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 3, 2022

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 8.01 Other Events.

A Kura Oncology, Inc. (the "Company") abstract, which will be the subject of an oral presentation at the 2022 Annual Meeting of the American Society of Hematology ("ASH") on December 10, 2022 (the "ASH Meeting"), was posted on the ASH website on November 3, 2022.

The ASH abstract, which was submitted on August 2, 2022, using an early summer data cutoff, reports updated data from the Company's KOMET-001 clinical trial of ziftomenib and highlights the encouraging safety profile and clinical activity of ziftomenib in patients with relapsed/refractory acute myeloid leukemia ("AML"). The abstract includes 30 all-comer AML patients from the Phase 1a dose-escalation portion of the trial and 24 NPM1-mutant or KMT2A-rearranged AML patients from the Phase 1b portion, with 12 patients at 200 mg and 12 patients at 600 mg.

The Company anticipates sharing a more mature dataset from KOMET-001, including preliminary data from an additional 18 patients enrolled in a Phase 1b extension, during its oral presentation at the ASH Meeting.

A copy of the ASH abstract is filed herewith as Exhibit 99.1 and is incorporated herein 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 3, 2022

By: /s/ Teresa Bair

Teresa Bair
Chief Legal Officer

Update on a Phase 1/2 First-in-Human Study of the Menin-KMT2A (MLL) Inhibitor Ziftomenib (KO-539) in Patients with Relapsed or Refractory Acute Myeloid Leukemia

Harry P. Erba[1], Amir Fathi[2], Ghayas Issa[3], Jessica Altman[4], Pau Montesinos[5], Mrinal Patnaik[6], James Foran[7], Stephane DeBotton[8], Maria Baer[9], Gary Schiller[10], Roland Walter[11], Marina Kremyanskaya[12], Kristin Pettit[13], Stephen Strickland[14], Blake Tomkinson[15], Marilyn Tabachri[15], Mollie Leoni[15], Stephen Dale[15], Eunice Wang[16]

Histone-lysine-*N*-methyltransferase 2A (KMT2A) is a central regulator of target genes that drive leukemogenic transformation. *KMT2A* rearrangements (*KMT2A*r) and *nucleophosmin* 1 mutations (*NPM1*m), founding events in leukemia, occur in 5-10% and 30% of acute myeloid leukemia (AML) patients (pts), respectively. KMT2Ar functions in a protein complex that requires menin to cause epigenetic dysregulation in AML. KMT2Awt-menin also cooperates with NPM1m causing dysregulation of an overlapping set of leukemogenic genes dependent on KMT2A-menin interaction. Thus, the strong rationale for targeting menin in *KMT2A*r/*NPM1*m AML.

We present here preliminary KOMET-001 (NCT04067336) data, an ongoing Phase (P) 1/2 study of ziftomenib (KO-539), an inhibitor of KMT2A-menin interaction, in adult pts with relapsed/refractory (R/R) AML. Ziftomenib is dosed orally, once daily, in 28-day cycles.

P1a dose escalation enrolled 30 adult pts with R/R AML regardless of genotype. Pts received 50-1000 mg ziftomenib to assess safety, tolerability, pharmacokinetics, and anti-leukemic activity. Median age was 65.5 years (range [r] 22-85); 33%/13% had *KMT2A*r/*NPM1*m AML. Pts had a median of 3 prior therapies (r 1-10), with 17% having ≥ 1 prior stem cell transplant (SCT).

In P1a, treatment-emergent adverse events (TEAEs) ≥ Grade (Gr) 3 in ≥ 10% of all pts (N=30) were anemia, pneumonia (27%); neutropenia (17%); thrombocytopenia (13%); febrile neutropenia, decreased appetite (10%). Two dose-limiting toxicities (DLTs) occurred: pneumonitis (400 mg), differentiation syndrome (DS, 1000 mg); per protocol, the DLT in 1/1 pt at 1000 mg resulted in de-escalation.

Clinical benefit with disease control (eg, decreasing blast counts [BC] or hydroxyurea requirement) occurred across dose levels. At 100 mg, 1 complete remission (CR) was observed in a pt with *SETD2*, *RUNX1* mutations. At 200 mg, the 2 *NPM1*m pts responded (1 experienced a CR without measurable residual disease [MRD-] with >100 weeks duration (ongoing), and 1 had morphologic leukemic-free state [MLFS]). At 600 mg, 1 of 2 *KMT2A* pts had stable disease with significantly decreased BC lasting >4 months.

P1b dose-validation explored 2 P1a doses (200/600 mg) in 24 pts with *KMT2A*r/*NPM1*m AML to determine an optimal biologically active dose. *KMT2A*r/*NPM1*m pts randomized to 200/600 mg were 9/3 and 6/6. Median age 46 years (r 31-82), 62.5/37.5% had *KMT2A*r/*NPM1*m AML, and 33% had ≥1 prior SCT. The 200/600mg pts had a median of 2.5/4 prior therapies (r 1-12/2-8).

In P1b, TEAEs ≥ Gr 3 in ≥ 10% of all pts (N=24) were anemia, febrile neutropenia, neutropenia, thrombocytopenia (25% each); DS, leukocytosis (17% each); sepsis, leukopenia (13% each). At 200 mg (N=12), TEAEs ≥ Gr 3 in ≥ 10% were neutropenia, thrombocytopenia (33% each); febrile neutropenia, anemia, sepsis (25% each); DS, leukocytosis, respiratory failure (17% each). At 600 mg (N=12), TEAEs ≥ Gr 3 in ≥ 10% were febrile neutropenia, anemia (25% each); DS, leukocytosis, neutropenia, thrombocytopenia, leukopenia, diarrhea (17% each).

The on-target effect of DS occurred in 7 P1b pts. At 200 mg, 3 were *KMT2A* pts, of which 2 had events ≥ Gr 3 including 1 death. Of the 4 pts at 600 mg, 2 were Gr 3 (1 each *KMT2A*/*NPM1*), and 2 were Gr 2 (1 each *KMT2A*/*NPM1*). Since implementation of DS Guidance, reported DS event severity has declined.

P1b clinical efficacy was dose-dependent. At 200 mg, changes in bone marrow (BM) morphology and stable/decreasing BC were seen. Three pts were dose-escalated to 600 mg with improvement: 1 achieved a BM BC < 5% and significant reduction of high burden extramedullary disease despite persistence of a small disease focus; 1 had significantly reduced BC and disease control; 1 attained MLFS and remains on treatment. At 600 mg, 25% of P1b pts had a best response of CR/CR with partial hematologic recovery (CRh); 33.3% of *NPM1*m pts achieved CR/CRh. Composite CR was 33% with 75% MRD-. Overall response rate (ORR) was 42%. Pts who experienced DS had an ORR of 75%. At data cutoff, 50% of pts remain on treatment.

Overall, P1a results demonstrated a manageable safety profile and preliminary efficacy that informed the P1b doses to determine the optimal biologically active dose. P1b results suggest that with appropriate DS management, ziftomenib is well tolerated. Additionally, the 600 mg dose demonstrates meaningful signs of efficacy in heavily pretreated R/R AML pts, warranting further investigation of ziftomenib as a monotherapy and in combination with rational therapeutic partners.

Table 1: Preliminary Efficacy Data for the Phase 1b Portion of KOMET-001

	200 mg (N=12)	600 mg (N = 12)
CR/CRh Rate, n (%)[1]	0	3 (25.0)
95% CI[2]	(0.0, 26.5)	(5.5, 57.2)
Complete Remission Rate, n (%)	0	2 (16.7)
95% CI[2]	(0.0, 26.5)	(2.1, 48.4)
CRc Rate, n(%)[3]	0	4 (33.3)
95% CI[2]	(0.0, 26.5)	(9.9, 65.1)
MRD Negativity Rate, n (%)	0	3 (75.0)
95% CI[2]	(NA, NA)	(19.4, 99.4)
Overall Response Rate, n (%)[4]	0	5 (41.7)
95% CI[2]	(0.0, 26.5)	(15.2, 72.3)
MRD Negativity Rate, n(%)	0	3 (60.0)
95% CI[2]	(NA, NA)	(14.7, 94.7)

Abbreviations: CI = confidence interval; CR = complete remission; CRc = composite complete remission; CRh = complete remission with partial hematologic recovery; CRi = complete remission with incomplete hematologic recovery; CRp = complete remission with incomplete platelet recovery; MLFS = morphologic leukemia-free state; MRD = measurable residual disease; n/N = number of patients; NA = not applicable; ORR = overall response rate; PR = partial response.

[1] CR/CRh response rate is defined as the proportion of patients achieving a best overall response of CR or CRh.
[2] Clopper-Pearson 95% confidence intervals are calculated based on binomial distribution.
[3] CRc response rate is defined as the proportion of patients achieving a best overall response of CRi (including CRp), CRh, or CR.
[4] ORR is defined as the proportion of patients achieving a best overall response of MLFS, PR, CRi (including CRp), CRh, or CR.