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): June 2, 2021

NURIX THERAPEUTICS, INC.
(Exact Name of Registrant as Specified in its Charter)

Delaware	**001-39398**	**27-0838048**
(State or Other Jurisdiction of Incorporation or Organization)	**(Commission File Number)**	**(IRS Employer Identification No.)**

1700 Owens Street, Suite 205
San Francisco, California **94158**
(Address of Principal Executive Offices) **(Zip Code)**

(415) 660-5320
(Registrant's Telephone Number, Including Area Code)

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:

☐ 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, $0.001 par value per share	**NRIX**	**Nasdaq Global 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.

On June 2, 2021, Nurix Therapeutics, Inc. (the "Company") issued a press release announcing a poster presentation at the EULAR 2021 Virtual Congress ("EULAR") to be held virtually June 2-5, 2021. At EULAR, the Company will be presenting a poster entitled "NX-5948, a Selective Degrader of BTK, Significantly Reduces Inflammation in a Model of Autoimmune Disease" (the "Poster").

Copies of the press release and Poster are furnished as Exhibit 99.1 and Exhibit 99.2, respectively, and are incorporated herein by reference.

The information furnished with this Item 7.01, including Exhibit 99.1 and Exhibit 99.2, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, regardless of any general incorporation language in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Exhibit Title or Description
99.1	Press Release dated June 2, 2021
99.2	Poster "NX-5948, a Selective Degrader of BTK, Significantly Reduces Inflammation in a Model of Autoimmune Disease"

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended the Registrant has duly caused this Report to be signed on its behalf by the undersigned hereunto duly authorized.

NURIX THERAPEUTICS, INC.

Date: June 2, 2021

By: /s/ Christine Ring
Christine Ring, Ph.D., J.D.
General Counsel

Nurix Therapeutics Announces Presentation of Preclinical Data from NX-5948 Program Demonstrating Significant Reduction of Inflammation in a Model of Autoimmune Disease

Orally available NX-5948 is a potent selective degrader of Bruton's Tyrosine Kinase (BTK) without IMiD activity

Data were presented at the European Alliance of Associations for Rheumatology (EULAR) 2021 Virtual Congress

SAN FRANCISCO, June 2, 2021 (GLOBAL NEWSWIRE) — Nurix Therapeutics, Inc. (Nasdaq: NRIX), a biopharmaceutical company developing targeted protein modulation drugs, today announced the presentation of preclinical data from its NX-5948 program, demonstrating that daily oral dosing of NX-5948 resulted in a robust resolution of symptoms and inflammation in an animal model of arthritis. The data, which support further investigation of NX-5948 for clinical development as a potential treatment for autoimmune disorders, were presented at the EULAR 2021 Virtual Congress which is being held June 2-5, 2021.

"NX-5948 demonstrates highly selective, complete and durable BTK degradation activity resulting in profound reduction in inflammation in models of severe arthritis," said Robert J. Brown, M.D., Nurix's senior vice president of clinical development. "We remain on track to initiate clinical trials of NX-5948 in B cell malignancies in the second half of this year and, based on our recent findings, will consider the potential for future expansion of indications into selected autoimmune diseases in 2022."

"The data presented at this year's EULAR congress highlight the power of Nurix's DELigase platform to generate differentiated products that have the potential to address challenging diseases such as autoimmune disorders," said Arthur T. Sands, M.D., Ph.D., Nurix's chief executive officer. "The unique activities of NX-5948 include its ability to cross the blood-brain barrier in animal models. We plan to further explore this property in models of multiple sclerosis and other central nervous system diseases."

Aberrant activation of B cells and autoantibody-mediated tissue damage are hallmarks of autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus. In B cells and myeloid cells, BTK transduces signals downstream of the B cell receptor (BCR), toll-like receptors, and Fc receptors and its overexpression in B cells leads to hyperactive BCR signaling, plasma cell generation, and autoantibody secretion. This makes degradation of BTK a potentially powerful therapeutic strategy to address autoimmune disease.

The data presented at the EULAR Congress demonstrate that NX-5948 is a highly selective and potent degrader of BTK in primary human B cells (DC_{50}= 0.034nm) resulting in robust inhibition of anti-IgM- and TLR7-mediated B cell activation. Previous data have demonstrated that NX-5948 lacks IMiD activity with no Cereblon neo-substrate Aiolos degradation at clinically relevant concentrations (DC_{50}>10 micromolar). *In vivo* studies in both mice and non-human primates (NHPs) demonstrated that a single oral dose of NX-5948 resulted in rapid, dose-dependent, and durable BTK degradation in B cells with BTK levels remaining fully suppressed 24 hours post a single oral dose in NHPs. Importantly, data obtained from a mouse model of collagen-induced arthritis (CIA) demonstrated that in mice treated with NX-5948, symptoms of arthritis improved, with a significant reduction in arthritis clinical score, superior disease-related symptom control relative to ibrutinib, and similar activity to that of dexamethasone. Treatment with NX-5948 also resulted in a reduction in anti-type II collagen titer and serum levels of the pro-inflammatory cytokine, IL-6. Treatment with NX-5948 was well-tolerated and, unlike dexamethasone, did not lead to body weight loss.

A copy of the poster can be found on the Investor page of the Nurix website under Scientific Presentations.

About NX-5948 NX-5948 is an investigational, orally bioavailable, small molecule degrader of BTK that has been designed to lack IMiD activity for potential applications in indications where sparing IMiD activity may be beneficial. NX-5948 has demonstrated the ability to cross the blood brain barrier in animal models, suggesting potential utility in both autoimmune diseases and B-cell malignancies that involve the central nervous system. Nurix is investigating development of NX-5948 for the potential treatment of certain auto-immune diseases as well as certain B-cell malignancies.

About Nurix Therapeutics, Inc. Nurix Therapeutics is a biopharmaceutical company focused on the discovery, development, and commercialization of small molecule therapies designed to modulate cellular protein levels as a novel treatment approach for cancer and other challenging diseases. Leveraging Nurix's extensive expertise in E3 ligases together with its proprietary DNA-encoded libraries, Nurix has built DELigase, an integrated discovery platform to identify and advance novel drug candidates targeting E3 ligases, a broad class of enzymes that can modulate proteins within the cell. Nurix's drug discovery approach is to either harness or inhibit the natural function of E3 ligases within the ubiquitin proteasome system to selectively decrease or increase cellular protein levels. Nurix's wholly owned pipeline comprises targeted protein degraders of Bruton's tyrosine kinase, a B-cell signaling protein, and inhibitors of Casitas B-lineage lymphoma proto-oncogene B, an E3 ligase that regulates T cell activation. Nurix is headquartered in San Francisco, California. For more information, please visit http://www.nurix.com.

Forward Looking Statements

This press release contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995. These forward-looking statements reflect the current beliefs and expectations of management. All statements other than statements of historical fact are statements that could be deemed forward-looking statements, including, without limitation, statements concerning Nurix's future plans and prospects, the planned timing of Nurix's clinical trial programs for its drug candidates and the expansion of Nurix's DELigase™ platform. Although Nurix believes that the expectations reflected in such forward-looking statements are reasonable, it can give no assurance that such expectations will prove to be correct. Forward-looking statements are subject to risks and uncertainties that may cause Nurix's actual activities or results to differ significantly from those expressed in any forward-looking statement, including the risks and uncertainties described under the heading "Risk Factors" in documents Nurix files from time to time with the Securities and Exchange Commission (SEC) including Nurix's Annual Report on Form 10-K filed with the SEC on February 16, 2021, Nurix's Quarterly Report on Form 10-Q filed with the SEC on April 13, 2021, and other SEC filings. These forward-looking statements speak only as of the date of this press release, and Nurix undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof, except as required by applicable law.

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NX-5948, a Selective Degrader of BTK, Significantly Reduces Inflammation in a Model of Autoimmune Disease

Daniel W Robbins, Mark Noviski, May Tan, Cristiana Guiducci, Timothy Ingallinera, Dane E Karr, Aileen Kelly, Zef Konst, Austin Tenn-McClellan, Jenny McKinnell, Luz Perez, Gwenn Hansen and Ryan Rountree
Nurix Therapeutics, 1700 Owens Street, San Francisco, CA, USA 94158

Introduction

- Bruton's tyrosine kinase (BTK) transduces signals downstream of the B cell receptor (BCR), toll-like receptors, and Fc receptors in B cells and myeloid cells
- Overexpression of BTK in B cells can lead to hyperactive BCR signaling, plasma cell generation, and autoantibody secretion
- Aberrant activation of B cells and autoantibody mediated tissue damage are hallmarks of autoimmune diseases such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA).
- NX-5948 is a chimeric targeting molecule (CTM) that engages the E3 ligase cereblon (CRBN) to promote the selective degradation of BTK

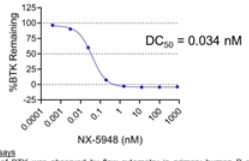
Results

- NX-5948 is a potent degrader of BTK in primary human B cells (DC_{50} = 0.34 nM) and inhibits BCR signaling
- NX-5948 is highly selective for BTK degradation by proteomic analysis with limited activity toward the CRBN neo substrate Aiolos (DC_{50} > 10 µM)
- In vivo, once daily oral administration of NX-5948 in mice and cynomolgus monkey demonstrated potent degradation of BTK in circulating B cells
- NX-5948 demonstrated significant anti-inflammatory activity and resulted in improvement of clinical symptoms in a mouse collagen-induced arthritis (CIA) model

Conclusions

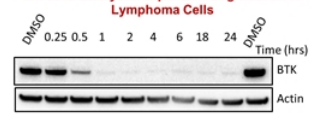
- NX-5948 mediates potent anti-inflammatory activity via BTK degradation with resultant inhibition of B cell activation
- Preclinical animal models support clinical development of NX-5948 to treat autoimmune diseases

NX-5948 is a Potent Degrader of BTK in Primary Human B Cells



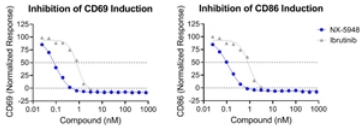
BTK Degradation Assays
Robust degradation of BTK was observed by flow cytometry in primary human B cells after 4 hour treatment with NX-5948.

NX-5948 Catalyzes Rapid BTK Degradation in Lymphoma Cells



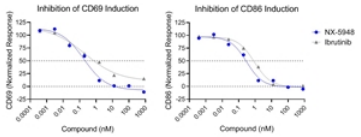
Degradation Time-Course Assay
Degradation of BTK is observed within 1 hour and is complete within 2 hours with 10 nM of NX-5948 in Ramos cells

NX-5948 is a Potent Inhibitor of Anti-IgM-Mediated B Cell Activation



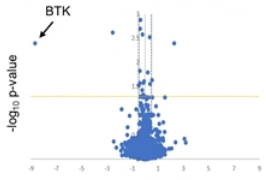
B Cell Activation Assay
NX-5948-mediated degradation of BTK prevents anti-IgM-induced upregulation of activation markers CD69 and CD86 on B cells. Human PBMCs were pre-incubated with NX-5948 or ibrutinib for 4 hours and then stimulated with 10 µg/ml anti-IgM for 18 hours. CD69 and CD86 levels on B cells were assessed by flow cytometry.

NX-5948 is a Potent Inhibitor of TLR7-Mediated B cell Activation



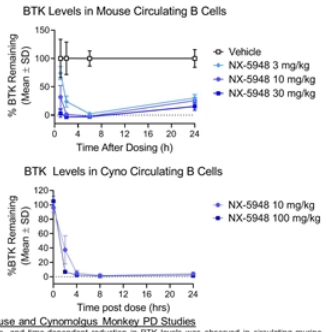
B Cell TLR7 Activation Assay
NX-5948-mediated degradation of BTK prevents Imiquimod-induced upregulation of activation makers CD69 and CD86 on B cells. Human PBMCs were pre-incubated with NX-5948 or ibrutinib for 4 hours and then stimulated with 5 µg/ml Imiquimod for 20 hours. CD69 and CD86 levels on B cells were assessed by flow cytometry.

NX-5948 is a Selective BTK Degrader



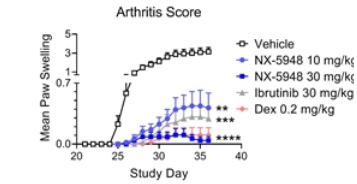
Proteomics Study
NX-5948 selectively degrades BTK in TMD8 cells. Cells were treated with DMSO or NX-5948 (50 nM) for 6 hours in triplicate. Effects on protein levels were analyzed using label-free proteomics.

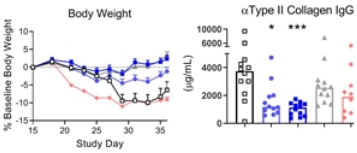
Oral Dosing of NX-5948 Promotes Rapid and Complete BTK Degradation in Mouse and Non-Human Primate B Cells

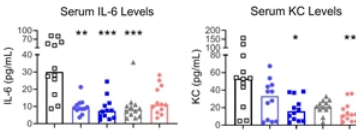


Mouse and Cynomolgus Monkey PD Studies
Dose- and time-dependent reduction in BTK levels was observed in circulating murine and non-human primate, cynomolgus monkey B cells following a single oral dose of NX-5948. BTK levels were fully suppressed in 1 to 6 hours. In mice, BTK levels increased 24 hours after dosing from BTK resynthesis. In cynomolgus monkeys, BTK levels remained suppressed at 24 hours. BTK levels were measured in circulating B cells by flow cytometry.

NX-5948 is Efficacious and Well-Tolerated in a Mouse Collagen-Induced Arthritis Model and Suppresses Antibody Titers and Cytokine Levels







Mouse Collagen-Induced Arthritis (CIA) Model
Daily oral treatment with NX-5948 at 30 mg/kg resulted in a lower mean arthritis score t
ibrutinib and provided similar clinical benefit as dexamethasone with minimal body weight los
compared to dexamethasone and vehicle. CIA was induced by immunization on Day 0
boosted on Day 21 with type II collagen in complete Freund's adjuvant; treatment with vehicl
therapeutic agents was initiated on Day 18. At the end of the study on Day 36, serum
collected and serum levels of anti-type II collagen IgG, the inflammatory cytokine IL-6, or
neutrophil chemokine KC/CXCL1 were determined. Statistical significance was determi
between vehicle control and treated groups with one-way Kruskal-Wallis ANOVA and Dun
multiple comparisons test.

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