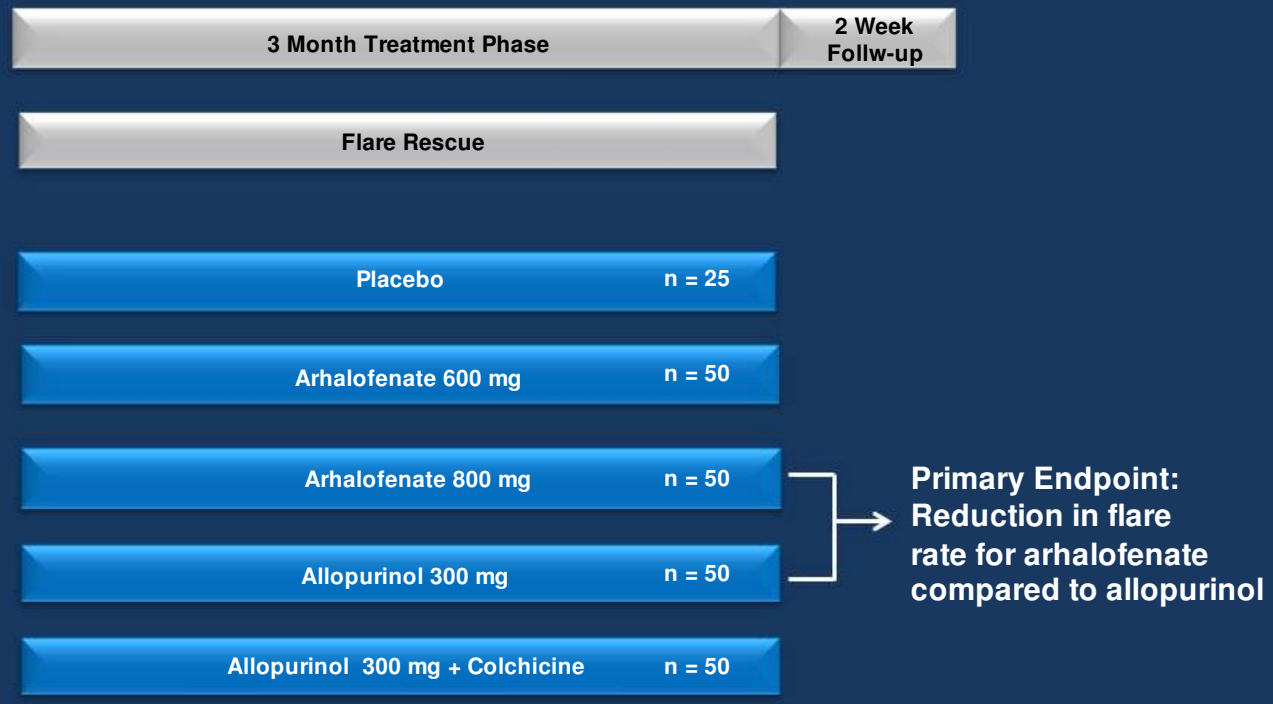
Arhalofenate Phase 2b Study Design



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Arhalofenate Phase 2b Flare Study
Most frequent adverse events and safety summary

	Placebo	Arhalofenate 600 mg	Arhalofenate 800 mg	Allopurinol 300 mg + COL	Allopurinol 300 mg
N	28	53	51	53	54
AEs	17 (60.7%)	24 (45.3%)	21 (41.2%)	24 (45.3%)	22 (40.7%)
SAEs	0	1	0	1	3
Discon due to AE or Lab	1	1	1	5	3
CK increased	0	3 (5.7%)	2 (3.9%)	3 (5.7%)	3 (5.6%)
URT Infection	2 (7.1%)	3 (5.7%)	2 (3.9%)	2 (3.8%)	0
Headache	1 (3.6%)	3 (5.7%)	2 (3.9%)	0	2 (3.7%)
Hypertension	2 (7.1%)	1 (1.9%)	2 (3.9%)	2 (3.8%)	1 (1.9%)
Creatinine >1.5X and >ULN	0	0	0	0	0

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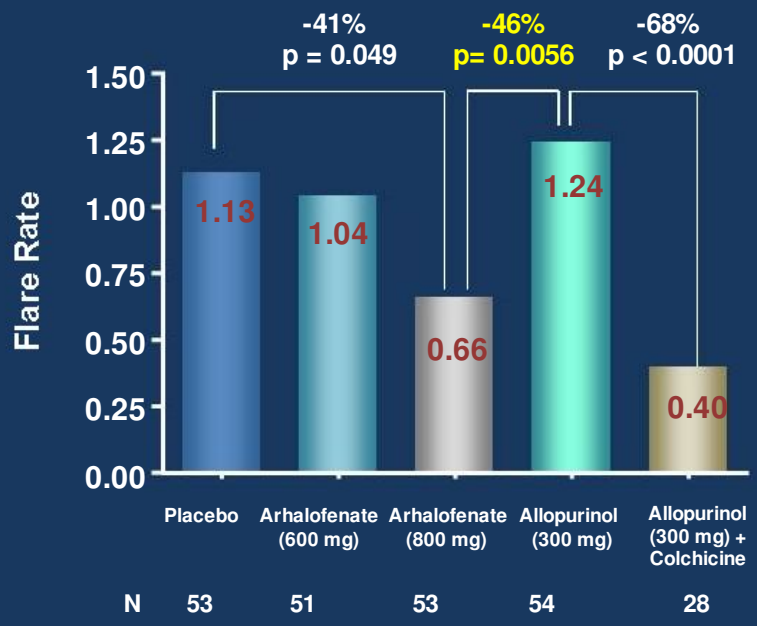
Arhalofenate Phase 2b Flare Study

Arhaolfenate 800 mg dose met the primary flare end point



Arhalofenate (800 mg) vs. allopurinol (300 mg) + colchicine were not statistically different (p = .091)

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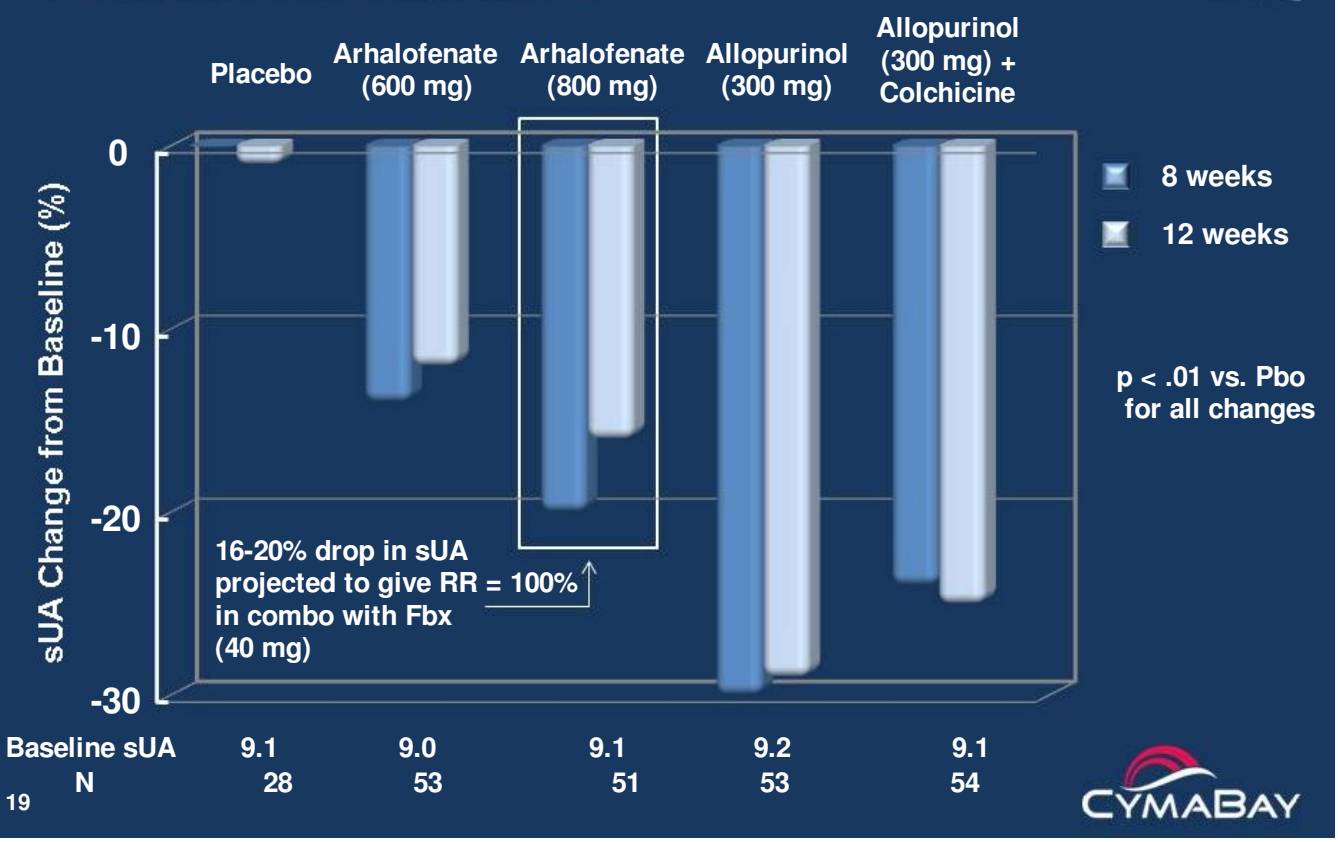
Arhalofenate Phase 2b Flare Study
Mean lowering of serum uric acid



Baseline sUA	9.1	9.0	9.1	9.2	9.1
N	28	53	51	53	54

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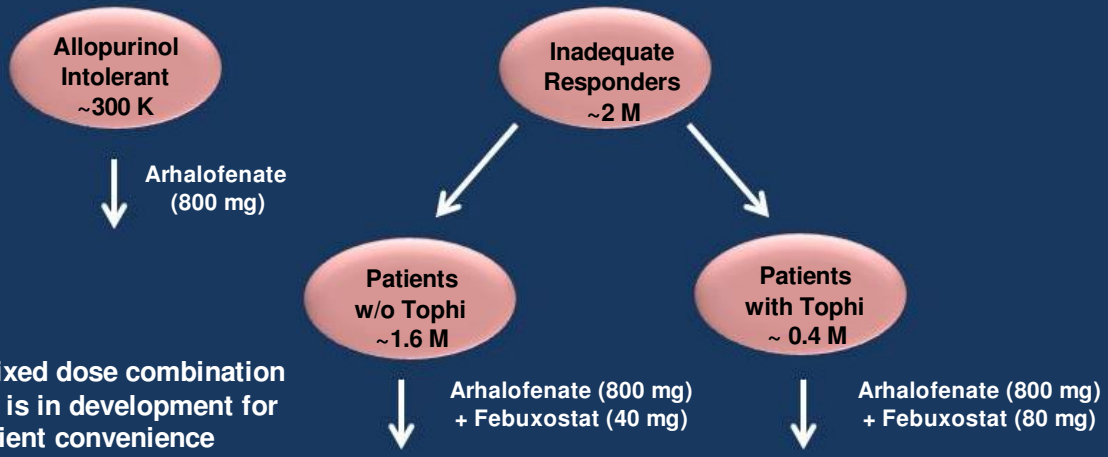
Arhalofenate Target Population
Major target is inadequate responders on current ULT



A fixed dose combination pill is in development for patient convenience

Benefits to Patient

Great majority reach their sUA goal
Experience fewer flares
Don't need colchicine prophylaxis
No need for dose titration

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Comparison of Arhalofenate with Lesinurad

	Arhalofenate (800 mg)	Lesinurad (200 mg)
sUA lowering	16-24% (Ph 2)	16% (Ph 2)*
Monotherapy for allopurinol intolerant	Yes	No**
Responder Rate (< 6 mg/dL) increase with XOI		
On top of Allo (300 mg)		38% (Ph 2)*; 25% (Ph 3)**
On top of Fbx (40 mg)	57% (Ph 2)	
Flare benefit	Yes	No**
Colchicine Prophylaxis needed	No	
Renal safety	No signal	

* Ardea presentation materials **ACR presentation, 2014

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Arhalofenate Clinical Studies
Safety summary

- **Completed 9 Phase 2 clinical studies (200-800 mg)**
  - More than 1,000 subjects exposed to arhalofenate for up to 6 months
- **General safety**
  - Adverse events similar to placebo and balanced across dose groups
  - Low incidence of asymptomatic liver transaminase elevations
  - No change in neutrophils or increase in infections
- **Renal safety**
  - No kidney stones or decrease in urine pH
  - No creatinine signal
- **No dose-limiting toxicity has been identified**

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Arhalofenate Nonclinical Studies
Non-clinical development status

- **Drug materials**
  - Economical, proprietary synthesis
  - Tablet formulations developed
  - Fixed dose formulation with febuxostat in development

- **Completed preclinical safety package**
  - Sub-chronic and chronic toxicology
  - Safety pharmacology and reproductive toxicology
  - Two-year carcinogenicity studies
  - Carcinogenicity and CV safety review by FDA completed

- **All results support further development**

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Phase 3 Program for Arhalofenate
Preliminary study design

- **Non-tophaceous gout (n = 1,000) in allopurinol inadequate responders**
  - Febuxostat (40 mg) + colchicine vs. febuxostat (40 mg) + arhalofenate (800 mg) for 6 months + 6 month safety extension
  - Primary endpoint: sUA responder rate (< 6 mg/dL) at 6 months
  - Secondary end point: average flares/patient at 6 months

- **Tophaceous gout (n = 300) in allopurinol inadequate responders**
  - Febuxostat (80 mg) + colchicine vs. febuxostat (80 mg) + arhalofenate (800 mg) for 6 months
  - Primary endpoint: sUA responder rate (< 5 mg/dL) at 6 months
  - Secondary endpoints: flare rate and tophi resolution at 12 months

- **Patients intolerant to allopurinol (n = 200)**
  - Placebo and arhalofenate (800 mg) for 6 months with 6-month safety extension
  - Primary endpoint: average flares per patient at 6 months
  - Secondary end point: sUA responder rate (< 6 mg/dL) at 6 months

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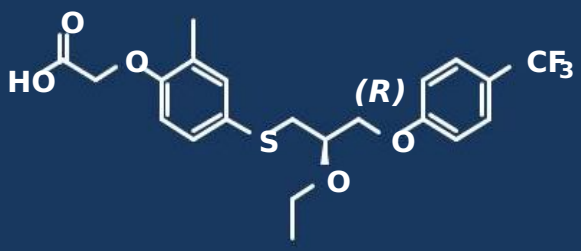
MBX-8025

- **MBX-8025**
  - Potent selective PPARδ agonist
  - Once daily orally administered



- **Status**
  - Originally in development for mixed dyslipidemia
  - Phase 2 study demonstrated favorable effects on lipids and liver biomarkers
  - FDA now requiring a preapproval CV outcome study for this indication

- **Redirecting program to higher unmet need indications**
  - Homozygous familial hypercholesterolemia (HoFH)
  - Primary biliary cirrhosis (PBC)
  - Severe refractory hypertriglyceridemia (SHTG)
  - Nonalcoholic steatohepatitis (NASH)

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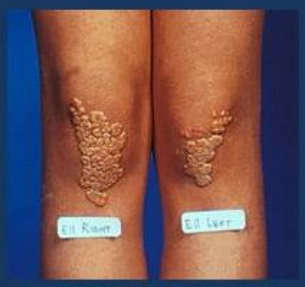
Homozygous Familial Hypercholesterolemia (HoFH)
Rare disease of impaired cholesterol metabolism

- **Orphan disease affecting 1 in 1 million**
  - Markedly elevated LDL-C (>500 mg/dL) caused by loss-of-function mutations in LDL receptor (LDL-R)
  - Cardiovascular disease (MI, stroke, CAD) before the age of 20
  - Mean age of death is in the 30s
  - Current therapy is focused on lowering LDL-C
- **Therapies that raise LDL-R activity are minimally effective**
  - Statins, bile acid sequestrants, cholesterol absorption inhibitors, PCSK9 inhibitors
- **Current therapeutic options**
  - LDL apheresis
  - Juxtapid (lomitapide)
  - Kynamro (mipomersen)

28 year-old female with cutaneous xanthoma



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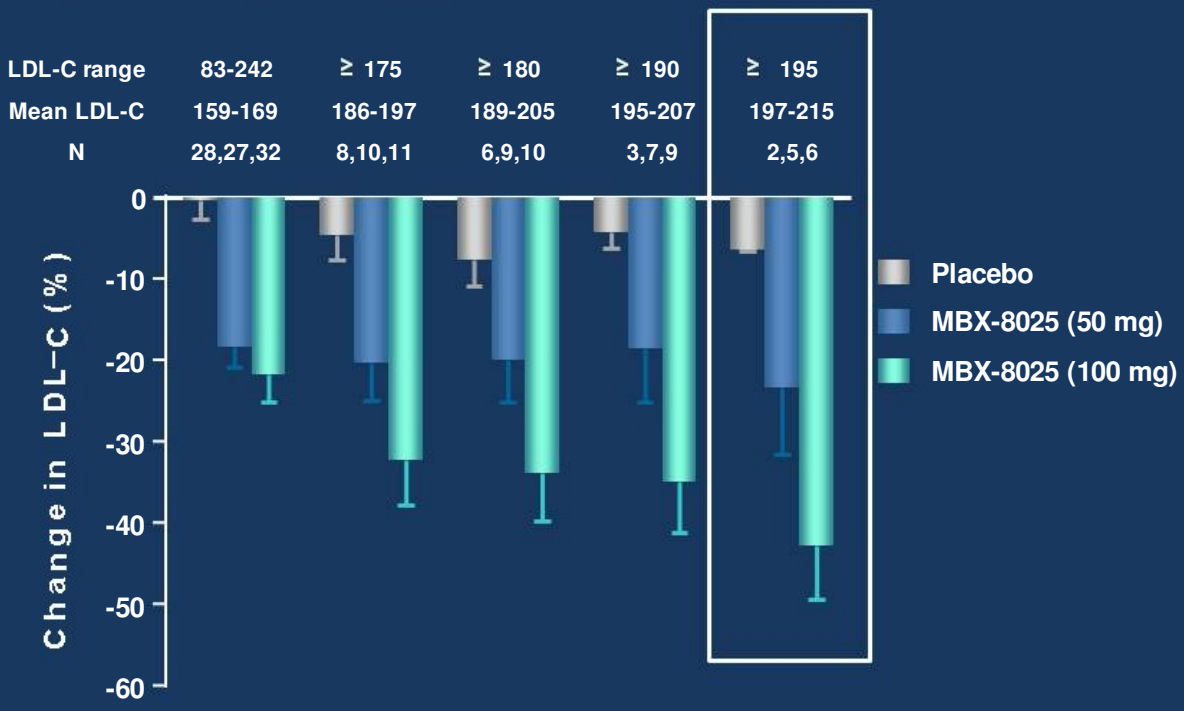
Unmet Medical Need in HoFH Remains High
Patients need more effective and better tolerated therapies

- **LDL apheresis is inconvenient and has many complications**

- **Juxtapid (Microsomal Transfer Protein Inhibitor)**
  - Only 28% of patients achieve LDL < 100 mg/dL
  - Dose limiting GI tolerability
  - Risk of hepatotoxicity due to increase in hepatic fat
  - Black box and REMs requiring monthly liver testing

- **Kynamro (oligonucleotide inhibitor of apo-B synthesis)**
  - Risk of hepatotoxicity due to increase in hepatic fat
  - Black box and REMs requiring monthly liver testing
  - Flu-like symptoms and injection site reactions

MBX-8025 Phase 2 Mixed Dyslipidemia Study
Change in LDL-C as a function of baseline LDL-C



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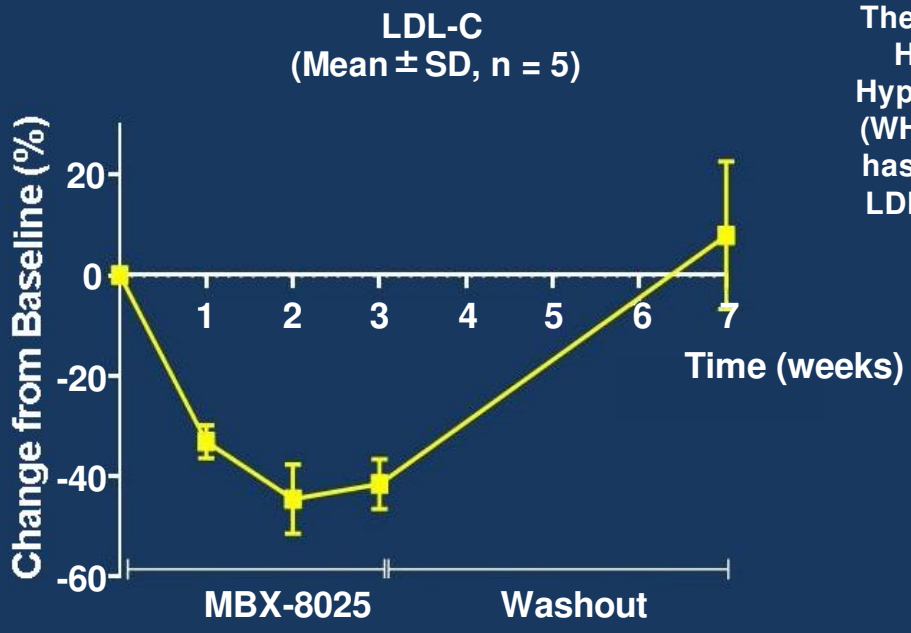
Effect of MBX-8025 in the WHHL Rabbit Model of HoFH
Significant and sustained reductions in LDL-C



The Watanabe Heritable Hyperlipidemic (WHHL) rabbit has low (<5%) LDL-R activity

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MBX-8025 for the Treatment of HoFH
Rationale and pilot study design

- **Rationale**
  - MBX-8025 exhibited a strong anti-atherogenic profile in patients with mixed dyslipidemia including reductions in LDL-C
  - Data from the WHHL rabbit model suggest that the decreases in LDL-C would be retained in the setting of low LDL-R activity characteristic of HoFH

- **Pilot study design**
  - Open label dose escalation study in up to 8 patients
  - Doses are 50, 100 and 200 mg
  - Study duration of 3 months
  - Study to be conducted in 2-3 countries in Europe
  - Start of study planned for 1H 2015

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CymaBay Projected Milestones

- **Arhalofenate**
  - Dose first patient in Phase 2b study — 1H 2014 ✓
  - Phase 2 febuxostat combo headline data — 1Q 2015 ✓
  - Phase 2b flare study headline data — 2Q 2015 ✓
  - End-of-Phase 2 meeting — 2H 2015
  - Start Phase 3 — 1H 2016

- **MBX-8025**
  - Select indication for proof-of-concept — 2H 2014 ✓
  - Start pilot study in HoFH — 1H 2015

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