TISSUE-SPECIFIC RNA DRUG DELIVERY PLATFORM TECHNOLOGY



BOUND Therapeutics

BOUND Tx Generative AI/ML Approaches for Protein-Ligand Interaction Design

Target Selection

Binding Site Selection

Al Molecular Pattern Discovery

Ligand Selection

Choose a Natural 3D Conformation for a **Protein Target Candidate**

Automated 3D Protein **Structure Analysis for Identification of Putative Binding Pockets**

Al-Driven Ligand Library **Generation and In-Silico Experiments** for Optimal Molecular Structure

Al Employs **Reinforcement** Learning to Generate **Optimal Ligand Structures**, Guided by Desired Structural

Identification

and Chemical Properties

BOUND Tx RNA-Peptide Drug Design

Mechanism of Action (MOA)

Ligand Receptor

Outside Cancer Cell

Cancer cells exhibit upregulated expression of receptors that selectively bind to abundant circulating growth factors, facilitating enhanced proliferation and growth of malignant cells

BOUND Tx Cancer-Blocking RNA Drug with Receptor-Targeting Peptide Ligand

Design the RNA drug to target any cancer-driver RNA

BOUND Tx platform utilizes a receptortargeting peptide ligand to deliver a drug cargo, comprising a conjugated RNA analog, that once it's internalized inside the cell, will block a cancerdriving RNA (oncogene) in the cytoplasm.

Inside Cancer Cell



driver RNA (oncogene) inside the cancer cell.

Innovative RNA Sequence Design, **Substitution and Back Bone** Optimization

- **Protease Resistance: ensuring** stability pre/post-cellular uptake
- Enhanced Specificity and Efficacy
- Low Immunotoxicity
- High Therapeutics Index (TI)

Non-Canonical Cyclic Peptides

Ligand

- **Protease Resistance and High Binding Affinity to Specific**
- Targets: ensuring stability during
- circulation and ligand binding
- **Compact Size (< 2 KD): facilitates** tissue penetration and cellular internalization

Low Immunotoxicity

(oncogene).

Pending and Issued Patents

"Compositions and Methods for the Treatment of Cancer" PCT/US2024/019719, 2024

"Compositions and Methods for MYC Messenger **RNA Inhibitors**", **US 11,306,312**, **issued 2022** • Expires in Feb. 2038, could be extended as far as Aug. 2043

Technology Feature	BOUND Tx	Competitive Technology
Tissue Targeted Delivery	Targeted Extrahepatic Delivery to Solid Tumors via Specific Cellular Membrane Proteins	Limited Systemic Deliverable Targets: Hepatic, Circulation and Local
Formulation	Soluble in saline, isotonic. No issues with dosing volume, range and route or injection site reactions (ISR)	Costly formulation, limited dosing volume, range and route due to ISR
Tissue Penetration	Small size, good in-vivo biodistribution and cellular PK profile	Large molecular weight, poor cellular PK
Delivery Ligand	Proprietary Al design, cargo tailored structure chemistry, rapid turnaround	Costly high-throughput compound screening
herapeutic Index (TI)	WIDE	NARROW

Tumor Specific Delivery of BOUND Tx BND6482 miR-21 Inhibitor in Triple Negative Breast Cancer (TNBC)

AntimiR-21 TNBC Cell Inhibition

Time-course Fluorescent-Labeled BND6482

In-vivo POC Efficacy: BND6482 Inhibited

Delivery in TNBC Tumors

TNBC Tumor Growth



BOUND Tx BND5412 miR-21 inhibitor led to an over 8fold reduction in cell proliferation across seven human triple-negative breast cancer cell models during a 4-day period

Contact



Changes in BOUND TX AlexaFluor647-BND6482 miR-21 Inhibitor Accumulation in EMT6 Triple-Negative Breast Cancer Allografts in Female Balb/c Mice Following a Single 5 mg/kg Intraperitoneal Injection

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Administration of BOUND Tx BND6482 at 5 mg/kg-IP **BIW**, the progression of tumor volumes in **EMT6** orthotopic TNBC allografts localized within mammary adipose tissue was halted. In contrast, the controls: vehicle, mismatched RNA, anti-Trop-2irinotecan conjugate Trodelvy (SOC) demonstrated persistent tumor growth. Sample size N=5. Error bars represent the standard error of the mean (SEM)

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