Nurix Therapeutics Announces New Preclinical Data Highlighting Breadth of Targeted Protein Degradation Pipeline at AACR 2026

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Data showcase advances across oncology pipeline including pan-mutant BRAF, CBL-B and AURKA programs

Presentations reinforce potential of degraders to overcome limitations of inhibitors and expand therapeutic reach

Featured AACR Advances session underscores Nurix's scientific leadership in targeted protein degradation and induced proximity pharmacology

BRISBANE, Calif., April 22, 2026 (GLOBE NEWSWIRE) -- Nurix Therapeutics, Inc. (Nasdaq: NRIX), a clinical-stage biopharmaceutical company developing targeted protein degradation therapies, today announced new preclinical data from multiple oncology programs at the American Association for Cancer Research (AACR) Annual Meeting 2026.

The presentations highlight continued progress across Nurix's oncology pipeline, including programs targeting pan-mutant BRAF, CBL-B and Aurora Kinase A (AURKA), as well as a featured AACR Advances session presentation highlighting the broader scientific progress and clinical translation of targeted protein degradation. Collectively, these data provide additional mechanistic validation of Nurix's approach to CBL-B, Aurora kinase A (AURKA) and mutant BRAF to address key limitations of traditional approaches, including resistance, incomplete pathway suppression, and inability to target non-enzymatic protein functions.

"These data, together with our participation in the AACR Advances session, highlight the growing clinical and scientific validation of targeted protein degradation as a new therapeutic modality," said Arthur T. Sands, M.D., Ph.D., president and chief executive officer. "Across multiple programs, we are seeing consistent evidence that these therapies can drive deeper and more durable biological responses, supporting their potential to deliver meaningful benefit for patients."

AACR Advances Session
Later today, April 22, 2026, Gwenn Hansen, Ph.D., chief scientific officer of Nurix, will present *"Designing Effective Degrader Therapeutics: What Early Clinical Experience Has Taught Us" as* part of the AACR Advances *session "Induced Proximity Pharmacology: Degraders and Beyond."* Dr. Hansen's remarks will provide a broad perspective on recent advances in targeted protein degradation, including insights from early clinical experience and the evolving potential of induced proximity approaches to expand the druggable target space and improve therapeutic outcomes.

Pan-Mutant BRAF Degrader Program
In a poster presentation titled *"NRX-0305, an orally bioavailable, CNS penetrant pan-mutant BRAF degrader demonstrates robust efficacy in intracranial models of melanoma brain metastasis and primary glioma,"* Nurix reported that NRX-0305 achieves dose-proportional pharmacokinetics across plasma, tumor, and brain, enabling robust degradation of mutant BRAF and downstream pathway inhibition. These properties translate into potent antitumor activity in intracranial glioma and melanoma models while selectively sparing wildtype BRAF and avoiding paradoxical MAPK pathway activation. In a clinically relevant BRAF inhibitor–resistant melanoma brain metastasis patient-derived xenograft (PDX) model, NRX-0305 significantly extended survival versus both vehicle and dabrafenib, delivering a 142% increase in lifespan, compared with approximately 12% for the approved BRAF inhibitor.

Additional data were presented in a poster titled *"NRX-0305 is an orally bioavailable, pan-mutant BRAF degrader that exhibits single-agent and combination efficacy with MEKi or anti-EGFR across Class 1/2/3 BRAF-mutant cancers."* In preclinical tumor models, NRX-0305 demonstrates broad activity across mutant BRAF classes, including activity across 14 PDX models spanning Class 1 treatment-resistant, Class 2, and Class 3 BRAF mutations. Combination of NRX-0305 with MEK inhibitors or anti-EGFR therapy enhanced tumor regressions in Class 2 and drove complete responses in Class 1 and 3 models. Notably, the complete regressions are achieved at lower MEK inhibitor dose levels, supporting the potential for an improved therapeutic window relative to current treatment approaches.

CBL-B Program
In an oral presentation titled *"Discovery and characterization of CBL-B intramolecular glue inhibitors that increase T cell activation and suppress tumor growth,"* Nurix reported the discovery and characterization of novel intramolecular glue inhibitors targeting CBL-B, an E3 ubiquitin ligase that negatively regulates T, B, and NK cell activation. Using mechanism-agnostic screening assays guided by CBL-B biology, Nurix identified a novel series of intramolecular glue inhibitors that stabilize the closed, inactive conformation of CBL-B, representing a first-in-class mechanism of action. Through structure-guided optimization, this series was

advanced to NX-1607, a potent and selective CBL-B inhibitor with sub-nanomolar binding affinity. In preclinical studies, NX-1607 enhanced T cell activation, as evidenced by increased IL-2 and IFN-γ secretion in response to TCR stimulation, and demonstrated single-agent anti-tumor activity across multiple syngeneic tumor models, including colorectal, triple-negative breast cancer, and B cell lymphoma. NX-1607 also synergized with anti-PD-1 therapy to significantly enhance survival across multiple models. Early clinical data demonstrated dose-dependent pharmacokinetics and modulation of the proximal pharmacodynamic biomarker pHS1 in CD8 T cells, providing initial evidence of target engagement in patients.

Aurora Kinase A (AURKA) Degrader Program
In a poster presentation titled *"NRX-4972, a selective, oral, Aurora kinase A degrader, demonstrates increased efficacy in an SCLC tumor model, and greater in vitro synergy than an AURKA inhibitor,"* Nurix reported new data demonstrating that targeted degradation of AURKA enables more complete biological modulation compared to inhibition alone. NRX-4972 exhibits central nervous system penetration and a favorable pharmacokinetic and pharmacodynamic profile, translating into superior antitumor activity in aggressive small cell lung cancer models, particularly with an optimized twice-daily dosing regimen. In the H82 SCLC model, twice-daily administration of NRX-4972 resulted in 60% of mice surviving to the end of the study, whereas none of the mice treated with AURKA inhibitors alisertib or LY3295668 survived. Mechanistically, degradation of AURKA results in downregulation of MYC and enhanced induction of DNA damage, apoptosis, and G2/M arrest. NRX-4972 also demonstrated broader and more potent synergy than an AURKA inhibitor in an in vitro screen of combination agents across triple-negative breast cancer, SCLC, and NSCLC cell lines, further supporting its therapeutic potential.

About NRX-0305
NRX-0305 is a potent, selective, and orally bioavailable central nervous system (CNS)-penetrant pan-mutant BRAF degrader that Nurix is exploring for use in oncology. Nurix has reported preclinical data demonstrating potent anti-tumor activity in multiple cell line-derived and patient-derived xenograft disease models representing Class 1, Class 2, and Class 3 B-RAF mutations. Anti-tumor activity was also observed in the setting of CNS disease and treatment-resistance, suggesting the potential for utility across a broad range of solid tumor types.

About NX-1607
NX-1607 is an investigational first-in-class oral inhibitor of the E3 ligase Casitas B-lineage lymphoma proto-oncogene B (CBL-B) being developed for immuno-oncology indications, including a range of solid tumor types. CBL-B is a cytoplasmic E3 ubiquitin ligase that negatively regulates T cell activation, making it an attractive target for immuno-oncology and offering a novel therapeutic approach to treat solid tumors. Inhibition of CBL-B in preclinical studies reverses T cell exhaustion, alleviates tumor induced immunosuppression, and may also exert direct antitumor effects. Nurix is evaluating NX-1607 in an ongoing Phase 1 trial in adults in a range of oncology indications. This study includes a thorough investigation of both dose and schedule in the Phase 1a portion. Additional information on the NX-1607 clinical trial can be accessed at www.clinicaltrials.gov (NCT05107674).

About NRX-4972
NRX-4972 is a CNS-penetrant, orally bioavailable and highly selective degrader of Aurora A kinase (AURKA). AURKA is an oncogene frequently overexpressed in adult solid tumors, hematologic malignancies, and pediatric cancers. Several AURKA inhibitors are effective in preclinical tumor models, but this activity has failed to translate into clinical efficacy. To address the limitations of inhibitors, Nurix has designed bifunctional targeted protein degraders of AURKA that enable removal of both enzymatic and scaffolding functions.

About Nurix Therapeutics, Inc.
Nurix Therapeutics is a clinical stage biopharmaceutical company focused on the discovery, development and commercialization of targeted protein degradation medicines, the next frontier in innovative drug design aimed at improving treatment options for patients with cancer and autoimmune diseases. Nurix's wholly owned, clinical stage pipeline includes degraders of Bruton's tyrosine kinase (BTK), a B-cell signaling protein, and inhibitors of Casitas B-lineage lymphoma proto-oncogene B (CBL-B), an E3 ligase that regulates activation of multiple immune cell types including T cells and NK cells. Nurix also is advancing multiple potentially first-in-class or best-in-class degraders and degrader antibody conjugates (DACs) in its preclinical pipeline. Nurix's partnered drug discovery pipeline consists of a preclinical stage degrader of STAT6 in collaboration with Sanofi, a clinical stage degrader of IRAK4 in collaboration with Gilead, as well as multiple additional programs under collaboration agreements with Gilead Sciences, Inc., Sanofi S.A. and Pfizer Inc., within which Nurix retains certain options for co-development, co-commercialization and profit sharing in the United States for multiple drug candidates. Powered by an AI-integrated discovery engine capable of tackling virtually any protein class, and coupled with unparalleled ligase expertise, Nurix has built a formidable advantage in translating the science of targeted protein degradation into clinical advancements. Nurix aims to establish degrader-based treatments at the forefront of patient care, writing medicine's next chapter with a new script to outmatch disease. Nurix is headquartered in San Francisco, California. For additional information visit http://www.nurixtx.com.

Forward-Looking Statements
This press release contains forward-looking statements within the meaning of the U.S. Private Securities Litigation Reform Act of 1995 and other federal securities laws. Any statements contained herein that do not describe historical facts are forward-looking statements that involve risks and uncertainties that could cause actual results to differ materially from those discussed in such forward-looking statements. Such risks and uncertainties include, among others, the risks described under the heading "Risk Factors" in Nurix's Quarterly Report on Form 10-Q for the fiscal period ended February 28, 2026, and subsequent filings with the SEC. Any of these risks and uncertainties could materially and adversely affect Nurix's business and results of operations, which could, in turn, have a significant and adverse impact on Nurix's stock price. Nurix cautions you not to place undue reliance on any forward-looking statements, which speak only as of the date they are made. Nurix undertakes no obligation to update publicly any

forward-looking statements to reflect new information, events or circumstances after the date they were made or to reflect the occurrence of unanticipated events.

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