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): November 4, 2020

KURA ONCOLOGY, INC.

(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-37620**	**61-1547851**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

12730 High Bluff Drive, Suite 400, San Diego, CA	**92130**
(Address of Principal Executive Offices)	**(Zip Code)**

Registrant's Telephone Number, Including Area Code: (858) 500-8800

N/A
(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 Instructions 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))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, par value $0.0001 per share	KURA	The Nasdaq Global Select Market

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

A Kura Oncology, Inc. (the "Company") abstract, which will be the subject of an oral presentation at the 2020 Annual Meeting of the American Society of Hematology ("ASH") on December 5, 2020, was posted on the ASH website on November 4, 2020.

The ASH abstract reports preliminary data from the Company's KOMET-001 Phase 1/2A clinical trial of KO-539, a potent, selective and oral small molecule inhibitor of the menin-KMT2A(MLL) interaction with downstream effects on HOXA9 and MEIS1 gene expression, in relapsed/refractory Acute Myeloid Leukemia ("AML"). The trial is using an accelerated, adaptive design with dose selection based on a modified toxicity probability interval. This trial design enables treatment of a single patient per dose level early on, exposing fewer patients to lower doses that are believed to be sub-therapeutic.

Although the first several escalations were conducted with single patient cohorts, the Company advanced to a more traditional 3 + 3 design for dose escalation, concurrent with the submission of the ASH abstract in early August. The Company increased the size of the cohorts because, in addition to encouraging safety and tolerability, the Company observed evidence of anti-leukemic activity and elected to gather data in a larger number of patients.

The Company anticipates sharing a more mature dataset, including preliminary data from approximately 10 patients with relapsed/refractory AML, at ASH on December 5, 2020, and is encouraged with the progress made with the study as KOMET-001 continues in dose escalation. Given the favorable safety and tolerability seen thus far, the Company now expects to determine a recommended Phase 2 dose for KO-539 in the first quarter of 2021.

The Company continues to add clinical sites in anticipation of moving into the expansion cohorts, pending additional clinical data. The planned expansion cohorts include NPM1-mutant AML and KMT2A(MLL)-rearranged AML – selected patient populations where the Company believes KO-539 has the potential to demonstrate increased clinical benefit.

In addition, the Company is exploring options to potentially broaden the opportunity in the treatment of acute leukemias in adults, as well as the combination of KO-539 with chemotherapy and targeted therapies in the front line.

A copy of the ASH abstract is furnished herewith as Exhibit 99.1. The information contained in this Current Report on Form 8-K under Item 7.01 and Exhibit 99.1 hereto are being furnished and shall not be deemed to be "filed" for the purposes of Section 18 of the Securities and Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section and will not be incorporated by reference into any registration statement filed by the Company, under the Securities Act of 1933, as amended, unless specifically identified as being incorporated therein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit Number	Description
99.1	ASH Abstract
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

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 thereunto duly authorized.

KURA ONCOLOGY, INC.

Date: November 4, 2020

By: /s/ James Basta

James Basta
Chief Legal Officer

Abstract accepted for oral presentation at the 62nd American Society of Hematology Annual Meeting

Preliminary data on a Phase 1/2A first in human study of the menin-KMT2A (MLL) inhibitor KO-539 in patients with relapsed or refractory acute myeloid leukemia

Eunice S. Wang[1], Jessica Altman[2], Kristen Petit[3], Stephane DeBotton[4], Roland Walter[5], Pierre Fenaux[6], Francis Burrows[7], Blake Tomkinson[7], Bridget Martell[7] and Amir T Fathi[8]

1. Roswell Park Comprehensive Cancer Center, Buffalo, NY
2. Northwestern Medical Faculty Foundation, Chicago IL
3. University of Michigan, Ann Arbor, MI
4. Institut Gustave Roussy Service d'Hématologie Clinique, France
5. Fred Hutchinson Cancer Research Center, Seattle, WA
6. Hospital Saint Louis, France
7. Kura Oncology, San Diego, CA
8. Massachusetts General Hospital, Harvard Medical School, Boston, MA

The histone-lysine-*N*-methyltransferase 2A (KMT2A) gene (formerly known as mixed-lineage leukemia (MLL)) plays an essential role in regulating gene expression including homeobox (HOX) and MEIS1 genes. In 5-10% of AML cases, specific KMT2A gene perturbations can occur which result in an aggressive and poor prognostic group of blood cancers. The KMT2A complex also appears to play a central role in the epigenetic dysregulation in AMLs with co-mutations such as *NPM1*, *IDH1/2*, *EZH2*, and *DNMT3A*. Therefore, there is strong rationale for targeting these AML subsets which may be exquisitely sensitive to inhibition of the menin-KMT2A chromatin complex.

KO-539 is a novel, once daily, oral investigational drug candidate targeting the menin-KMT2A protein-protein interaction.

KOMET-001 (NCT04067336) is an ongoing Phase 1/2A open-label study evaluating KO-539 in adult patients (pts) with relapsed and/or refractory AML agnostic to oncogenic mutational type. The Phase 1 dose-escalation objectives are to assess safety and tolerability, characterize the pharmacokinetics (PK), and determine a recommended Phase 2 dose. The Phase 2A dose expansion portion will assess anti-leukemic activity, PK, safety and tolerability in select genetic subtypes of AML. Preclinically, the drug is shown to be highly protein bound (>99%) across animal species. Using physiologically-based PK (PBPK) modeling, the estimated human efficacious dose was estimated to be 600 mg po qd.

As of data cutoff on August 10, 2020, 6 pts with relapsed and/or refractory AML have been enrolled in the trial. Dose escalation began with single pt cohorts at 50 mg po qd in 28 day cycles and has proceeded through to 200 mg dosing cohorts. An expansion of 3 pts at 200 mg was initiated to better characterize the PK and exposure of KO-539.

To date, 3 enrolled pts have been studied for safety and have not experienced any dose-limiting toxicities (DLTs) within the 28 day DLT-assessment window. Grade 3 (G3) or higher drug related adverse events have included G3 tumor lysis syndrome (TLS) at 50 mg and a G3 embolic event at 100 mg. KO-539 has been well tolerated with no dose interruptions or discontinuations due to drug related adverse events. There were no treatment-related deaths, and two pts discontinued treatment due to disease progression. Peak drug concentrations were attained between 2-3 hours after daily oral dosing with an elimination half-life of greater than 24 hours.

KO-539 has demonstrated evidence of biologic activity in pts in the first 3 dose levels treated to date. The 50 mg pt with a KMT2A-r and the 200 mg pt with a p53 mutation and PICALM-AF10 fusion exhibited evidence of tumor lysis syndrome and markedly decreased hydroxyurea requirements with blood count stabilization, respectively. A third pt (100 mg dose level) with SETD2 and RUNX1 co-mutations achieved a complete remission with confirmed negative MRD by flow cytometry after two cycles of therapy and continues on treatment. The biologic activity of KO-539 at lower doses may be explained by inhibition of the CYP3A4 enzyme by concomitantly administered azole antifungals. KO-539 is metabolized into at least two metabolites with comparable activity to KO-539; total drug concentrations (i.e., KO-539 plus active metabolites) exhibited a dose-dependent increase.

Although KO-539 is a CYP3A4 substrate, preclinical data suggest both KO-539 and its metabolites act as inhibitors, potentially providing an advantage in overcoming drug resistance attributable to CYP3A4 metabolism by bone marrow stroma. The physiology of the bone marrow sinusoids also allows both unbound and protein-bound drug to reach the sites of leukemic involvement. The high level of protein binding may therefore provide an opportunity for organ-specific targeted action while possibly limiting off target effects. The potential advantage associated with the CYP3A4 inhibitory characteristics of KO-539 to overcome drug-resistance in the bone marrow stroma also continues to be investigated.

In conclusion, the early biologic activity of KO-539 in relapsed AML is encouraging, and its unique PK characteristics may be advantageous for clinical benefit. In addition to the above, any updated safety, PK, and efficacy data will be presented at the time of the conference.