Kura Oncology Announces Publication in Blood Highlighting Ziftomenib's Differentiated Binding Profile and Preclinical Activity

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– Publication details the discovery and preclinical characterization of ziftomenib, including its potency, selectivity, and mechanism of action –

– Findings demonstrate differentiated menin binding and activity against certain resistance-associated mutations, further supporting ziftomenib's potential across genetically defined acute leukemias –

SAN DIEGO, Aug. 10, 2026 (GLOBE NEWSWIRE) -- Kura Oncology, Inc. (Nasdaq: KURA), a biopharmaceutical company focused on precision medicines for cancer, today announced the publication of a manuscript in *Blood*, the flagship journal of the American Society of Hematology, detailing the discovery and preclinical development of ziftomenib, a potent and selective menin inhibitor approved by the U.S. Food and Drug Administration in 2025 for adult patients with relapsed or refractory *NPM1*-mutated acute myeloid leukemia (AML).

The publication describes the scientific foundation underlying ziftomenib's development, including its differentiated binding profile, potent and selective inhibition of the menin-KMT2A interaction, activity across multiple genetically defined leukemia models, and activity against certain treatment-emergent MEN1 mutations associated with resistance to other menin inhibitors.

"The publication in *Blood* captures the remarkable scientific journey that led to ziftomenib, from identifying the menin-KMT2A interaction as a compelling therapeutic target to designing a potent and selective inhibitor with the properties needed for clinical development," said Francis Burrows, Ph.D., Chief Scientific Officer of Kura Oncology. "The findings also provide important insight into the molecular properties that distinguish ziftomenib and supported its advancement from discovery through clinical development."

Researchers from the University of Michigan pioneered the discovery of small molecules that target the interaction between menin and KMT2A, which plays an important role in leukemias with genetic changes such as *NPM1* mutations and *KMT2A* rearrangements. A collaboration between the University of Michigan and Kura Oncology, initiated in 2014, ultimately led to the development of ziftomenib, a menin inhibitor that Kura Oncology subsequently advanced through clinical development and FDA approval.

Study findings in preclinical leukemia models
As reported in *Blood*, across *KMT2A*-rearranged, *NPM1*-mutated and *NUP98*-rearranged leukemia models, ziftomenib demonstrated potent on-target activity, including suppression of key KMT2A-regulated genes such as *MEIS1* and *HOXA9*, induction of differentiation, and reduced leukemia cell viability.

Ziftomenib also was shown to induce leukemia regression and extended survival in multiple xenograft and patient-derived xenograft models, including durable responses observed after treatment discontinuation in a patient-derived model.

The research further evaluated ziftomenib against treatment-emergent *MEN1* mutations associated with resistance to menin inhibition. Researchers found that ziftomenib retained activity against certain resistance-associated menin mutations while other menin inhibitors did not, providing additional insight into ziftomenib's differentiated binding profile and the molecular determinants of resistance. Clinical studies have shown a low frequency of treatment-emergent MEN1 resistance mutation with ziftomenib; in KOMET-001, MEN1-M3271 emerged in only 1 of 29 evaluable patients.[i]

"Ziftomenib represents the culmination of years of discovery, development, and dedication from an extraordinary team of scientists, clinicians, and colleagues who share a common goal: making a meaningful difference for patients," said Troy E. Wilson, Ph.D., J.D., President and Chief Executive Officer of Kura Oncology. "This publication provides important scientific validation of the properties that enabled ziftomenib to advance from an early discovery program to an approved medicine for adult patients with relapsed or refractory *NPM1*-mutated AML. More importantly, these findings reinforce our conviction in ziftomenib's potential as a foundational therapy across the AML treatment continuum as we continue to pursue combination strategies with established standards of care and development across molecularly defined patient populations."

Kura continues to advance ziftomenib as a potential foundational therapy across the AML treatment continuum by combining it with multiple standards of care in molecularly defined patient populations. These efforts are designed to build upon ziftomenib's established activity in *NPM1*-mutated AML and expand its potential benefit to additional patients with menin-dependent acute leukemias, which represent up to 50% of eligible patients.

About Ziftomenib
Ziftomenib (marketed as KOMZIFTI® in the U.S.) is a once-daily, oral menin inhibitor approved by the U.S. Food and Drug Administration for adult patients with relapsed or refractory acute myeloid leukemia (AML) with a susceptible *NPM1* mutation who have no satisfactory alternative treatment options. Ziftomenib is being studied across the AML treatment continuum, including in combination studies in newly diagnosed and relapsed or refractory *NPM1*-mutated AML, *KMT2A*-rearranged AML, and *FLT3*-mutated AML. Ziftomenib is also being explored in additional oncology indications, including advanced gastrointestinal stromal tumors.

About Kura Oncology
Kura Oncology is a biopharmaceutical company committed to realizing the promise of precision medicines for the treatment of cancer. Kura's pipeline of small molecule drug candidates is designed to target cancer signaling pathways and address high-need hematologic malignancies and solid tumors. Kura continues to pioneer advancements in menin inhibition and farnesyl transferase inhibition. For additional information, please visit the Kura website and follow us on X and LinkedIn.

Forward-Looking Statements

This news release contains certain forward-looking statements that involve risks and uncertainties that could cause actual results to be materially different from historical results or from any future results expressed or implied by such forward-looking statements. Such forward-looking statements include statements regarding, among other things, the potential therapeutic profile and clinical utility of ziftomenib; ziftomenib's potential as a foundational therapy across the AML treatment continuum; the potential significance of preclinical findings; the potential to expand the benefit of ziftomenib to additional patient populations; and Kura's ongoing and planned clinical development of ziftomenib, including combination strategies with established standards of care. Factors that may cause actual results to differ materially include the risk that preclinical findings may not be predictive of clinical results; risks related to the conduct, timing, enrollment, and results of clinical trials; regulatory developments; and other risks associated with the process of discovering, developing, and commercializing drugs that are safe and effective for use as human therapeutics, and the endeavor of building a business around such drugs. You are urged to consider statements that include the words "may," "will," "would," "could," "should," "believes," "estimates," "projects," "potential," "expects," "plans," "anticipates," "intends," "continues," "designed," "goal," or the negative of those words or other comparable words to be uncertain and forward-looking. For a further list and description of the risks and uncertainties the Company faces, please refer to the Company's periodic and other filings with the Securities and Exchange Commission, which are available at www.sec.gov. Such forward-looking statements are current only as of the date they are made, and Kura assumes no obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise.

IMPORTANT SAFETY INFORMATION FOR KOMZIFTI FROM THE U.S. PRESCRIBING INFORMATION

Boxed WARNING: DIFFERENTIATION SYNDROME

Differentiation syndrome, which can be fatal, has occurred with KOMZIFTI. Signs and symptoms may include fever, joint pain, hypotension, hypoxia, dyspnea, rapid weight gain or peripheral edema, pleural or pericardial effusions, pulmonary infiltrates, acute kidney injury, and rashes. If differentiation syndrome is suspected, interrupt KOMZIFTI, and initiate oral or intravenous corticosteroids with hemodynamic and laboratory monitoring until symptom resolution; resume KOMZIFTI upon symptom improvement.

WARNINGS AND PRECAUTIONS

Differentiation Syndrome

KOMZIFTI can cause fatal or life-threatening differentiation syndrome (DS). DS is associated with rapid proliferation and differentiation of myeloid cells. Symptoms of DS, including those seen in patients treated with KOMZIFTI, may include fever, hypoxia, joint pain, hypotension, dyspnea, rapid weight gain or peripheral edema, pleural or pericardial effusions, acute kidney injury, and rashes.

In the clinical trial, DS occurred in 29 (26%) of 112 patients with R/R AML with an *NPM1* mutation who were treated with KOMZIFTI at the recommended dosage. DS was Grade 3 in 13% and fatal in two patients. In broader evaluation of all patients with any genetic form of AML treated with KOMZIFTI monotherapy in clinical trials, DS occurred in 25% of patients. Four fatal cases of DS occurred out of 39 patients with *KMT2A*-rearranged AML treated with KOMZIFTI. KOMZIFTI is not approved for use in patients with *KMT2A*-rearranged AML.

In the 112 patients with an *NPM1* mutation, DS was observed with and without concomitant hyperleukocytosis, in as early as 3 days and up to 46 days after KOMZIFTI initiation. The median time to onset was 15 days. Two patients experienced more than one DS event. Treatment was interrupted and resumed in 15 (13%) patients, while it was permanently discontinued in 2 (2%) patients.

Prior to starting treatment with KOMZIFTI, reduce the WBC counts to less than 25 x 10^9/L. If DS is suspected, interrupt KOMZIFTI, initiate oral or intravenous corticosteroids (e.g., dexamethasone 10 mg every 12 hours) for a minimum of 3 days with hemodynamic and laboratory monitoring. Resume treatment with KOMZIFTI at the same dose level when signs and symptoms improve and are Grade 2 or lower. Taper corticosteroids over a minimum of 3 days after adequate control or resolution of symptoms. Symptoms of DS may recur with premature discontinuation of corticosteroid treatment.

QTc Interval Prolongation

KOMZIFTI can cause QTc interval prolongation. In the clinical trial, QTc interval prolongation was reported as an adverse reaction in 12% of 112 patients treated with KOMZIFTI at the recommended dosage for R/R AML with an *NPM1* mutation. QTc interval prolongation was Grade 3 in 8% of patients. The heart-rate corrected QT interval (using Fridericia's method) (QTcF) was greater than 500 msec in 9% of patients, and the increase from baseline QTcF was greater than 60 msec in 12% of patients. KOMZIFTI dose reduction was required for 1% of patients due to QTc interval prolongation. QTc prolongation occurred in 14% of the 42 patients less than 65 years of age and in 10% of the 70 patients 65 years of age or older.

Correct electrolyte abnormalities, including hypokalemia and hypomagnesemia, prior to treatment with KOMZIFTI. Perform an ECG prior to initiation of treatment with KOMZIFTI, and do not initiate KOMZIFTI in patients with QTcF > 480 msec. Perform an ECG at least once weekly for the first four weeks on treatment, and at least monthly thereafter. Interrupt KOMZIFTI if the QTc interval is > 500 ms or the change from baseline is > 60 ms (Grade 3). In patients with congenital long QTc syndrome, congestive heart failure, electrolyte abnormalities, or those who are taking medications known to prolong the QTc interval, more frequent ECG monitoring may be necessary. Concomitant use of KOMZIFTI with drugs known to prolong the QTc interval may increase the risk of QTc interval prolongation, result in a greater increase in the QTc interval and adverse reactions associated with QTc interval prolongation, including Torsades de Pointes, other serious arrhythmias, and sudden death.

Embryo-Fetal Toxicity

Based on findings in animals and its mechanism of action, KOMZIFTI can cause embryo-fetal harm when administered to a pregnant woman. Advise pregnant women of the potential risk to the fetus. Advise females of reproductive potential to use effective contraception during treatment with KOMZIFTI and for 6 months after the last dose. Advise males with female partners of reproductive potential to use effective contraception during treatment with KOMZIFTI and for 3 months after the last dose.

ADVERSE REACTIONS

Fatal adverse reactions occurred in 4 (4%) patients who received KOMZIFTI, including 2 with differentiation syndrome, 1 with infection, and 1 with sudden death. Serious adverse reactions were reported in 79% of patients who received KOMZIFTI. Serious adverse reactions occurring in ≥ 5% of

patients included infection without an identified pathogen (29%), febrile neutropenia (18%), bacterial infection (16%), differentiation syndrome (16%), and dyspnea (6%).

Dosage interruption of KOMZIFTI due to an adverse reaction occurred in 54% of patients. Adverse reactions that required dose interruption in ≥ 2% of patients included infection without an identified pathogen (15%), differentiation syndrome (13%), febrile neutropenia (5%), pyrexia (4%), electrocardiogram QT prolonged (4%), leukocytosis (4%), bacterial infection (3%), cardiac failure (2%), cholecystitis (2%), diarrhea (2%), pruritus (2%), and thrombosis (2%). Dose reduction of KOMZIFTI due to an adverse reaction occurred in 4% of patients. Permanent discontinuation of KOMZIFTI due to an adverse reaction occurred in 21% of patients. Adverse reactions that required permanent discontinuation of KOMZIFTI in ≥ 2% of patients were infection without an identified pathogen (8%), bacterial infection (4%), cardiac arrest (2%), and differentiation syndrome (2%).

Most common (≥ 20%) adverse reactions, including laboratory abnormalities, were aspartate aminotransferase increased (53%), infection without an identified pathogen (52%), potassium decreased (52%), albumin decreased (51%), alanine aminotransferase increased (50%), sodium decreased (49%), creatinine increased (45%), alkaline phosphatase increased (41%), hemorrhage (38%), diarrhea (36%), nausea (35%), fatigue (34%), edema (30%), bacterial infection (28%), musculoskeletal pain (28%), bilirubin increased (27%), potassium increased (26%), differentiation syndrome (26%), pruritus (23%), febrile neutropenia (22%), and transaminases increased (21%).

DRUG INTERACTIONS

Drug interactions may occur when KOMZIFTI is concomitantly used with:

- Strong or Moderate CYP3A4 Inhibitors: Monitor patients more frequently for KOMZIFTI-associated adverse reactions.
- Strong or Moderate CYP3A4 Inducers: Avoid concomitant use of KOMZIFTI.
- Gastric Acid Reducing Agents: Avoid concomitant use of KOMZIFTI with proton pump inhibitors (PPIs), H2 receptor antagonists (H2RAs), or locally acting antacids. If concomitant use with H2RAs or locally acting antacids cannot be avoided, modify KOMZIFTI administration time.
    - Take KOMZIFTI 2 hours before or 10 hours after administration of an H2 receptor antagonist.
    - Take KOMZIFTI 2 hours before or 2 hours after administration of a locally acting antacid.
- Drugs that Prolong the QTc Interval: Avoid concomitant use of KOMZIFTI. If concomitant use cannot be avoided, obtain ECGs when initiating, during concomitant use, and as clinically indicated. Interrupt KOMZIFTI if the QTc interval is > 500 ms or the change from baseline is > 60 ms.

USE IN SPECIFIC POPULATIONS

Pregnancy: Based on findings in animals and its mechanism of action, KOMZIFTI can cause embryo-fetal harm when administered to a pregnant woman. Advise pregnant women of the potential risk to a fetus. Verify pregnancy status in females of reproductive potential prior to starting KOMZIFTI.

Lactation: Because of the potential for adverse reactions in the breastfed child, advise women not to breastfeed during treatment with KOMZIFTI and for 2 weeks after the last dose.

Infertility: Based on findings in animals, KOMZIFTI may impair fertility in females and males of reproductive potential.

Please see full Prescribing Information, including **Boxed WARNING**.

Contacts

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[i] **Erba H, Wang E, Issa G, et al.** *Activity, Tolerability, and Resistance Profile of the Menin Inhibitor Ziftomenib in Adults With Relapsed/Refractory NPM1-Mutated AML.* **Clinical Lymphoma, Myeloma & Leukemia.** 2023;23(Suppl 1):S304-S305. doi: **10.1016/S2152-2650(23)01067-4**.