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Inhibitor Therapeutics, Inc. Exclusive License with Johns Hopkins University

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

TAMPA, Fla., Dec. 13, 2023 /PRNewswire/ -- 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.

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