UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 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): December 4, 2016

Agios Pharmaceuticals, Inc.
(Exact Name of Registrant as Specified in Charter)

Delaware	001-36014	26-0662915
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

88 Sidney Street, Cambridge, MA	02139
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code: (617) 649-8600

(Former Name or Former Address, if Changed Since Last Report)

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 December 4, 2016, Agios Pharmaceuticals, Inc. (the "Company") issued a press release announcing new clinical data from both the Company's ongoing phase 2 DRIVE PK study evaluating AG-348 in patients with pyruvate kinase deficiency and the Company's ongoing phase 1 single ascending dose and multiple ascending dose study of AG-519 in healthy volunteers. On December 5, 2016, the Company issued a press release announcing new clinical data from the dose escalation portion of the Company's ongoing phase 1 study evaluating single agent AG-120 in patients with isocitrate dehydrogenase-1 mutant positive advanced hematologic malignancies. The Company presented these data at the 2016 American Society of Hematology Annual Meeting and Exposition taking place December 3-6, 2016 in San Diego, California.

The full text of the press releases issued in connection with these announcements are attached as Exhibit 99.1 and Exhibit 99.2 to this Current Report on Form 8-K and incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) The following exhibits are included in this report:

Exhibit No.	Description
99.1	Press release issued by Agios Pharmaceuticals, Inc. on December 4, 2016.
99.2	Press release issued by Agios Pharmaceuticals, Inc. on December 5, 2016.

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.

AGIOS PHARMACEUTICALS, INC.

Date: December 6, 2016

By: /s/ David P. Schenkein
David P. Schenkein, M.D.
President and Chief Executive Officer

EXHIBIT INDEX

Exhibit No.	Description
99.1	Press release issued by Agios Pharmaceuticals, Inc. on December 4, 2016.
99.2	Press release issued by Agios Pharmaceuticals, Inc. on December 5, 2016.

Exhibit 99.1

Agios Announces New Data from AG-348 and AG-519 Demonstrating Potential for First Disease-modifying Treatment for Patients with PK Deficiency

- AG-348 Is Well-Tolerated and Demonstrates Clinically Relevant, Rapid and Sustained Hemoglobin Increases in 15 of 26 Patients with at Least One Missense Mutation and 15 out of 32 Patients Overall -

- AG-519 Is Well-Tolerated and Demonstrates Robust Dose-Dependent Changes in ATP and 2,3-DPG Blood Levels in Normal Healthy Volunteers Consistent with PKR Enzyme Activation -

- Company to Host Investor Event and Webcast Today at 8:00 p.m. PT -

SAN DIEGO, December 4, 2016 — Agios Pharmaceuticals, Inc. (Nasdaq:AGIO) presented new data on two molecules from its wholly owned PKR activator program demonstrating the potential for the first disease-modifying treatment for patients with PK Deficiency at the 2016 American Society of Hematology Annual Meeting and Exposition (ASH). Agios' PKR program consists of AG-348, which is being evaluated in the Phase 2 DRIVE PK study in patients with pyruvate kinase (PK) deficiency, and AG-519, a second PKR activator being evaluated in an ongoing Phase 1 trial in healthy volunteers. PK deficiency is a rare, potentially debilitating, congenital anemia. Data from these studies were last reported at the Congress of the European Hematology Association (EHA) in June 2016.

Updated data from DRIVE PK with additional patients and longer follow-up demonstrate that 47% of all efficacy evaluable patients (n=15/32) and 58% of evaluable patients with at least 1 missense mutation (n=15/26) treated with AG-348 experienced a maximum hemoglobin (Hb) increase from baseline of >1.0 gram per deciliter (g/dL). Efficacy evaluable patients were required to have received AG-348 for at least three weeks. Hb increases >1.0 g/dL were observed in patients randomized to two doses and the maximum increase ranged from 1.2–5.2 g/dL with a mean maximum increase of 3.6 g/dL. Hb increases were also rapid and sustained, with a median time to a hemoglobin increase of >1.0 g/dL of 1.4 weeks. Agios also presented the first data demonstrating a direct link between increases in hemoglobin levels and activation of the PKR pathway (rate of metabolism) in patients treated with AG-348.

In addition, data from the ongoing Phase 1 trial in healthy volunteers establish that AG-519 is well-tolerated and demonstrates clear proof-of-mechanism as a potent, oral, selective PKR activator. The robust dose-dependent changes in ATP (adenosine triphosphate) and 2,3-DPG (2,3-diphosphoglycerate) blood levels reported with AG-519 are consistent with PKR enzyme activation. The activity of AG-519 as an activator of both wild-type and mutant forms of PKR is similar to that of AG-348.

"With data from additional patients and longer follow-up, it is highly encouraging that AG-348 continues to demonstrate robust, rapid and sustained increases in hemoglobin in patients with PK

deficiency," said Rachael Grace, M.D., of the Dana-Farber Boston Children's Cancer and Blood Disorder Center and a principal investigator for the study. "These findings are important for physicians and patients as they offer the potential for the first disease-modifying treatment for patients suffering from this chronic anemia and its associated complications."

"The rapid and sustained hemoglobin increases shown in DRIVE PK and AG-519's robust PK/PD profile continue to support moving a PKR activator into pivotal development next year," said Chris Bowden, M.D., chief medical officer at Agios. "In addition, our scientists continue to lead advances in the understanding of the biology of PK deficiency and have demonstrated the first data linking an increase in hemoglobin to direct activation of the PKR pathway."

Updated Phase 2 DRIVE-PK Data for AG-348

DRIVE PK is a global Phase 2, open-label safety and efficacy trial and the first study evaluating AG-348 in adult, transfusion-independent patients with PK deficiency. The study includes two arms of approximately 25 patients each, receiving a dose of 50 milligrams (mg) or 300 mg twice daily for at least six months (24 weeks). The target enrollment has been reached with a total of 52 patients enrolled. As of the September 23, 2016 data cut-off:

- Thirty-four patients had been treated in the study and are included in the safety analysis and 32 patients with at least 3 weeks of data are included in the efficacy analysis.
- Seventeen patients completed the initial 24 week treatment period.
- In the 32 patients for whom efficacy could be evaluated, the mean baseline Hb was 9.2 g/dL.
- Twenty-eight of the 34 patients (82%) had been splenectomized prior to study entry.

A safety analysis was conducted based on all 34 treated patients as of the data cut-off.

- AG-348 was well-tolerated, and the majority of treatment-related adverse events (AEs) were Grade 1-2; the most frequent being headache, nausea and insomnia.
- Two patients experienced serious adverse events (SAEs).
    - One Grade 2 AE of osteoporosis was previously reported in a patient with osteopenia at baseline assessment.
    - One patient experienced withdrawal hemolysis and anemia after AG-348 was temporarily discontinued due to a rapid treatment-related Hb increase, but stayed in the study and is continuing to receive treatment with AG-348 at a lower dose.
- Sex steroids were assessed at baseline, week 12 and week 24 for male and female patients. Increases in free testosterone and decreases in estradiol indicate aromatase inhibition by AG-348. Bone density scan data (n = 17) show high variability and are inconclusive. Clinical significance of the aromatase inhibition remains unclear.

In the efficacy analysis (n=32), 15 of 32 total evaluable patients and 15 of 26 evaluable patients with at least one missense mutation achieved rapid, robust and sustained hemoglobin increases from baseline of >1.0 g/dL as of the data cut-off.

- In patients who had hemoglobin increases of >1.0 g/dL, the mean maximum hemoglobin increase was 3.6 g/dL (range 1.2–5.2 g/dL).
- The median time to a hemoglobin increase of >1.0 g/dL was 1.4 weeks (range 1.1–21.0 weeks).
- Further data are needed to obtain a greater understanding of the relationship between genotype and response. Preliminary observations show:
    - Of the 26 evaluable patients with at least one missense mutation, 15 have shown an increase in hemoglobin of >1.0 g/dL.
    - None of the six patients with two non-missense mutations showed increases in hemoglobin of >1.0 g/dL.
    - Five patients homozygous for R479H (missense-missense) were also non-responders.
- Additional studies were conducted on the red blood cells of eight DRIVE PK patients.
    - In this subset, four patients who had hemoglobin level increases >1.0 g/dL on AG-348 experienced a greater than 50% average increase in the rate of metabolism of the PKR pathway.
    - None of the four patients with <1.0 g/dL increase experienced significant metabolic changes.

Phase 1 Healthy Volunteer Study Results for AG-519

Data were reported from four single ascending dose (SAD) cohorts of healthy volunteers (eight per group, 32 volunteers total) receiving daily doses of 50 mg, 250 mg, 750 mg or 1250 mg of AG-519 or placebo. Data were also reported from five multiple ascending dose (MAD) cohorts of healthy volunteers (eight per group, 40 volunteers total) dosed twice daily with 10 mg, 25 mg, 125 mg, 300 mg or 375 mg of AG-519 or placebo for 14 days.

- AEs from the SAD and MAD cohorts were mild or moderate (Grade 1 or 2) in severity, the most common being headache.
    - A single case of Grade 2 thrombocytopenia was previously reported in a subject receiving 375 mg of AG-519 q12hr, which resolved spontaneously within seven days after the last dose.
    - After the data cut-off, one ongoing SAE of drug-related cholestatic hepatitis was reported in the bioavailability and food effect study after a dose of 300 mg. This event is being further evaluated.
- Pharmacodynamic data from the MAD cohorts showed a mean decrease of up to 61% in blood 2,3-DPG levels and a mean increase of up to 63% in blood ATP levels from baseline. In contrast, healthy volunteers receiving placebo showed minimal changes in 2,3-DPG or ATP levels.
- Volunteers treated with AG-519 exhibited no changes in sex steroids levels, consistent with a lack of aromatase enzyme inhibition.

- The study is ongoing with final data not yet available for the bioavailability and food effect study and a Japanese volunteer cohort. The study allows for an optional additional open-label, multiple-dose cohort, which has not yet been initiated.

About Pyruvate Kinase Deficiency and Genetic Background

PK deficiency is a rare inherited disease that presents as hemolytic anemia, which is the accelerated destruction of red blood cells. The inherited mutations in PKR enzymes cause a deficit in cellular energy within the red blood cell, as evidenced by lower pyruvate kinase enzyme activity and a decline in ATP levels and a build-up of upstream metabolites, including 2,3-DPG.

The current standard of care for PK deficiency is supportive, including blood transfusions, splenectomy, chelation therapy to address iron overload and/or interventions for other treatment- and disease-related morbidities. There is no approved therapy to treat the underlying cause of PK deficiency.

PK deficiency is an autosomal recessive disease whereby all patients inherit two mutations, one from each parent. More than 250 different mutations have been identified to date. The mutations observed in PK deficiency patients are classified in two main categories. A missense mutation causes a single amino acid change in the protein, generally resulting in some functional protein. A non-missense mutation is any mutation other than a missense mutation, generally resulting in little functional protein. It is estimated that 53 percent of patients with PK deficiency have two missense mutations, 25 percent have one missense and one non-missense mutation, and 22 percent have two non-missense mutations[1].

Boston Children's Hospital, in collaboration with Agios, is conducting a Natural History Study to better understand the symptoms and complications of PK deficiency, identify patients and treatment centers, and capture other clinical data, including quality of life measures and genetic information.

About Agios' PKR Activators

Agios has discovered and is currently evaluating two orally available, potent, selective small molecule activators of PKR in clinical trials, AG-348 and AG-519. Agios scientists previously reported that AG-348 is a potent activator of the wild-type and mutated PKR enzymes in humans. Agios retains worldwide development and commercialization rights to AG-348 and AG-519.

Investor Event and Webcast Information

Agios will host an investor event on Sunday, December 4, 2016 beginning at 8:00 p.m. PT (11:00 p.m. ET) in San Diego to review data presented at ASH, including new data from the

ongoing studies of AG-348 and AG-519. The event will be webcast live and can be accessed under “Events & Presentations” in the Investors and Media section of the company’s website at www.agios.com.

About Agios

Agios is focused on discovering and developing novel investigational medicines to treat cancer and rare genetic metabolic disorders through scientific leadership in the field of cellular metabolism. In addition to an active research and discovery pipeline across both therapeutic areas, Agios has multiple first-in-class investigational medicines in clinical and/or preclinical development. All Agios programs focus on genetically identified patient populations, leveraging our knowledge of metabolism, biology and genomics. For more information, please visit the company’s website at www.agios.com.

Cautionary Note Regarding Forward-Looking Statements

This press release contains forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. Such forward-looking statements include those regarding: the potential benefits of Agios’ product candidates targeting pyruvate kinase-R mutations, including AG-348 and AG-519; Agios’ plans for the further clinical development of AG-348 and AG-519; and Agios’ strategic plans and prospects. The words “anticipate,” “believe,” “estimate,” “expect,” “intend,” “may,” “plan,” “predict,” “project,” “would,” “could,” “potential,” “possible,” “hope” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Such statements are subject to numerous important factors, risks and uncertainties that may cause actual events or results to differ materially from Agios’ current expectations and beliefs. For example, there can be no guarantee that any product candidate Agios is developing will successfully commence or complete necessary preclinical and clinical development phases; that positive safety and efficacy findings observed in early stage clinical trials will be replicated in later stage trials; or that development of any of Agios’ product candidates will successfully continue. There can be no guarantee that any positive developments in Agios’ business will result in stock price appreciation. Management’s expectations and, therefore, any forward-looking statements in this press release could also be affected by risks and uncertainties relating to a number of other important factors, including: Agios’ results of clinical trials and preclinical studies, including subsequent analysis of existing data and new data received from ongoing and future studies; the content and timing of decisions made by the U.S. FDA and other regulatory authorities, investigational review boards at clinical trial sites and publication review bodies; Agios’ ability to obtain and maintain requisite regulatory approvals and to enroll patients in its planned clinical trials; unplanned cash requirements and expenditures; competitive factors; Agios’ ability to obtain, maintain and enforce patent and other intellectual property protection for any product candidates it is developing; Agios’ ability to maintain key collaborations, such as its agreements with Celgene; and general economic and market conditions. These and other risks are described in greater detail under the caption “Risk Factors” included in Agios’ Quarterly Report on Form 10-Q for the quarter ended September 30, 2016, and other filings that Agios may

make with the Securities and Exchange Commission in the future. Any forward-looking statements contained in this press release speak only as of the date hereof, and Agios expressly disclaims any obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise.

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1 Bianchi P et al. poster, 2015 ASH Annual Meeting

Contacts

Investors:
Kendra Adams, 617-844-6407
Senior Director, Investor & Public Relations
Kendra.Adams@agios.com

Renee Leck, 617-649-8299
Senior Manager, Investor & Public Relations
Renee.Leck@agios.com

Media:
Dan Budwick, 973-271-6085
Senior Vice President, Media Relations
Pure Communications Inc.
dan@purecommunicationsinc.com

Exhibit 99.2

Agios Announces New Clinical Data from Dose-Escalation Portion of Phase 1 Trial of Single Agent AG-120 Showing Durable Molecular Responses in Patients with Advanced Hematologic Malignancies

- Overall Response Rate of 38% and Complete Remission Rate of 18% with Patients on Study up to 24.2 Months -

- For Relapsed/Refractory Acute Myeloid Leukemia Patients, Overall Response Rate of 33% and Complete Remission Rate of 16% with 6.5 Month Median Duration of Response -

- First Demonstration that Treatment with Single Agent AG-120 Can Result in Clearance of Mutant IDH1 -

SAN DIEGO, December 5, 2016 — Agios Pharmaceuticals, Inc. (Nasdaq:AGIO) presented new clinical data from the ongoing Phase 1 study evaluating single agent AG-120 in advanced hematologic malignancies at the 2016 American Society of Hematology Annual Meeting and Exposition (ASH). AG-120 is a first-in-class, oral, selective, potent inhibitor of mutant isocitrate dehydrogenase-1 (IDH1).

As of August 1, 2016, data from the completed dose-escalation portion of the Phase 1 trial from 78 patients with advanced IDH1 mutant positive hematologic malignancies treated with AG-120, including 63 patients with relapsed and/or refractory (R/R) acute myeloid leukemia (AML), continue to show a favorable safety profile and durable clinical activity. Data from the ongoing expansion phases were not reported. For all dose escalation patients, an overall response rate of 38% (30 of 78) and a complete remission rate of 18% (14 of 78) were observed. For the 63 R/R AML patients, the overall response rate and complete remission rates were 33% (21 of 63) and 16% (10 of 63) respectively. Patients were on study treatment for up to 24.2 months with a median duration of response of 10.2 months for all responders and 6.5 months for the R/R AML responding patients.

In order to study the depth of response to single agent AG-120, molecular detection of the mutant IDH1 burden in blood and bone marrow samples (collected at pre-treatment and at least one on-treatment time point) were analyzed using next generation sequencing (NGS, FoundationOne® Heme assay) in 67 patients from the dose-escalation portion of the study. Molecular clearance was defined as reduction of the IDH1 mutation below the limit of detection of the assay (1% for IDH). Molecular data show that treatment with AG-120 resulted in clearance of the IDH1 mutation in 36% of patients (5 of 14) in complete remission compared to 4% of patients (2 of 53) that did not achieve complete remission (p-value=0.003). This is the first demonstration that treatment with single agent AG-120 can result in mIDH1 clearance.

“AG-120 continues to demonstrate an impressive single-agent efficacy and safety profile in this cohort of high-risk relapsed or refractory AML patients, with some responses maintained for approximately two years,” said Courtney DiNardo, M.D., lead investigator and assistant

professor, department of leukemia at the University of Texas MD Anderson Cancer Center. “In addition, new molecular data for AG-120 suggests some patients experience clearance of the IDH1 mutant gene in their blood or bone marrow as assessed by next generation sequencing, demonstrating the depth of response that can occur with AG-120 therapy.”

“We are encouraged by the durable clinical activity of AG-120 and are working to bring this medicine to waiting patients with IDH1 mutant positive AML whose disease has progressed after standard treatments,” said Chris Bowden, M.D., chief medical officer of Agios. “We plan to explore a similar expedited regulatory strategy for AG-120 that is being utilized for enasidenib (AG-221), which could result in an NDA submission in 2017.”

Updated Phase 1 Dose-Escalation Data for AG-120 in Advanced Hematologic Malignancies

Clinical and molecular data reported are from 78 patients treated with AG-120 in the dose escalation phase of the ongoing Phase 1; data from the ongoing expansions were not reported. Doses were administered from 200 mg to 1,200 mg total daily doses. As of August 1, 2016, seven patients (9%) remain on treatment. The median age of these patients is 68 (ranging from 36-89). Patients received a median of two prior chemotherapy regimens (ranging from zero to five). A safety and efficacy analysis was conducted for all 78 treated dose-escalation patients. In addition, longitudinal mutant IDH1 (mIDH1) variant allele frequency (VAF) data were available for 67 patients.

Safety Data

A safety analysis conducted for all 78 treated patients as of the data cut-off shows that AG-120 continues to demonstrate a favorable safety profile.

- The majority of adverse events reported by investigators were mild to moderate, with the most common regardless of causality being fatigue, nausea, diarrhea, pyrexia and peripheral edema.
- Fifty-three patients experienced at least one serious adverse event (SAE), the majority being disease related.
- The maximum tolerated dose was not reached. The recommended Phase 2 dose was 500 mg once daily, which is being studied in the ongoing expansion phase of the trial.
- Nine patients discontinued from the study due to death, including one reported as possibly related to AG-120.
- All cause mortality at 30 and 60 days were 12% and 21%, respectively.

Efficacy Data

Thirty out of 78 treated patients achieved investigator-assessed objective responses for an overall response rate of 38%.

- Of the 30 patients who achieved an objective response, there were 14 (18%) complete remissions (CR), eight CRs with incomplete neutrophil recovery or platelet recovery (CRi/CRp), six marrow CR (mCR)/morphologic leukemia-free state (MLFS) and two partial remissions (PR).

- Of the 63 patients with R/R AML, 21 (33%) achieved an objective response, including 10 (16%) CRs, eight CRi/CRp, two MLFS and one PR.
- Responses were durable, with a median response duration of 10.2 months (3.7- not estimable (NE)) overall and 6.5 months (3.7-NE) in the subset of patients with R/R AML.
- Median duration of treatment is 3.2 months (ranging from 0.1 to 24.2 months).

IDH1 Mutational Clearance

Longitudinal mIDH1 VAF data were reported for 67 patients. Patients with IDH1 mutational clearance (IDH1-MC) were defined as having:

- mIDH1 detected at screening (any sample type), and
- no reported mIDH1 mutation in at least one on-study time point (FoundationOne® Heme sensitivity of 1%).

Importantly, IDH1-MC was observed in 36% of CRs (5 of 14) and 4% of non-CRs (2 of 53). IDH1-MC was enriched in patients achieving CR (p-value = .003). The median time to mutational clearance was 2.7 months (ranging from 1.1 to 3.8 months). This is the first demonstration that treatment with single agent AG-120 can result in mIDH1 clearance. Agios is continuing to study the potential relationship between IDH1-MC and clinical benefit for patients with AML.

About the Ongoing Phase 1 Trial for AG-120 in Advanced Hematologic Malignancies

AG-120 is being evaluated in an ongoing Phase 1 trial that includes a dose-escalation phase and four expansion arms, including:

- Arm 1: 125 IDH1 mutant positive AML patients who relapsed after bone marrow transplantation, are in second or later relapse, refractory to initial induction or reinduction treatment, or who relapse within one year of initial treatment, excluding patients with favorable-risk status
- Arm 2: 25 untreated IDH1 mutant positive AML patients who are not candidates for standard-of-care chemotherapy
- Arm 3: 25 patients with other non-AML IDH1 mutant, relapsed or refractory advanced hematologic malignancies
- Arm 4: 25 patients with relapsed IDH1 mutant positive AML not eligible for arm 1 who have failed or are unable to receive standard of care

About Variant Allele Frequency (VAF)

Sequencing studies have demonstrated that most tumors exhibit extensive intra-tumor genetic heterogeneity characterized by individual cells that have different somatic mutations. For single-nucleotide mutations, or variants, the VAF is defined as the fraction of DNA sequence reads covering the variant position that contains the variant allele. This technique makes it possible to infer the subpopulations of tumor cells by counting the number of DNA sequence reads that contain a specific somatic mutation.

About IDH Mutations and Cancer

IDH1 and IDH2 are two metabolic enzymes that are mutated in a wide range of hematologic and solid tumor malignancies. Normally, IDH enzymes help to break down nutrients and generate energy for cells. When mutated, IDH increases production of an oncometabolite 2-hydroxyglutarate (2HG) that alters the cells' epigenetic programming, thereby promoting cancer. 2HG has been found to be elevated in several tumor types. Agios believes that inhibition of the mutated IDH proteins may lead to clinical benefit for the subset of cancer patients whose tumors carry them.

About Acute Myelogenous Leukemia (AML)

AML, a cancer of blood and bone marrow characterized by rapid disease progression, is the most common acute leukemia affecting adults. Immature white blood cells known as myeloblasts, or "blasts" proliferate in the bone marrow rather than mature into normal blood cells. The decrease in normal blood cells can result in severe complications for patients including infections and dependence on blood product transfusions. AML incidence significantly increases with age, and according to the American Cancer Society, the median age of onset is 66. Less than 10 percent of U.S. AML patients are eligible for bone marrow transplant and the vast majority of patients do not respond to chemotherapy and progress to relapsed/refractory AML. The five-year survival rate for AML is approximately 20 to 25 percent. IDH1 and IDH2 mutations are present in about 15 to 23 percent of AML cases.

About Agios

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Contacts

Investors:
Kendra Adams, 617-844-6407
Senior Director, Investor & Public Relations
Kendra.Adams@agios.com

Renee Leck, 617-649-8299
Senior Manager, Investor & Public Relations
Renee.Leck@agios.com

Media:
Dan Budwick, 973-271-6085
Senior Vice President, Media Relations
Pure Communications Inc.
dan@purecommunicationsinc.com