# UNITED STATES
# SECURITIES AND EXCHANGE COMMISSION
**Washington, D.C. 20549**

# FORM 8-K

**CURRENT REPORT**
**Pursuant to Section 13 or Section 15(d)**
**of the Securities Exchange Act of 1934**

**Date of Report (Date of earliest event reported): December 14, 2023 (December 12, 2023)**

# Inhibitor Therapeutics, Inc.
**(Exact name of registrant as specified in its charter)**

| **Delaware** | **001-13467** | **30-0793665** |
|---|---|---|
| **(State or other jurisdiction of incorporation)** | **(Commission File Number)** | **(IRS Employer Identification No.)** |

**4905 South West Shore Blvd**
**Tampa, FL 33611**
**(813) 864-2562**
**(Address, including Zip Code and Telephone Number, including Area Code, of Principal Executive Offices)**

**Not Applicable**
**(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 to 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))

| **Title of each class** | **Trading Symbol(s)** | **Name of each exchange on which registered** |
|---|---|---|
| None | N/A | N/A |

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 1.01. Entry into a Material Definitive Agreement.**

On December 12, 2023, Inhibitor Therapeutics, Inc. (the "Company") entered into an Exclusive License Agreement (the "Agreement") with Johns Hopkins University ("JHU"). Pursuant to the Agreement, JHU granted to the Company the exclusive worldwide patent rights to a Granted US Patent, No 8,980,930 entitled "New Angiogenesis Inhibitors" (the "Patent"). The Patent relates to the treatment of prostate cancer, basal cell carcinoma including basal cell carcinoma nevus syndrome, and lung cancer.

Pursuant to the Agreement: (i) the Company has received an exclusive worldwide license to the Patent; (ii) the Company shall pay to JHU an upfront license fee (iii) the Company shall be required to make certain Minimum Annual Royalty ("MAR") payments to JHU no later than January 1st of each calendar year in accordance with an agreed upon schedule), (iv) the Company shall be required to pay to JHU a royalty on cumulative net sales, with an additional supplement due where a licensed product is given exclusivity in the U.S. by patent rights, (v) if the Company enters into any sublicense they will pay to JHU a certain percentage of all consideration received from sublicensee but excluding (i) any consideration received by Licensee for Royalties on Sublicensee Sales (Royalties on Sales by Sublicensees will be treated as if Licensee made the Sale), and (ii) any payment of Past Patent Costs or Patent Costs made by Sublicensee to License), (vi) should the Company receive compensation in the form of a voucher, the Company will pay a certain percentage of the sale to JHU and (vii) the Company shall be required to pay to JHU certain development-related milestone payments upon the Company meeting each of a series of agreed upon milestones. The Agreement contains other customary terms and conditions.

JHU has the right to terminate the Agreement upon the occurrence of certain events, including delinquency in payments, failure to timely reach milestones, noncompliance with audit or insurance obligations, or the Company entering into voluntary bankruptcy or insolvency. The Company may terminate the Agreement without cause upon 90 days advance written notice.

The preceding is a summary of the Agreement, qualified in its entirety by reference to the text of the Agreement filed as an Exhibit to the Company's Annual Report on Form 10-K for the Fiscal Year end and incorporated by reference herein.

**Item 8.01 Other Events.**

On December 13, 2023, the Company issued a press release announcing the execution of the License Agreement. A copy of the press release is attached as Exhibit 99.1 hereto.

**Item 9.01 Financial Statements and Exhibits.**

(d) The following exhibit is filed with this report.

| Exhibit No. | Description of Exhibit |
|---|---|
| 99.1 | Press Release dated December 13, 2023, regarding the Exclusive License Agreement by and between Inhibitor Therapeutics, Inc., and Johns Hopkins University |
| 104 | Cover Page Interactive Data File (embedded within the Inline XBRL document). |

**SIGNATURES**

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

Dated: December 14, 2023

INHIBITOR THERAPEUTICS, INC.

By: */s/ Francis E. O'Donnell*
Name: Francis E. O'Donnell
Title: Executive Chairman and CEO

INHIBITOR THERAPEUTICS

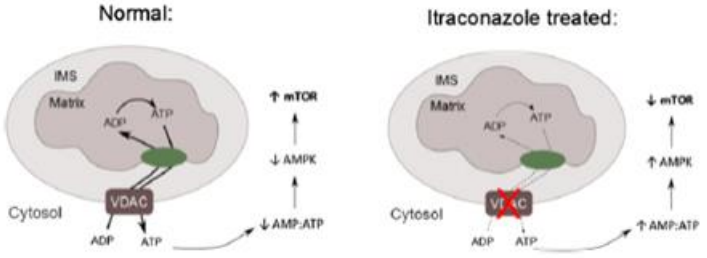
**Inhibitor Therapeutics, Inc. Exclusive License with Johns Hopkins University**

*A critical milestone completed on the mission path of commercializing Itraconazole in numerous oncology indications.*

Inhibitor Therapeutics, Inc. ("Inhibitor") (OTCQB:INTI) has entered into an exclusive, worldwide licensing agreement (the "License") with Johns Hopkins University (JHU) for their U.S Patent 8,980,930 (Canada Patent 2,572,223) "New Angiogenesis Inhibitors". Angiogenesis Inhibitors, as described by the National Cancer Institute, are unique cancer fighting agents as they block the growth of blood vessels that support tumor growth rather than blocking the growth of the tumor cells themselves. Inventors affiliated with JHU developed this patent, listing Itraconazole as an Active Pharmaceutical Ingredient (API) that has anti-angiogenic properties.

The License notes field of use is for use in Prostate Cancer, Basal Cell Carcinoma including Basal Cell Carcinoma Nevus Syndrome (an orphan oncology disease), and Lung Cancer, all of which emphasize a significant unmet need. Inhibitor believes the License is a mutually beneficial agreement, yielding a modest annual royalty rate with milestone payments typical to such a license.

Within the literature *Head et al*[1] explains the long-term recognition that angiogenesis is a requirement for tumor growth and metastasis and that growing tumors can promote angiogenesis by secreting proangiogenic factors such as VEGF, basic FGF, EGF and others. These proangiogenic factors stimulate the proliferation, migration, and differentiation of the endothelial cells that make up the inner layer of all blood vessels, causing them to form new vessels that grow towards the source of these factors, this process termed "tumor angiogenesis". It is identified that the major target of itraconazole in endothelial cells is VDAC1. VDAC1 knockdown profoundly inhibits mTOR activity and cell proliferation in HUVEC revealing a previously unknown connection. Inhibition of VDAC1 by itraconazole leads to an increase in cellular AMP:ATP ratio and activation of the AMP-activated protein kinase (AMPK), an upstream regulator of mTOR. VDAC1-knockout cells are resistant to AMPK activation and mTOR inhibition by itraconazole, demonstrating that VDAC1 is the mediator of this activity. In their testing it was found that by using a phenotypical approach starting with the effect of itraconazole the G1-S cell cycle transition of the endothelial cells, itraconazole specifically inhibited the mTOR signaling pathway by downregulating the kinase activity of mTORC1.




*Chow Et Al*[2] completed a study utilizing RNA sequencing to decipher the biological pathway propelling BCC growth. From the assay results, it was observed that BCCs exhibited a considerable amplification in the expression of the mTOR pathway. This pathway plays an essential role in regulating angiogenesis, the growth of new blood vessels from pre-existing ones. Thus, this indicates that it is the heightened activity of the mTOR pathway, and the consequent enhancement of angiogenesis, that stimulates the growth.

Data from Inhibitor's completed Phase 2b SCORING Trial complements the literature with reference to the Lesion Response of the study which shows that across the 477 target lesions the investigators reported reductions of any size from baseline for 399 (83.65%) lesions, 64 (13.42%) had no change and 14 increased in size. A pre-determined reduction of 30% or greater was seen in 275 of the 477 (57.7%) target lesions, including 130 lesions which resolved completely (27.3%). A total of 13 new 'surgically eligible' lesions across 8 of the 38 patients developed over the duration of the study.

For more information about Inhibitor and our mission please visit us on our website (www.inhibitortx.com) and for any further or specific queries you may have please visit our contact us page, submit your details/query, and a representative will be happy to get in touch.

**Forward-Looking Statements**

This press release contains projections and other forward-looking statements regarding future events and our future financial performance. All statements other than present and historical facts and conditions contained in this release, including any statements regarding future results of operations and financial positions, business strategy and plans, expectations for future product sales, our ability to convert our pipeline to revenue and our objectives for future operations, are forward-looking statements (within the meaning of the Private Securities Litigation Reform Act of 1995, Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended). These statements are only predictions and reflect our current beliefs and expectations with respect to future events and are based on assumptions and subject to risk and uncertainties and subject to change at any time. We undertake no obligation to update the information made in this release in the event facts or circumstances subsequently change after the date of this press release. We operate in a very competitive and rapidly changing environment. New risks emerge from time to time. Given these risks and uncertainties, you should not rely on or place undue reliance on these forward-looking statements. Actual events or results may differ materially from those contained in the projections or forward-looking statements.

References


1. Head SA, Shi W, Zhao L, Gorshkov K, Pasunooti K, Chen Y, Deng Z, Li RJ, Shim JS, Tan W, Hartung T, Zhang J, Zhao Y, Colombini M, Liu JO. Antifungal drug itraconazole targets VDAC1 to modulate the AMPK/mTOR signaling axis in endothelial cells. Proc Natl Acad Sci U S A. 2015 Dec 29;112(52):E7276-85. doi: 10.1073/pnas.1512867112. Epub 2015 Dec 10. PMID: 26655341; PMCID: PMC4703001.

2. Chow RY, Levee TM, Kaur G, Cedeno DP, Doan LT, Atwood SX. MTOR promotes basal cell carcinoma growth through atypical PKC. Exp Dermatol. 2021 Mar;30(3):358-366. doi: 10.1111/exd.14255. Epub 2020 Dec 20. PMID: 33617094; PMCID: PMC9924159.