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): December 7, 2020

NURIX THERAPEUTICS, INC.

(Exact Name of Registrant as Specified in its Charter)

Delaware (State or Other Jurisdiction of Incorporation or Organization)

001-39398 (Commission File Number) 27-0838048 (IRS Employer Identification No.)

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

94158 (Zip Code)

(415) 660-5320

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)
- $\label{eq:pre-communications} \square \quad \text{Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))}$
- ☐ 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:

	Trading	Name of each exchange
Title of each class	symbol(s)	on which registered
Common Stock, \$0.001 par value per share	NRIX	Nasdag 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. \Box

Item 7.01 Regulation FD Disclosure.

On December 7, 2020, Nurix Therapeutics, Inc. (the "Company") presented at the 62nd American Society of Hematology Annual Meeting and Exposition (the "Presentation"). A copy of the Presentation is attached hereto as Exhibit 99.1, and is incorporated herein by reference.

The information furnished with this Item 7.01, including Exhibit 99.1, 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

Forward Looking Statements

This Current Report on Form 8-K, including Exhibit 99.1, contains forward-looking statements and information relating to Nurix
Therapeutics, Inc. (the "Company," "we," "us" or "our"). Forward-looking statements are neither historical facts nor assurances of future performance.
Instead, they are based on our current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, our
development plans, our preclinical results and other future conditions. All statements, other than statements of historical facts, contained in this
presentation are forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including statements regarding
our future financial or business performance, conditions, plans, prospects, trends or strategies and other financial and business matters; our current and
prospective product candidates; the potential advantages of NX-2127; the extent animal model data predicts human efficacy; and the success and timing
of our development and commercialization of our product candidates. In addition, when or if used in this presentation, the words "may," "could,"
"should," "anticipate," "believe," "estimate," "expect," "intend," "plan," "predict" and similar expressions and their variants, as they relate to the
Company may identify forward-looking statements. Although we believe the expectations reflected in such forward-looking statements are reasonable,
we can give no assurance that such expectations will prove to be correct. Readers are cautioned that actual results, levels of activity, performance or
events and circumstances could differ materially from those expressed or implied in our forward-looking statements due to a variety of factors, including
risks and uncertainties related to our ability to advance our product candidates; obtain regulatory approval of and ultimately commercialize our product
candidates; the timing and results of preclinical and clinical trials; our ability to fund development activities

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit

Exhibit Title or Description

99.1 <u>Presentation dated December 7, 2020</u>

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: December 7, 2020

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

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NX-2127, a Degrader of BTK and IMiD Neosubstrates, for the Treatment of B-Cell Malignancies

Presenter: Daniel Robbins, Ph. D.

drobbins@nurixtx.com

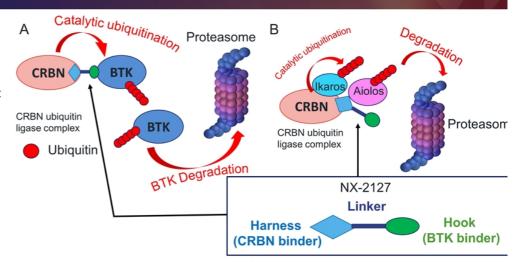
Disclosures

All authors of this presentation are current or former employees and shareholders of Nurix Therapeutics.



NX-2127 has a dual degradation mechanism of action for two clinically validated targets

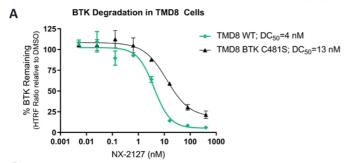
- BTK is a tyrosine kinase involved in B cell development, differentiation and signaling
- BTK inhibitors are approved for treatment of B cell malignancies
- Mutations to BTK have conferred resistance to approved agents indicating an area of unmet medical need
- IMiD therapies have shown efficacy in some aggressive B-cell malignancies
- The dual action of BTK degradation and IMiD activity may provide a unique treatment strategy for relapsed/refractory B-cell malignancies
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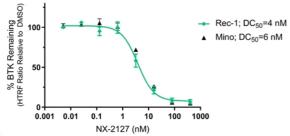
- (A) NX-2127 is a novel, hetero-bifunctional, orally administered, Chimeric Targeting Molecule (CTM) that induces the degradation of Bruton's Tyrosine Kinase (BTK) in cells through recruitment of cereblon (CRBN), a component of the CRL4-CRBN ubiquitin ligase complex
- (B) The engagement of NX-2127 also catalyzes neosubstrate degradation of Aiolos (IKZF3) and Ikaros (IKZF1), two transcription factors regulating T-cell function



NX-2127 catalyzes BTK degradation and antiproliferative activity in cancer cell lines

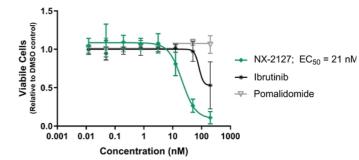


BTK Degradation in Mantle Cell Lymphoma Cells



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C Anti-proliferative activity in C481S TMD8 cells

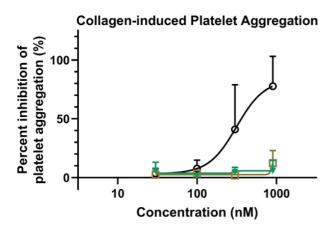


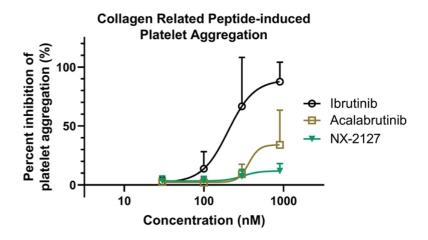
- (A) NX-2127 catalyzes potent degradation of BTK in DLBCL cell lines (TMD8), including cells with the ibrutinibresistance mutation BTK^{C481S}
- (B) NX-2127 catalyzes BTK degradation in Rec-1 and Mino mantle cell lymphoma cell lines
- (C) NX-2127 potently blocks cell proliferation of BTK^{C481S} TMD8 cells relative to ibrutinib

DC₅₀ = half maximal degradation concentration HTRF = Homogeneous Time Resolved Fluorescence



NX-2127 has minimal in vitro effects on platelet aggregation





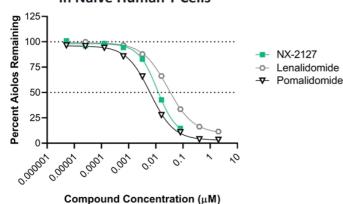
NX-2127 does not show significant inhibition of platelet aggregation in an in vitro platelet aggregation assay

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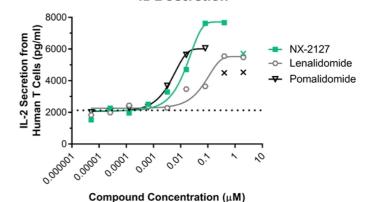
NX-2127 catalyzes Aiolos degradation and IL-2 production similar to IMiD drugs

IMiD Activity: Aiolos Degradation in Naïve Human T Cells



 NX-2127 degrades Aiolos with similar potency to that of pomalidomide and lenalidomide

IMiD Activity: T Cell Activation and IL-2 Secretion



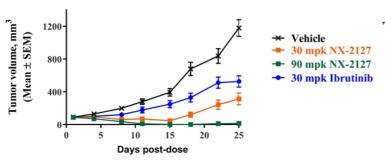
 NX-2127 exhibits IMiD-like activity by activation and IL-2 production following CD3/CD28 stimulation



Oral Administration of NX-2127 Demonstrates Cancer Growth Inhibition in Mouse Xenograft Tumor Model

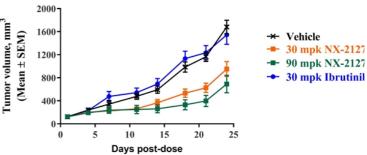
Tumor Growth Inhibition in Xenograft Model of Wild Type Lymphoma

TMD8 Tumor Growth



 NX-2127 demonstrates comparable tumor growth inhibition to ibrutinib in a xenograft mouse model containing tumors with a wild type BTK Tumor Growth Inhibition in Xenograft Model of Mutant Ibrutinib-Resistant Lymphoma

TMD8 BTK^{C481S} Tumor Growth

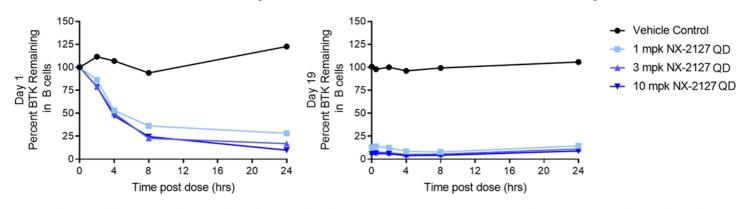


 NX-2127 shows more potent tumor growth inhibition compared to ibrutinib in a xenograft mouse model containing tumors with the most common human resistance mutation (C481S) in BTK target protein

Oral Dosing of NX-2127 Degrades BTK in Cynomolgus Monkey

BTK Levels in B cells at Day 1

BTK Levels in B cells at Day 19



- NX-2127 induces significant degradation of BTK in 4 hours and more than 90% degradation through 24 hours post dose
- Once daily, oral dosing of NX-2127 maintains suppression of BTK protein levels throughout the 19-day duration of the study (NX-2127 PK $\rm t_{1/2}$ = 5.4 h)
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Summary and Conclusions

- NX-2127 catalyzes potent BTK degradation *in vitro* and results in anti-proliferative effects in human lymphoma cell lines
- The IMiD activity of NX-2127 is similar to that of IMiD drugs pomalidomide and lenalidomide and results in T-cell activation
- NX-2127 demonstrates potent BTK degradation in vivo upon oral dosing in cynomolgus monkey and displays anti-tumor effects in both wild-type and C481S mutant mouse xenograft tumor models
- NX-2127 combines BTK degradation with IMiD activity to provide an attractive therapeutic strategy in the setting of resistance mutations and an expanded set of B-cell malignancies





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