

AFFIDAVIT OF MINA CHING

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5. The Internet Archive assigns a URL on its site to the archived files in the format `http://web.archive.org/web/[Year in yyyy][Month in mm][Day in dd][Time code in hh:mm:ss]/[Archived URL]` aka an "extended URL". Thus, the extended URL `http://web.archive.org/web/19970126045828/http://www.archive.org/` would be the URL for the record of the Internet Archive home page HTML file (`http://www.archive.org/`) archived on January 26, 1997 at 4:58 a.m. and 28 seconds (1997/01/26 at 04:58:28). The date indicated by an extended URL applies to a preserved instance of a file for a given URL, but not necessarily to any other files linked therein. Thus, in the case of a page constituted by a primary HTML file and other separate files (e.g., files with images, audio, multimedia, design elements, or other embedded content) linked within that primary HTML file, the primary HTML file and the other files will each have their own respective extended URLs and may not have been archived on the same dates.

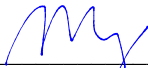




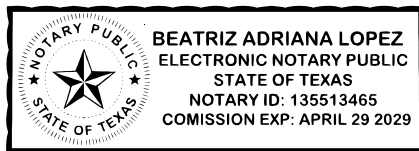
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7. Attached hereto as Exhibit B are true and accurate copies of the Internet Archive's records of the archived files for the URLs and the dates specified in the attached coversheet of each file.
8. I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

DATE: 03/21/2026


Mina Ching

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EXHIBIT A



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Jun 24, 2017	• Add Files Effects of AG-348, a pyruvate kinase activator, in patients with pyruvate kinase deficiency: Updated results from the DRIVE... 1.6 MB
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- - - - -	• Add Files



- Jun 06,2017 [Enasidenib in mutant IDH2 R/R AML: results of Phase 1 dose escalation and expansion study](#) 1 MB
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[ClarIDHy: a phase 3, multicenter, randomized, double-blind study of AG-120 vs placebo in patients with an advanced cholangiocarcinoma](#) 1.1 MB
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[Determination of IDH1 Mutational Burden and Clearance via Next-Generation Sequencing in Patients With IDH1 Mutation-Positive Hematologic Malignancies](#) 1.1 MB

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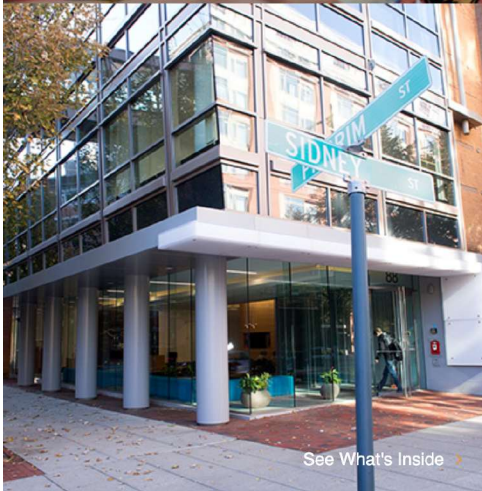


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What Drives Us?

The many friends and family of Agios employees affected by cancer are what motivates, inspires and drives us.

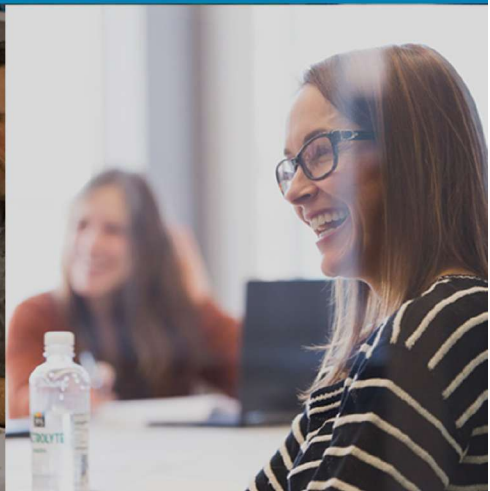
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Our Research Focus



Cancer Metabolism



Rare Genetic Diseases



Metabolic Immuno-Oncology



Inhibit key enzymes in cancer cell specific metabolic pathways to disrupt tumor cell proliferation and survival

Restore defective metabolic pathways in disease cells that cause rare genetic disorders of metabolism

Alter the metabolic state of immune cells to enhance the body's anti-tumor response

Boldly Pursuing Excellence and Innovation

We are a research-driven organization focused on creating transformative medicines by combining expertise in metabolism and precision medicine.

research platform



The Spirit of Agios

At Agios, our culture and values are embodied in the Greek word Arete, the ancient ideal of excellence and fulfillment of one's potential to which our employees aspire. Together, we are driven by a boundless passion for our work, and we pride ourselves on fostering an interconnected environment that encourages creative scientific collaboration across disciplines.

explore agios' culture





300+

Employees Making a Difference



100%

Committed to Helping Patients



3

Scientific Areas of Expertise

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OUR PIPELINE

Agios has leveraged its core capabilities in cellular metabolism and precision medicine to build a product engine that is focused in the therapeutic areas of cancer and rare genetic diseases.



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EXHIBIT B



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Effects of AG-348, a pyruvate kinase activator, in patients with pyruvate kinase deficiency: Updated results from the DRIVE PK study

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Wilma Barcellini⁵, D Holmes Morton⁶, Eduard van Beers⁷, Hassan Yaish⁸,
Yaddanapudi Ravindranath⁹, Kevin HM Kuo¹⁰, Sujit Sheth¹¹, Janet L Kwiatkowski¹²,
Bruce Silver¹³, Charles Kung¹⁴, Marvin Cohen¹⁵, Hua Yang¹⁴, Penelope A Kosinski¹⁴,
Lei Hua¹⁴, Ann Barbier¹⁴, Bertil Glader¹⁶

¹Dana-Farber Boston Children's Cancer and Blood Disorders Center, Boston, MA USA; ²Hammersmith Hospital, Imperial College Healthcare NHS Trust, London, UK; ³Unité des Maladies Génétiques du Globule Rouge, CHU Henri Mondor, Créteil, France; ⁴Hôpital Saint Vincent de Paul, Lille, France; ⁵Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, Milan, Italy; ⁶Central Pennsylvania Clinic, Belleville, PA, USA; ⁷Universitair Medisch Centrum Utrecht, Utrecht, Netherlands; ⁸University of Utah, Salt Lake City, UT, USA; ⁹Wayne State University School of Medicine, Children's Hospital of Michigan, Detroit, MI, USA; ¹⁰University of Toronto, Toronto, Canada; ¹¹Weill Cornell Medical College, New York, NY, USA; ¹²Children's Hospital of Philadelphia and Perelman School of Medicine of the University of Pennsylvania, Philadelphia, PA, USA; ¹³Bruce A Silver Clinical Science and Development, Dunkirk, MD, USA; ¹⁴Agios Pharmaceuticals, Inc., Cambridge, MA, USA; ¹⁵MBC Pharma Solutions, Newtown, PA, USA; ¹⁶Stanford University School of Medicine, Palo Alto, CA, USA

Presented at the 22nd Annual Meeting of the European Hematology Association, 22-25 June 2017, Madrid, Spain

Disclosures

- This study was funded by Agios Pharmaceuticals Inc.
- Rachael F Grace: Agios – study co-principal investigator and scientific advisor
- D Mark Layton: Agios – study investigator
- Frédéric Galactéros – study investigator
- Christian Rose: Agios – study investigator
- Wilma Barcellini: Agios – study investigator, no other disclosures
- D Holmes Morton – study investigator
- Eduard van Beers – study investigator
- Hassan Yaish – study investigator
- Yaddanapudi Ravindranath – study investigator
- Kevin HM Kuo – study investigator
- Sujit Sheth – study investigator
- Janet L Kwiatkowski: Agios – study investigator; Shire, Sideris, and Ionis Pharmaceuticals – consultant
- Bruce Silver, Marvin Cohen: Agios – consultants
- Bertil Glader: Agios – study co-principal investigator and scientific advisor
- Charles Kung, Hua Yang, Penelope A Kosinski, Lei Hua, Ann J Barbier: Agios – employees and stockholders
- Editorial assistance was provided by Christine Ingleby, PhD, Excel Scientific Solutions, Horsham, UK, and supported by Agios



Pyruvate kinase (PK) deficiency: a severe congenital anemia

Description

- Presents in childhood with severe hemolytic anemia

Etiology

- Caused by mutations in the *PK-LR* gene coding for erythrocyte pyruvate kinase (PK-R)

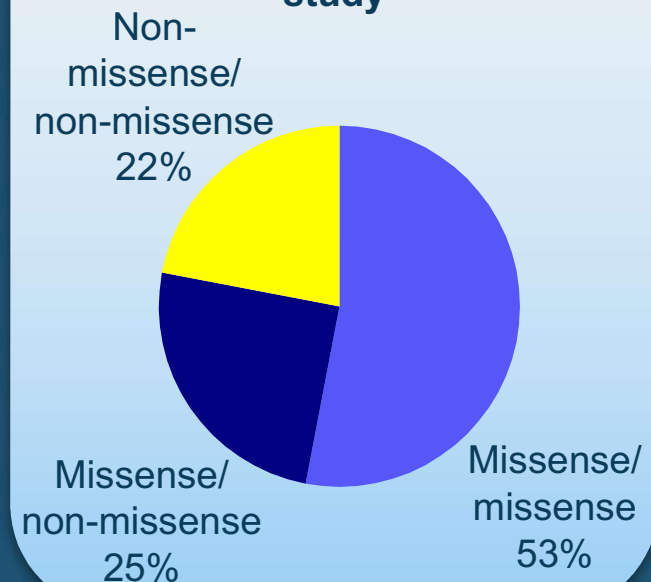
Disease Burden

- Lifelong hemolytic anemia
- Iron overload and jaundice
- Infection risk post-splenectomy

Diagnosis/ Treatment

- PK-R enzyme activity and/or genetic testing
- Supportive treatment: transfusions, splenectomy, iron chelation

Type of *PK-LR* mutations found in 74 unrelated cases enrolled in the PK deficiency natural history study



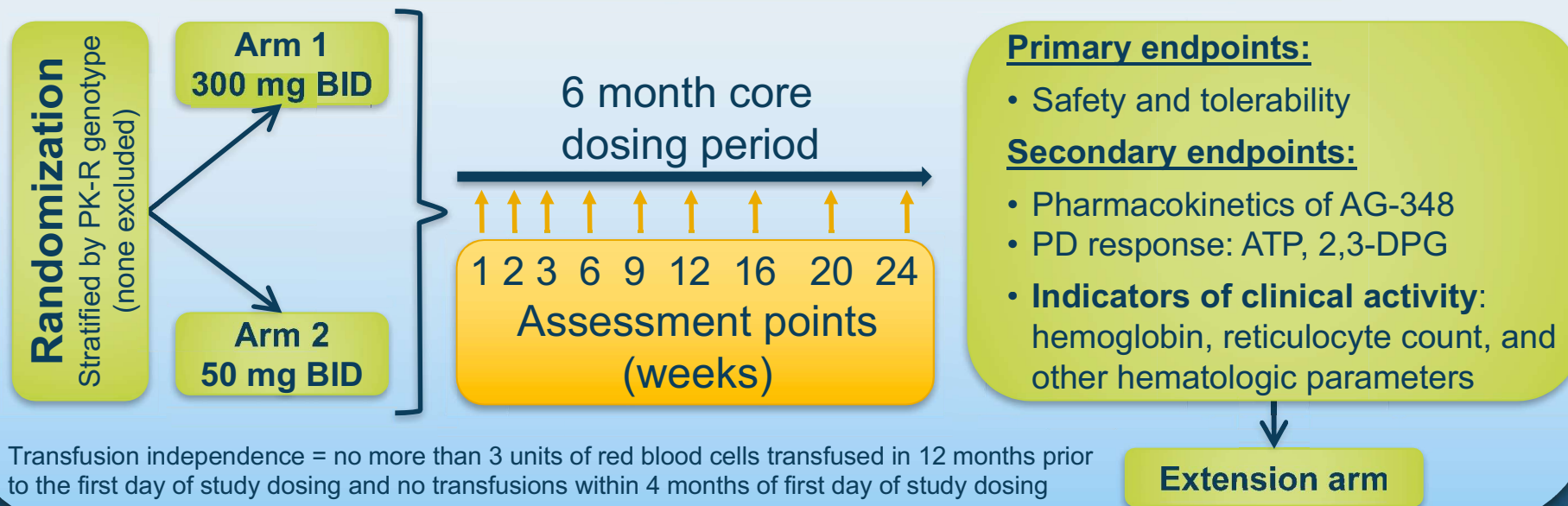
Study design



Open-label, global phase 2 study: 14 centers in the US, Canada, and EU

Transfusion-independent PK-deficient adults

(ClinicalTrials.gov NCT02476916) n=25 in each arm



Fully enrolled as of November, 2016

Demographics

Characteristics ^a	50 mg BID, n=27	300 mg BID, n=25	Total, N=52
Men, n (%)	18 (66.7)	14 (56.0)	32 (61.5)
Age at randomization in years, median (range)	29 (18, 58)	40 (21, 62)	34 (18, 62)
Race white ^b , n (%)	22 (81.5)	20 (80.0)	42 (80.8)
Hemoglobin (Hb) baseline, median (range)	9.6 (6.9, 12.3)	8.6 (6.5, 12.0)	8.9 (6.5, 12.3)
Duration of treatment, weeks, median (range)	23.0 (13.0, 76.9)	26.3 (12.9, 70.9)	24.9 (12.9, 76.9)
Splenectomized, n (%)	23 (85.2)	20 (80.0)	43 (82.7)
Iron chelation prior to enrolment, n (%)	14 (51.9)	11 (44.0)	25 (48.1)
Cholecystectomy, n (%)	19 (70.4)	19 (76.0)	38 (73.1)

^aData cut-off March 27, 2017; ^bNot reported in 3 patients, 3 patients were Asian, and 4 were "other"

Safety summary

- AG-348 was generally well tolerated
- The majority of adverse events (AEs) were grade 1–2
- Treatment related AEs leading to discontinuation, n=3
 - Chest discomfort/pleural effusion, pharyngitis/nausea, anemia
- 13 serious AEs in 10 patients
 - Six drug-related SAEs in 5 patients: Withdrawal hemolysis followed by anemia; anemia; osteoporosis; hypertriglyceridemia; pharyngitis
 - Grade 4 hypertriglyceridemia at Week 24, resolved upon AG-348 discontinuation (Grade 1 at baseline)

Patient disposition by study period	Ongoing, N	Completed, N	Discontinued ^a , N
Core	15	29 ^b	8
Extension	21	0	3

^aReasons for discontinuation: AEs (3), investigator decision (4), and withdrew consent (4)

^bFive subjects completing the Core period did not enter the Extension



Safety summary: Most common AEs

AEs, regardless of causality (occurring in >5 patients)	50 mg BID n=27		300 mg BID n=25		Total N=52	
	All grades	Grade ≥3	All grades	Grade ≥3	All grades	Grade ≥3
Patients experiencing at least 1 AE, n (%)	25 (92.6)	7 (25.9)	25 (100.0)	5 (20.0)	50 (96.2)	12 (23.1)
Headache	9 (33.3)	0	14 (56.0)	0	23 (44.2)	0
Insomnia	5 (18.5)	1 (3.7)	15 (60.0)	1 (4.0)	20 (38.5)	2 (3.8)
Nausea	10 (37.0)	0	9 (36.0)	0	19 (36.5)	0
Viral upper respiratory tract infection	7 (25.9)	0	2 (8.0)	0	9 (17.3)	0
Arthralgia	5 (18.5)	0	3 (12.0)	0	8 (15.4)	0
Fatigue	4 (14.8)	0	4 (16.0)	0	8 (15.4)	0
Back pain	4 (14.8)	0	3 (12.0)	0	7 (13.5)	0
Cough	4 (14.8)	0	3 (12.0)	0	7 (13.5)	0
Dizziness	4 (14.8)	0	3 (12.0)	1 (4.0)	7 (13.5)	1 (1.9)
Vomiting	3 (11.1)	0	4 (16.0)	0	7 (13.5)	0
Hot flush	1 (3.7)	0	6 (24.0)	0	7 (13.5)	0
Diarrhea	3 (11.1)	0	3 (12.0)	0	6 (11.5)	0
Influenza	5 (18.5)	1 (3.7)	1 (4.0)	0	6 (11.5)	1 (1.9)

Grade 3 AEs not reported in previous slide or table above: colitis (n=1), thrombosis (n=1), pharyngitis (n=1), post procedural hemorrhage (n=1), hypertension (n=1)

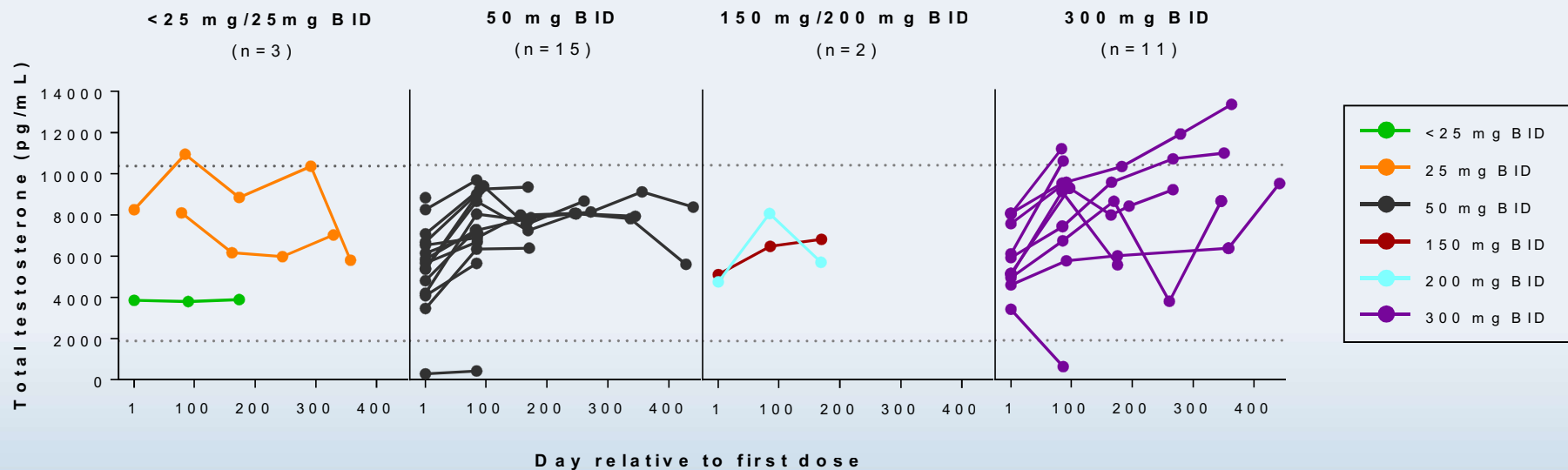
Dose titration in DRIVE PK

- Patients randomized to 50 mg or 300 mg BID AG-348 starting dose
 - Dose decreased:
 - AEs (e.g. insomnia)
 - Hb exceeding midpoint of normal range (Male: Hb >15.0 g/dL; Female>13.5 g/dL)
 - Dose increased:
 - Lack of Hb response
- Range of doses (5 mg QD–300 mg BID) used
- Efficacy and steroid hormone levels analyzed by the dose received for the longest duration in the Core period



Effect of AG-348 on hormones: total testosterone in men

- Preliminary findings are consistent with aromatase inhibition by AG-348 across multiple dose levels in men
 - Most testosterone values remained within the normal range
 - DEXA scan data inconclusive
 - Longer follow up required to assess clinical impact



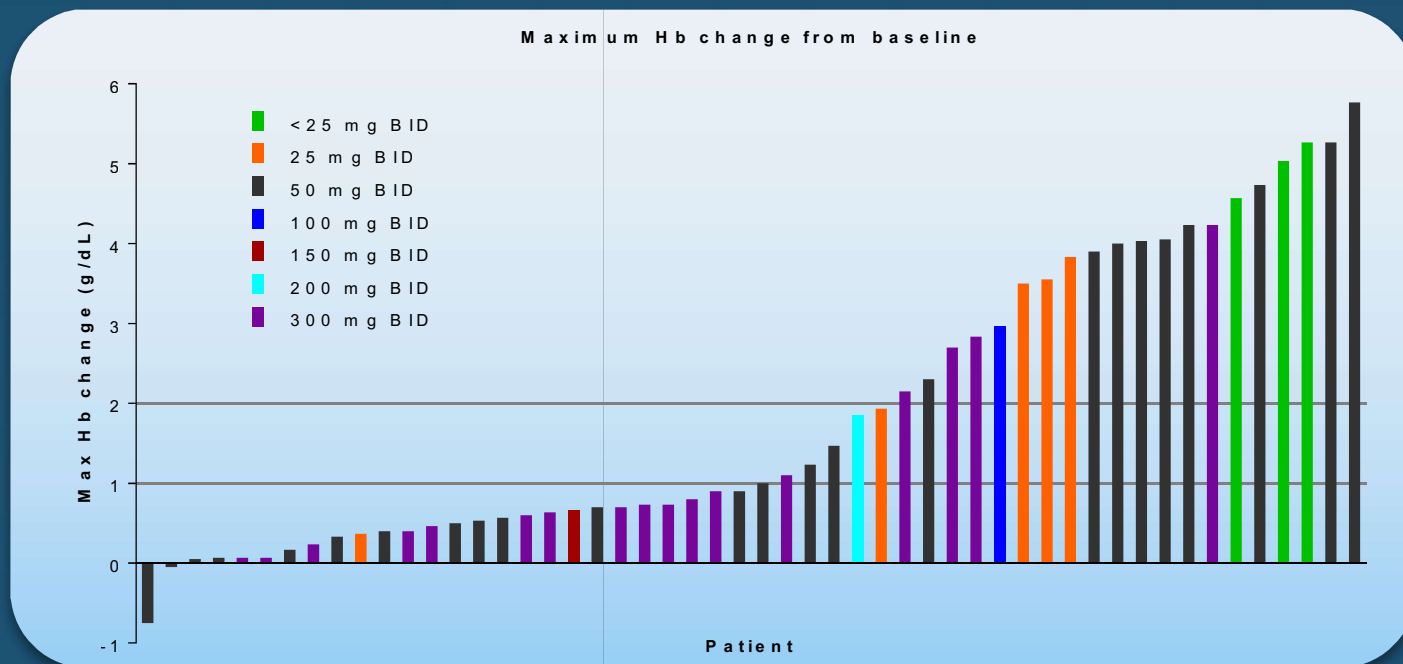
Normal reference low and high limits shown as horizontal dotted lines; DEXA = dual energy X-ray absorptiometry

Clinical activity results



Maximum Hb increase observed during the Core period

- 25/52 (48%) patients had a maximum Hb increase of >1.0 g/dL
 - The mean maximum increase was 3.5 g/dL (range 1.1-5.8 g/dL)

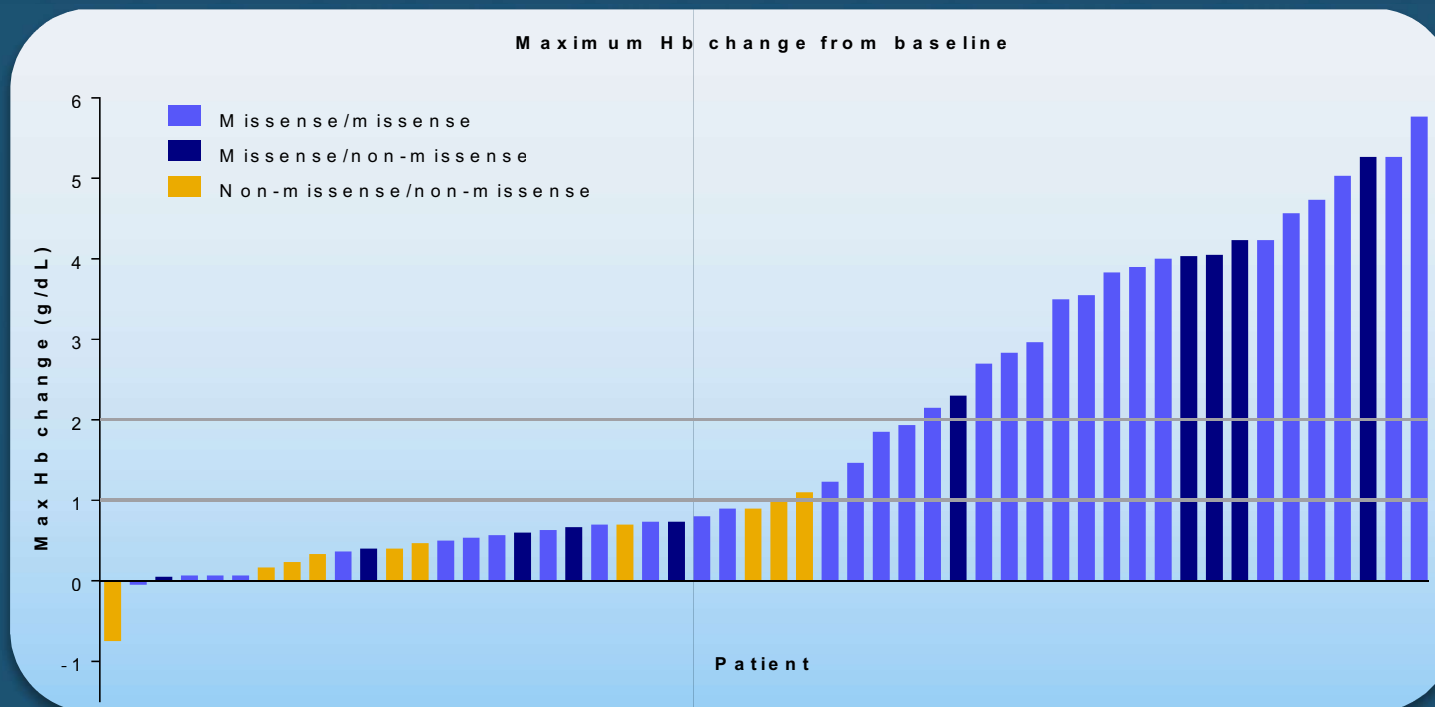


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The baseline value is the average of all central assessments within the screening period (42 days prior to Day 1)

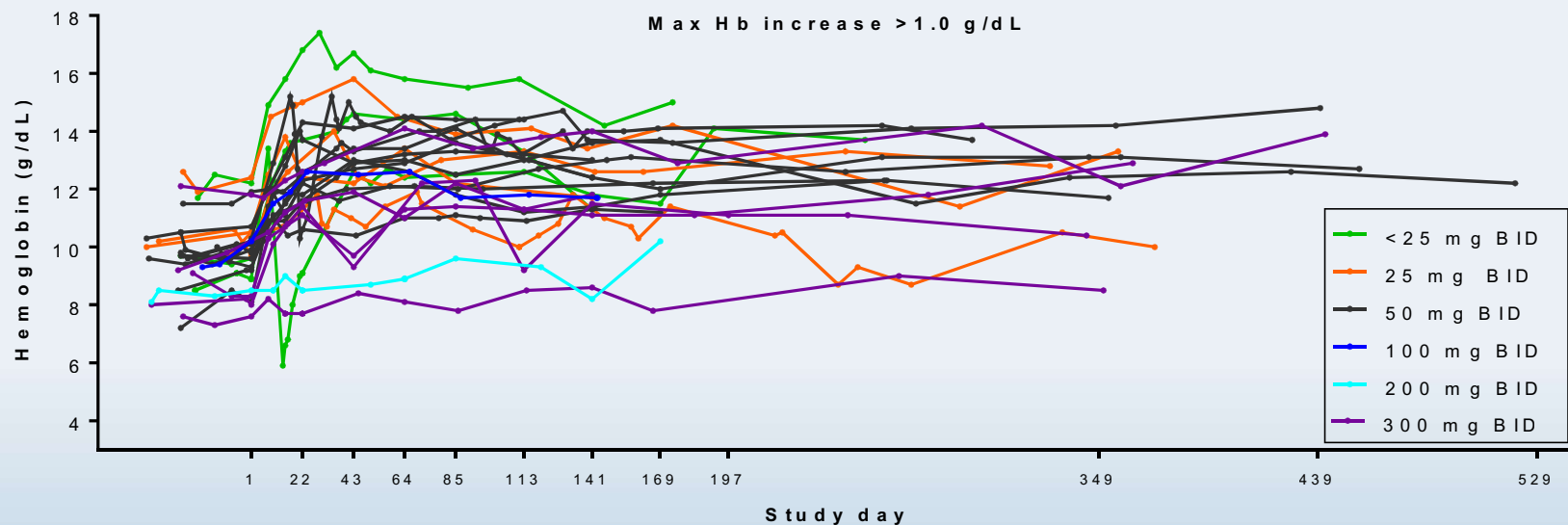
Maximum Hb increase observed by genotype

- 25/52 (48%) patients had a max increase in Hb >1.0 g/dL
 - 24/42 (57%) patients who had ≥ 1 missense mutation had Hb increase >1.0 g/dL



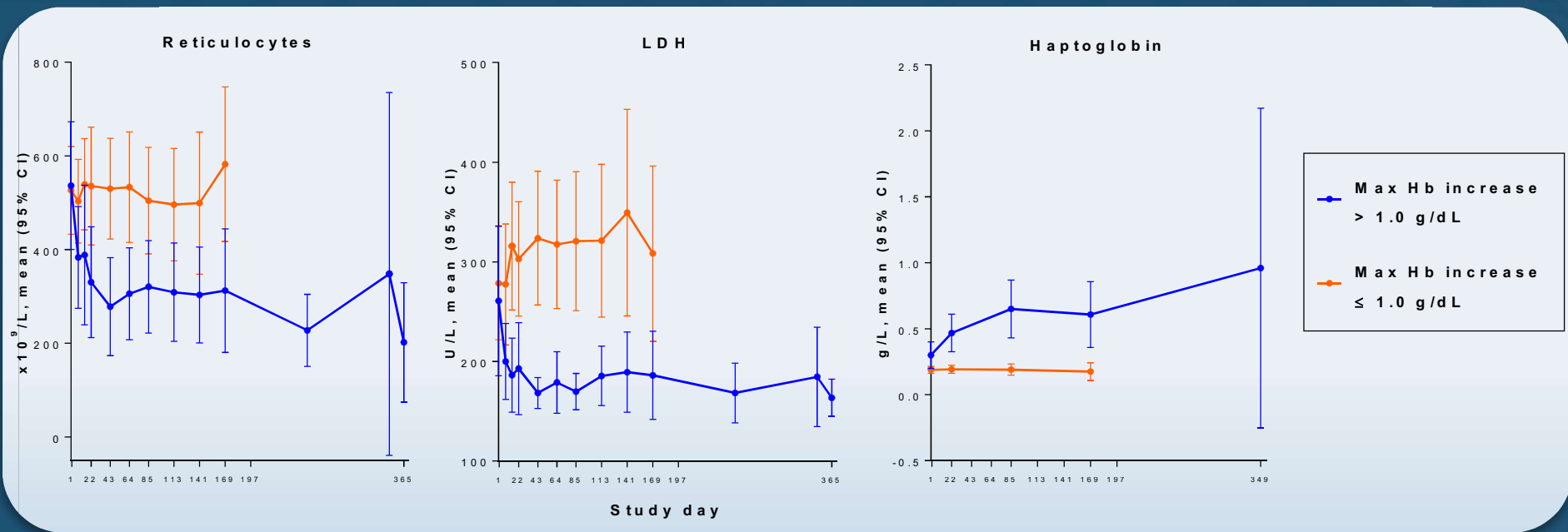
Majority of Hb increases are rapid and sustained

- Median time to the first observation of a Hb increase >1.0 g/dL above baseline was 10 days (range 7–141 days)
 - Median baseline Hb in subjects who experienced a maximum Hb increase of >1.0 g/dL was 9.7 g/dL (range 7.5–12.3 g/dL) vs. 8.0 g/dL (range 6.5–10.1) in subjects who did not
- In 8 patients, the dose had to be held or reduced due to rapid rise in Hb



Hemolysis markers improve in patients with Hb increase of >1.0 g/dL

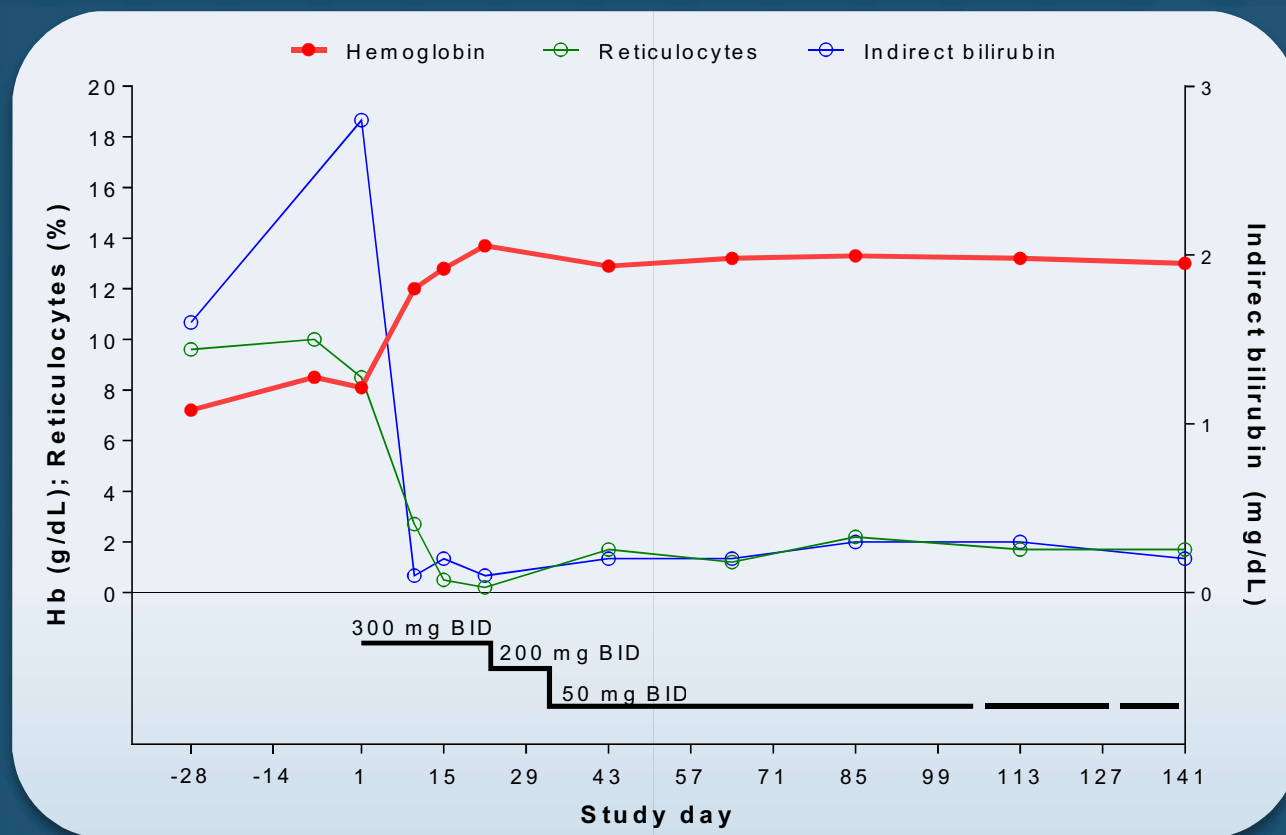
- Rapid decreases in reticulocytes and LDH in the first weeks of dosing
- Steady increase in haptoglobin



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 Only visits with 25 subjects included. Figure shows mean and laboratory results. LDH = lactate dehydrogenase

Hb response with improvements in hemolysis parameters: Single subject experience with AG-348

- 35 yo female,
white,
missense/missense
genotype
(T384M/R479H)



DRIVE-PK conclusions

- AG-348 is a novel, first-in-class PK-R activator in clinical testing as a potential disease-altering therapy for patients with PK deficiency
- Chronic daily dosing with AG-348 is well tolerated
 - Clinical significance of AG-348 aromatase inhibition is unclear
 - One grade 4 serious AE of elevated triglycerides was observed
- 25 of 52 (48%) subjects had a maximum Hb increase of >1.0 g/dL
 - Responses are rapid in onset and durable
 - Other parameters (reticulocytes, LDH and haptoglobin) indicate decreased hemolysis in responders
 - Some genotype–Hb response correlations were observed
- These data highlight the potential of AG-348 as the first disease-altering treatment for patients with PK deficiency, providing rationale for pivotal studies



Acknowledgments

- We would like to thank the patients who agreed to participate in this study
- We would also like to thank all the clinical research sites and study investigators:
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 - Stanford University Medical Centre; Bertil Glader
 - University of Utah; Hassan Yaish
 - Weill Cornell – New York Presbyterian Hospital; Sujit Sheth
 - The Children’s Hospital of Philadelphia; Janet L Kwiatkowski
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 - Wayne State University School of Medicine; Yaddanapudi Ravindranath
 - University of Toronto – University Health Network; Kevin HM Kuo
 - Hammersmith Hospital; D Mark Layton
 - Universitair Medisch Centrum Utrecht; Eduard van Beers
 - Hôpital Henri Mondor; Frédéric Galactéros
 - Hôpital Saint Vincent de Paul; Christian Rose
 - Hôpital de la Timone; Emmanuelle Bernit
 - Fondazione IRCCS Ca’ Granda, Ospedale Maggiore Policlinico; Wilma Barcellini
- We would like to thank Drs Ellis Neufeld and David Nathan for helpful discussions
- This clinical study was funded by Agios Pharmaceuticals



JURAT ATTACHMENT

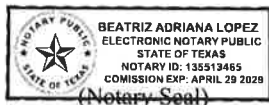
A notary public or other officer completing this certificate verifies only the identity of the individual who signed the document to which this certificate is attached, and not the truthfulness, accuracy, or validity of that document.

STATE OF Texas }

COUNTY OF Harris }

The foregoing instrument was subscribed and sworn before me this date of
03/21/2026, by Mina Ching

This notarial act was an online notarization.



Notary's Signature 

Registration No.: 135513465

Commission Expiration Date: April 29, 2029

