



Nurix Therapeutics Presents New Preclinical and Phase 1 Translational Data Supporting Bexobrutideg (NX-5948) in Chronic Spontaneous Urticaria at the 2026 Society for Investigative Dermatology Annual Meeting

May 14, 2026

In preclinical studies of CSU, Bexobrutideg demonstrates potent BTK degradation across key immune cell types and enhanced suppression of FcεRI-driven pathology versus remibrutinib

In healthy volunteers, both single- and multiple-ascending-dose oral dosing of bexobrutideg led to rapid and robust BTK degradation in blood and skin

Phase 1 studies support continued exploration of new tablet formulation in inflammation and immunology indications

BRISBANE, Calif., May 14, 2026 (GLOBE NEWSWIRE) -- Nurix Therapeutics (Nasdaq: NRIX), a clinical-stage biopharmaceutical company focused on the discovery, development and commercialization of targeted protein degradation medicines, today announced the presentation of new preclinical and Phase 1 translational data highlighting the potential of bexobrutideg (NX-5948), the company's wholly owned Bruton's tyrosine kinase (BTK) degrader, in chronic spontaneous urticaria (CSU). The data are being presented at the 2026 Society for Investigative Dermatology Annual Meeting, taking place May 13–16, 2026, in Chicago, Illinois.

The findings demonstrate that targeted degradation of BTK may provide broader and deeper suppression of pathogenic immune signaling than BTK inhibition alone, supporting the future clinical development of bexobrutideg in inflammation and immunology indications.

"These data reinforce the potential of targeted protein degradation to deliver a fundamentally different level of control over immune-mediated disease biology," said Arthur T. Sands, M.D., Ph.D., president and chief executive officer of Nurix Therapeutics. "By catalytically eliminating BTK, rather than transiently inhibiting it, a single molecule of bexobrutideg can degrade thousands of BTK proteins per hour. Importantly, we have demonstrated virtually complete BTK degradation in both blood and skin of healthy volunteers utilizing our new tablet formulation. Bexobrutideg has the potential to provide deeper and more durable suppression of pathogenic signaling, with the goal of delivering meaningful new treatment options for patients with chronic inflammatory diseases."

"The results highlight a categorical advantage of the degrader approach. While BTK inhibitors can only block kinase activity, complete protein removal by bexobrutideg eliminates both kinase and scaffolding functions simultaneously," said Gwenn M. Hansen, Ph.D., chief scientific officer at Nurix Therapeutics. "In the context of controlling FcεRI-driven mast cell and basophil degranulation, the key drivers of CSU pathology, this translated in preclinical studies into a ~25-fold greater potency compared to remibrutinib while maintaining a highly BTK-selective profile based on global proteomic analyses of more than 8,700 proteins."

Bexobrutideg Demonstrates Differentiated Activity in Chronic Spontaneous Urticaria

CSU is an autoimmune condition driven by aberrant activation of mast cells and basophils through FcεRI signaling, leading to chronic itching and swelling. Both upstream autoantibody production and downstream effector signaling are dependent on BTK, making it a highly relevant therapeutic target.

Bexobrutideg is an orally bioavailable BTK degrader designed to eliminate the BTK protein through recruitment of the cereblon E3 ligase complex, which efficiently marks the BTK protein for proteasomal degradation, thereby eliminating both the kinase-dependent and scaffolding functions of BTK.

Key Findings

- Bexobrutideg potently and selectively degrades BTK across primary human B cells, basophils, and mast cells in preclinical assays
- Bexobrutideg demonstrates deeper suppression of BCR signaling than BTK inhibitors in preclinical assays by eliminating both enzymatic and scaffolding functions of BTK
- Across a broad proteomic analysis of more than 8,700 proteins, bexobrutideg shows highly selective BTK degradation preclinically
- Bexobrutideg produced greater reductions in ear swelling and vascular permeability than remibrutinib in a preclinical mouse model of passive cutaneous anaphylaxis model
- In healthy volunteers, both single- and multiple-ascending-dose oral dosing of bexobrutideg led to rapid and robust BTK

degradation in blood and skin, a key tissue of disease involvement in CSU

These data support the potential for bexobrutideg to deliver deep, tissue-level suppression of pathogenic immune signaling in CSU. Taken together, the safety, pharmacokinetic, and pharmacodynamic profile observed to date suggests bexobrutideg may offer meaningful advantages over BTK inhibitors across CSU and other IgE-mediated and autoimmune diseases.

About Bexobrutideg (NX-5948) in Inflammation and Immunology

Bexobrutideg is an investigational, oral, brain-penetrant, highly selective small-molecule degrader of BTK being developed within Nurix's inflammation and immunology (I&I) portfolio. It is designed to remove BTK and thereby disrupt signaling pathways that drive abnormal B-cell activation and Fc receptor-mediated effector functions implicated in a range of autoimmune and allergic conditions. Bexobrutideg is currently being evaluated, with a new tablet formulation, in a first-in-human single-ascending-dose and multiple-ascending-dose (SAD/MAD) study in healthy volunteers ([NCT06717269](#)).

In oncology, bexobrutideg is currently being evaluated in the DAYBreak CLL-201 clinical trial ([NCT07221500](#)), a pivotal single-arm Phase 2 study of bexobrutideg in patients with relapsed or refractory CLL. Nurix also continues enrollment in the NX-5948-301 Phase 1a/1b clinical trial ([NCT05131022](#)) of bexobrutideg in patients with relapsed or refractory B cell malignancies.

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, SAR448272/NX-3911, in collaboration with Sanofi, a clinical stage degrader of IRAK4, GS6791, 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's dedicated team 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 Brisbane, 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, including statements regarding the therapeutic potential of bexobrutideg in CSU or other inflammation and immunology indications, the potential advantages of bexobrutideg over BTK inhibitors, the potential of BTK degraders to suppress pathogenic immune signaling or deliver a differentiated level of control over immune-mediated disease biology, the anticipated clinical development of bexobrutideg, including plans for future studies in inflammation and immunology indications, and the potential of the bexobrutideg tablet formulation, 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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