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): April 21, 2026

TONIX PHARMACEUTICALS HOLDING CORP.

(Exact name of registrant as specified in its charter)

Nevada	**001-36019**	**26-1434750**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

200 Connell Drive, Suite 3100, Berkeley Heights, New Jersey 07922
(Address of principal executive offices) (Zip Code)

Registrant's telephone number, including area code: (862) 799-8599

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 (see General Instruction A.2. below):

☐ 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	TNXP	The NASDAQ Global Select 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 April 23, 2026, Tonix Pharmaceuticals Holding Corp. (the "Company") announced an oral presentation and two poster presentations at the American Association for Cancer Research ("AACR") Annual Meeting 2026, held April 17-22, 2026. A copy of the press release that discusses this matter is attached hereto as Exhibit 99.01. Copies of the posters are attached hereto as Exhibits 99.02 and 99.03.

The information in this Item 7.01 of this Current Report on Form 8-K, including Exhibits 99.01, 99.02 and 99.03 attached hereto, shall not be deemed "filed" for purposes of Section 18 of the United States Securities Exchange Act of 1934 (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall they be deemed incorporated by reference in any filing under the United States Securities Act of 1933 or the Exchange Act, except as shall be expressly set forth by specific reference in such a filing.

Item 8.01 Other Events.

On April 21, 2026, the Company's collaborators at Columbia University presented an oral presentation on the Company's preclinical TNX-1700 program at the AACR entitled *TFF2 Deficiency Amplifies IL-1β–Driven Inflammation and Promotes Aging-Associated Gastric Tumor Progression*, which reviewed the results of a preclinical study comparing TFF2-expressing epithelial cells in the stomachs of aged and young mice, which were observed to be reduced in aged mice, with corresponding reductions in tissue and circulating TFF2 levels. Decline of TFF2 led to elevated IL-1β and promoted gastric inflammaging. Findings demonstrate that the murine version of TNX-1700 (mTNX-1700), a fusion protein of murine TFF2-murine albumin, reversed aging-associated inflammation in the mouse model. The stomachs of the aged mice exhibited increased susceptibility to tumor progression. Myeloid-derived suppressor cells accumulated and overexpressed IL-1β, interacting with IL-1R1$^+$ cancer-associated fibroblasts. mTNX-1700 attenuated tumor progression in the aged gastric microenvironment.

The Company presented the poster entitled *In Vitro Characterization of Fully Human Antagonistic Anti-BTLA Monoclonal Antibodies*, which reviews B and T Lymphocyte Attenuator ("BTLA") role as a potential target in immuno-oncology since its ligand herpesvirus entry mediator is expressed in and upregulated in the tumor microenvironment of many cancers and generally correlates with reduced overall survival. The Company believes that targeting BTLA offers opportunities for cancer immunotherapy and has the potential to demonstrate additive or synergistic activity when combined with other checkpoint antagonists, potentially overcoming resistance mechanisms and improving clinical outcomes. The Company studied four potent, high affinity, human/cyno cross-reactive, human antagonistic anti-BTLA monoclonal antibodies. Three antagonists had reduced FcgRI binding and no binding to FcgRIIB compared to an antibody control, which the Company believes has the potential to improve pharmacokinetics and confer a reduced risk of FcR-dependent adverse events, such as cytokine release syndrome or other immune-mediated toxicities.

The second poster presentation, entitled *Pharmacokinetics of TNX-1700 in Non-Human Primates and Human FcRn/Serum Albumin Transgenic Mice*, which evaluates the Company's TNX-1700 product candidate for the treatment of gastric and colorectal cancer in combination with PD-1 blockade in non-human primates and double-transgenic mice expressing human FcRn and human serum albumin. All animals survived without clinical signs or greater than 10% body-weight loss. TNX-1700 exhibited dose-independent, linear pharmacokinetics, with comparable pharmacokinetic profiles and exposure observed across species and doses. TNX-1700 was found to extend the half-life of TFF2 and achieved durable systemic exposure. The Company believes this supports its potential as a therapeutic candidate for gastric cancer. *In Vitro Characterization of Fully Human Antagonistic Anti-BTLA Monoclonal Antibodies*, reviews B and T Lymphocyte Attenuator ("BTLA") role as a potential target in immuno-oncology since its ligand herpesvirus entry mediator is expressed in and upregulated in the tumor microenvironment of many cancers and generally correlates with reduced overall survival. The Company believes that targeting BTLA offers opportunities for cancer immunotherapy and has the potential to demonstrate additive or synergistic activity when combined with other checkpoint antagonists, potentially overcoming resistance mechanisms and improving clinical outcomes. The Company studied TNX-4700, its product candidate for the treatment of cancer, four potent, high affinity, human/cyno cross-reactive, human antagonistic anti-BTLA monoclonal antibodies. Three antagonists had reduced FcgRI binding and no binding to FcgRIIB compared to an antibody control, which the Company believes has the potential to improve pharmacokinetics and confer a reduced risk of FcR-dependent adverse events, such as cytokine release syndrome or other immune-mediated toxicities.

Forward- Looking Statements

This Current Report on Form 8-K contains certain forward-looking statements within the meaning of Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934 and Private Securities Litigation Reform Act, as amended, including those relating to the Company's product development, clinical trials, clinical and regulatory timelines, market opportunity, competitive position, possible or assumed future results of operations, business strategies, potential growth opportunities and other statement that are predictive in nature. These forward-looking statements are based on current expectations, estimates, forecasts and projections about the industry and markets in which we operate and management's current beliefs and assumptions.

These statements may be identified by the use of forward-looking expressions, including, but not limited to, "expect," "anticipate," "intend," "plan," "believe," "estimate," "potential," "predict," "project," "should," "would" and similar expressions and the negatives of those terms. These statements relate to future events or our financial performance and involve known and unknown risks, uncertainties, and other factors which may cause actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Such factors include those set forth in the Company's filings with the SEC. Prospective investors are cautioned not to place undue reliance on such forward-looking statements, which speak only as of the date of this press release. The Company undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise.

Item 9.01 Financial Statements and Exhibits.

(d)

Exhibit No.	Description.
99.01	Press Release of the Company, dated April 23, 2026
99.02	In Vitro Characterization of Fully Human Antagonistic Anti-BTLA Monoclonal Antibodies
99.03	Pharmacokinetics of TNX-1700 in Non-Human Primates and Human FcRn/Serum Albumin Transgenic Mice
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURE

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

TONIX PHARMACEUTICALS HOLDING CORP.

Date: April 23, 2026

By: /s/ Bradley Saenger
Bradley Saenger
Chief Financial Officer

Exhibit 99.1

Tonix Pharmaceuticals Presents Updates on Preclinical Immuno-Oncology Programs at the American Association for Cancer Research (AACR) Annual Meeting 2026

TNX-1700 (TFF2-albumin fusion protein) reversed aging-associated gastric inflammation and significantly attenuated tumor progression in aged gastric microenvironment in preclinical models

TNX-1700 exhibited dose-independent, linear pharmacokinetics in animals

TNX-4700 (human anti-BTLA monoclonal antibody) demonstrated potent, high-affinity binding and functional antagonism

BERKELEY HEIGHTS, N.J., April 23, 2026 (GLOBE NEWSWIRE) -- Tonix Pharmaceuticals Holding Corp. (Nasdaq: TNXP) ("Tonix" or the "Company"), a fully integrated, commercial biotechnology company, today announced an oral presentation and two poster presentations on its preclinical immuno-oncology portfolio at the American Association for Cancer Research (AACR) Annual Meeting 2026, held April 17-22, 2026, in San Diego, California.

"We are pleased to report encouraging preclinical data on our TFF2-albumin fusion protein (TNX-1700) and our anti-BTLA monoclonal antibody (mAb) (TNX-4700) at AACR," said Bruce Daugherty, PhD, MBA, Executive Vice President of Research at Tonix Pharmaceuticals. "TNX-1700 and TNX-4700 are investigational immuno-oncology candidates in pre-clinical development. TNX-1700 is in development for the treatment of gastric and colorectal cancer in combination with PD-1 inhibitors. TNX-4700 is in development for the treatment of potentially several cancers since its ligand HVEM is expressed and/or upregulated in the tumor microenvironment and generally correlates with reduced overall survival."

Abstract #: 6822 Oral Presentation: "TFF2 Deficiency Amplifies IL-1β–Driven Inflammation and Promotes Aging-Associated Gastric Tumor Progression"

- Presenting author: Shuang Li, MD, PhD, Postdoctoral Research Scientist in the Timothy C. Wang, MD, Laboratory at the Herbert Irving Comprehensive Cancer Center, Columbia University Medical Center

Aging is a major risk factor for gastric cancer, but the underlying mechanisms remain poorly defined. The stomach undergoes profound epithelial and immune remodeling during aging. TFF2 is a mucosal protective factor implicated in epithelial repair and immune regulation. However, whether TFF2 regulates age-associated inflammation and tumor progression remains unknown.

TFF2-expressing epithelial cells were reduced in the stomachs of aged mice compared to young mice, with corresponding reductions in tissue and circulating TFF2 levels. Decline of TFF2 led to elevated IL-1β and promoted gastric inflammaging. The murine version of TNX-1700 (mTNX-1700 or TFF2-MSA) treatment reversed aging-associated inflammation. The aged stomach exhibited increased susceptibility to tumor progression. Myeloid-derived stem cells (MDSCs) accumulated and overexpressed IL-1β, interacting with IL-1R1^{+} cancer associated fibroblasts (CAFs). mTNX-1700 attenuated tumor progression in the aged gastric microenvironment.

Poster Presentation #7940: "Pharmacokinetics of TNX-1700 in Non-Human Primates and Human FcRn/Serum Albumin Transgenic Mice"

- Presenting author: Bruce Daugherty, PhD, MBA, Executive Vice President of Research, Tonix

TNX-1700 was evaluated in double-transgenic mice expressing human FcRn and human serum albumin (HSA) and in non-human primates. All animals survived without clinical signs or greater than 10% body-weight loss. TNX-1700 exhibited dose-independent, linear pharmacokinetics, with comparable pharmacokinetic profiles and exposure observed across species and doses. TNX-1700 substantially extends the half-life of TFF2 and achieves durable systemic exposure, supporting its potential as a therapeutic candidate for gastric cancer.

Poster Presentation #6550: *In Vitro* Characterization of Fully Human Antagonistic Anti-BTLA Monoclonal Antibodies

- Presenting author: Bruce Daugherty, PhD, MBA, Executive Vice President of Research, Tonix

B and T Lymphocyte Attenuator (BTLA) is a promising target in immuno-oncology since its ligand HVEM (herpesvirus entry mediator) is expressed in and upregulated in the tumor microenvironment of many cancers and generally correlates with reduced overall survival. Targeting BTLA offers opportunities for cancer immunotherapy and may demonstrate additive or synergistic activity when combined with other checkpoint antagonists, potentially overcoming resistance mechanisms and improving clinical outcomes.

Tonix studied several anti-BTLA mAbs, which demonstrated potent, high-affinity binding and functional antagonism of BTLA *in vitro*. Antagonists with reduced FcgRI binding and no binding to FcgRIIB may improve pharmacokinetics and confer a reduced risk of FcR-dependent adverse events, such as cytokine release syndrome or other immune-mediated toxicities.

Copies of the two poster presentations are available under the Scientific Presentations tab on the Tonix website at www.tonixpharma.com.

About Trefoil Factor Family Member 2 (TFF2)
Human TFF2 is a secreted protein expressed in gastrointestinal mucosa where it functions to protect and repair the mucosal lining. In gastric cancer, TFF2 is epigenetically silenced, and TFF2 is suggested to be protective against cancer development through several mechanisms, including its activity as a partial agonist of CXCR4 that modulates myeloid cell trafficking to reduce accumulation of immunosuppressive neutrophils.

About TNX-1700
TNX-1700, a fusion protein of TFF2 and albumin, is in preclinical and pre-Investigational New Drug (IND) stage of development as a treatment of gastric and colorectal cancer in combination with PD-1 blockade.[1] The Company in-licensed TFF2 technology from Columbia University. TNX-1700 is an immunotherapy being developed to treat gastric and colorectal cancers in combination with PD-1 blockers. Results of preclinical testing demonstrated that a mouse version of TNX-1700 was able to evoke an increase in anti-tumor immunity in combination with anti-PD-1 in several mouse models of gastric cancer by reducing immunosuppressive neutrophils and activating anti-tumoral CD8+ T cell responses. TNX-1700 administered as both monotherapy and in combination with anti-PD-1 dramatically reduced metastasis and increased survival in these models; these findings were recently published.[1] TNX-1700 addresses a central mechanism of therapeutic resistance to anti-PD-1 therapy in gastric cancer by targeting the CXCR4-driven myeloid axis to normalize cancer-induced myelopoiesis and reprogram the tumor microenvironment.

About BTLA
BTLA (B and T lymphocyte attenuator) is a protein on the surface of tumor infiltrating lymphocytes. Targeting BTLA is a promising target in immuno-oncology since its ligand HVEM is expressed and/or upregulated in the tumor microenvironment of many cancers including melanoma, non-small cell lung cancer, colorectal cancer, gastric cancer, glioblastoma, and prostate cancer and generally correlates with reduced overall survival. Targeting BTLA offers opportunities for cancer immunotherapy and may demonstrate additive or synergistic effects when combined with other checkpoint antagonists, potentially overcoming resistance mechanisms and improving clinical outcomes.

About TNX-4700
Tonix is developing TNX-4700 (anti-BTLA) mAb for immuno-oncology indications. The mAb technology was licensed from Curia.

Citations:
1. Qian J, et al. *Cancer Cell*. 2025. 43(8):1512-1529.e11.

Tonix Pharmaceuticals Holding Corp.
Tonix Pharmaceuticals* is a fully integrated, commercial-stage biotechnology company focused on central nervous system (CNS) and immunology treatments in areas of high unmet medical need. TONMYA® (cyclobenzaprine HCl sublingual tablets 2.8 mg), is the first new treatment for fibromyalgia in adults in more than 15 years. Tonix's CNS commercial infrastructure supports its marketed products, including its acute migraine products, Zembrace® Symtouch® (sumatriptan injection 3 mg) and Tosymra® (sumatriptan nasal spray 10 mg). Tonix is investigating TONMYA® in Phase 2 clinical trials to evaluate its potential in major depressive disorder and acute stress disorder/acute stress reaction. Tonix is also advancing a pipeline of immunology programs, including TNX-4800, a Phase 2 ready long-acting human anti-*Borrelia* OspA monoclonal antibody (mAb) for the prevention of Lyme disease in the U.S., and TNX-1500, a Phase 2 ready third-generation CD40 ligand inhibitor for the prevention of kidney transplant rejection. In addition, the Company is progressing TNX-2900 (intranasal potentiated oxytocin), which is Phase 2 ready for the treatment of Prader-Willi syndrome, a rare disease. To learn more, visit www.tonixpharma.com and follow the Company on LinkedIn and X.

**Tonix's product development candidates are investigational new drugs or biologics; their efficacy and safety have not been established and have not been approved for any indication.*

Forward Looking Statements
Certain statements in this press release are forward-looking within the meaning of the Private Securities Litigation Reform Act of 1995 including those relating to the completion of the offering, the satisfaction of customary closing conditions, the intended use of proceeds from the offering and other statements that are predictive in nature. These statements may be identified by the use of forward-looking words such as "anticipate," "believe," "forecast," "estimate," "expect," and "intend," among others. There are a number of factors that could cause actual events to differ materially from those indicated by such forward-looking statements. These factors include, but are not limited to, risks related to the failure to successfully launch and commercialize TONMYA® and any of our approved products; risks related to the failure to obtain FDA clearances or approvals and noncompliance with FDA regulations; risks related to the timing and progress of clinical development of our product candidates; our need for additional financing; uncertainties of patent protection and litigation; uncertainties of government or third party payor reimbursement; limited research and development efforts and dependence upon third parties; and substantial competition. As with any pharmaceutical under development, there are significant risks in the development, regulatory approval and commercialization of new products. Tonix does not undertake an obligation to update or revise any forward-looking statement. Investors should read the risk factors set in the Company's Annual Report on Form 10-K for the year ended December 31, 2025, as filed with the SEC on March 12, 2026, and periodic reports filed with the SEC on or after the date thereof. All of Tonix's forward-looking statements are expressly qualified by all such risk factors and other cautionary statements. The information set forth herein speaks only as of the date thereof.

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Exhibit 99.02

Abstract 6550

In Vitro Characterization of Fully Human Antagonistic anti-BTLA Monoclonal Antibodies

Bruce L. Daugherty[1], Hsunhui Yang[2], Subhra Mahapatra[2], Yadong Yu[2], Christine L. Hsieh[2], Brian A. Zabel[2], Seth Lederman[1]

[1]Tonix Pharmaceuticals, Inc., Berkeley Heights, NJ 07922, [2]Curia Bio, Inc., Hayward, CA 94545

Abstract

Introduction: B and T lymphocyte attenuator (BTLA) is an immune checkpoint receptor essential for immune homeostasis and tolerance. Unlike PD-1 or CTLA-4, BTLA is broadly expressed on lymphoid and some myeloid cells, including T cells, B cells, dendritic cells, and macrophages. Engagement with its ligand HVEM (herpesvirus entry mediator) transmits inhibitory signals that suppress immune activation. Within tumors, BTLA contributes to immune evasion by dampening anti-tumor responses. Given its unique expression profile and regulatory function, we developed monoclonal antibodies (mAbs) targeting BTLA as an immunotherapeutic strategy to enhance anti-tumor immunity.

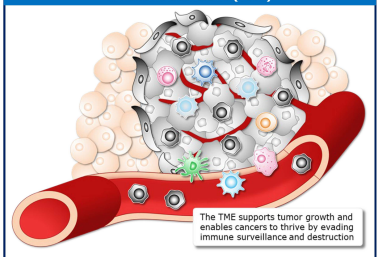
Methods: Fully human anti-BTLA mAbs identified by hybridoma screening were reformatted as huIgG4 (S228P/L235A) and transiently expressed using Curia's TunaCHO® platform. The S228P mutation prevents Fab-arm exchange, stabilizing mAb structure, while L235A minimizes FcγR binding and effector functions. Binding potency was determined by ELISA; kinetics by Octet® biolayer interferometry (BLI) against human and cynomolgus BTLA. BLI also assessed FcγR, FcRn (at pH 6.0 vs 7.2), and C1q binding to evaluate recycling potential and complement activation. Neutralization of HVEM binding to BTLA was quantified by modified ELISA and cell-based reporter assays.

Results: Six fully human anti-BTLA mAbs and clinical candidate comparator JS004 displayed similar ~1 nM binding potencies (EC50) values to human and cyno BTLA by ELISA. Clone 13-F7-A exhibited the highest affinity (KD < 1 nM) for both human and cyno BTLA among all 7 mAbs tested. Four mAbs and the comparator JS004 were high affinity binders to human and cyno BTLA (KD 1-32 nM). All six fully human mAbs exhibited reduced FcγR binding relative to JS004. Each bound FcRn at pH 6.0 but not at 7.2, consistent with normal endosomal recycling. None bound C1q, whereas a positive-control IgG1 did bind to C1q as expected. In an ELISA-based biofunctional assay, all mAbs blocked HVEM binding to BTLA with similar IC50 values (~1 nM). In a T cell reporter assay, four antibodies and the comparator JS004 reversed HVEM:BTLA-dependent suppression of TCR→NFAT→luciferase signaling (IC50: 9-31 nM).

Conclusions: We generated potent, high affinity, human/cyno cross-reactive, fully human antagonistic anti-BTLA mAbs. Targeting BTLA offers promising opportunities for cancer immunotherapy and may demonstrate strong synergy when combined with other checkpoint antagonists, potentially overcoming resistance mechanisms and improving clinical outcomes.

Introduction

Tumors Create a Toxic, Immunosuppressive Microenvironment (TME)



Solid Tumors with HVEM Upregulation

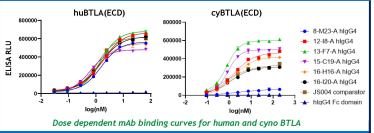
- Melanoma: HVEM is overexpressed in both metastatic melanoma samples and cultured cell lines. It is often found physically adjacent to BTLA+ tumor-infiltrating lymphocytes (TILs), making it a prime candidate for BTLA blockade[1].
- Non-Small Cell Lung Cancer (NSCLC): HVEM is upregulated in approximately 18-20% of NSCLC biopsies. Its expression is often independent of PD-L1, suggesting that BTLA-targeted therapy could benefit patients who do not respond to traditional PD-1 inhibitors[2].
- Colorectal Cancer (CRC): High HVEM levels in malignant lesions are linked to advanced pathological stages and poor prognosis. Preclinical studies show that blocking the HVEM-BTLA interaction can significantly reduce tumor growth in CRC models[3].
- Gastric & Digestive Cancers: Upregulation of both HVEM and BTLA in gastric cancer tissues correlates with lymph node metastasis and decreased overall survival[1].
- Hepatocellular Carcinoma (HCC): HVEM expression is associated with postoperative recurrence and poor survival outcomes in liver cancer[1].
- Glioblastoma & Glioma: Increased HVEM expression is found in aggressive glioma subtypes and is linked to poorer prognosis[3].
- Breast Cancer: High HVEM expression is linked to aggressive tumor features and poor prognosis in invasive breast cancer patients[3].
- Prostate Cancer: Recent studies indicate that HVEM negatively regulates the anti-tumor response in prostate cancer, and its blockade has shown promise in humanized mouse models[1].

Advantages of BTLA-targeted Cancer Immunotherapy

- PD-1/PD-L1 Independence...: HVEM expression is often not linked to PD-L1 status, offering an alternative pathway for "cold" tumors[4].
- ...as well as Synergistic Potential: Combining anti-BTLA mAbs with anti-PD-1 has shown enhanced T cell restoration in early clinical trials[5].
- Exhaustion Reversal: BTLA is typically expressed on terminally exhausted T cells that are the most dysfunctional and resistant to standard PD-1 therapy[6]; blocking BTLA can "un-brake" these specific cells to restore their killing capacity.
- Bidirectional Control: Unlike many checkpoints, BTLA:HVEM signaling is bidirectional. While BTLA sends "stop" signals to T cells, HVEM can send survival signals to tumor cells. Blocking this axis potentially hits both sides: it reactivates the immune system and stops the tumor from receiving survival cues[6].
- Broad Immune Impact: Beyond T cells, BTLA is expressed on B cells, NK cells, and macrophages. BTLA antagonism can improve NK cell activity and promote anti-tumor macrophage activity[7].

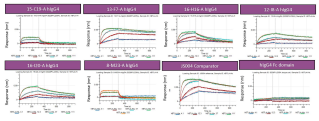
Results

Fig 1: ELISA Binding Potency



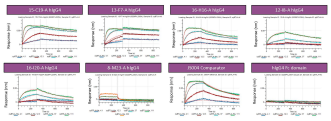
Dose dependent mAb binding curves for human and cyno BTLA

Fig 2: huBTLA Binding Affinity by BLI



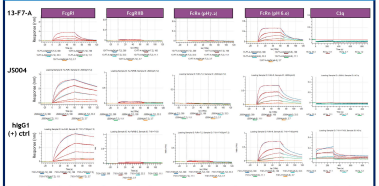
5 min association, 15 min dissociation, and 4 concentrations of huBTLA tested for binding to mAbs loaded on AHC biosensors

Fig 3: cyBTLA Binding Affinity by BLI



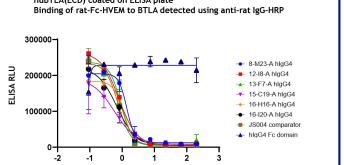
5 min association, 15 min dissociation, and 4 concentrations of cyBTLA tested for binding to mAbs loaded on AHC biosensors

Fig 4: FcγR, FcRn and C1q Binding by BLI



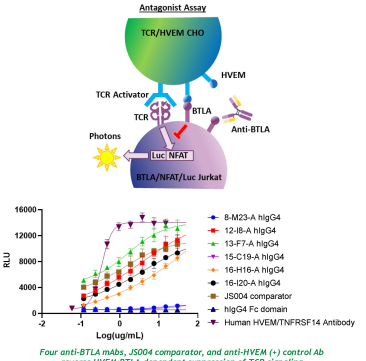
1 min association, 1 min dissociation, and 5 concentrations of mAbs tested for binding to FcgR and FcRn loaded on biosensors. mAbs were loaded on biosensors for C1q kinetics.

Fig 5: ELISA Biofunction IC50 Binding Inhibition



Dose dependent inhibition of BTLA binding to HVEM by anti-BTLA mAbs

Fig 6: T cell Reporter Assay



Four anti-BTLA mAbs, JS004 comparator, and anti-HVEM (+) control Ab reverse HVEM:BTLA-dependent suppression of TCR signaling

Binding Affinities and Functional Characterization of Fully Human BTLA mAbs

Ab ID	T cell Reporter Assay	ELISA	huBTLA	cyBTLA	baBTLA	cjBTLA	FcgRI	FcgRIIA	FcgRIIB	FcgRIIIA-F	FcgRIIIA-V	FcgRIIIB	FcRn pH7.2	FcRn pH6.0	C1q
13-C19-A	n.d.	0.97	3.15E-09	1.00E-09	3.83	0.86	1.54E-07	1.07E-08	1.74E-06	n.d.	n.d.	n.d.	n.d.	4.66E-08	n.d.
13-F7-A	10.8	0.24	8.57E-10	6.26E-10	1.66	0.77	2.74E-07	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	6.80E-08	n.d.
16-H16-A	30.9	1.12	1.52E-09	3.29E-09	1.22	1.65	2.42E-07	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	8.99E-08	n.d.
12-I8-A	20.5	0.8	1.29E-09	1.29E-09	1.65	1.65	1.74E-07	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	5.10E-08	n.d.
16-I20-A	20.9	0.69	0.94E-09	1.94E-09	1.42	1.20	1.18E-07	n.d.	8.21E-08	n.d.	n.d.	n.d.	n.d.	4.07E-08	n.d.
8-M23-A	n.d.	1.4	3.29E-07	2.14E-08	1.58	2.05	2.69E-07	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	6.56E-08	n.d.
JS004 Reference Ab	8.8	0.76	1.56E-09	5.76E-09	1.37	0.55	2.71E-06	n.d.	2.59E-07	n.d.	n.d.	n.d.	n.d.	6.30E-08	n.d.
hIgG4 Fc(-) ctrl	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	1.85E-07	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	1.94E-08	n.d.
Control hIgG1 (+) ctrl	n.t.	n.t.	n.t.	n.t.	n.t.	n.t.	1.28E-08	9.19E-07	9.92E-08	5.42E-08	5.97E-08	n.d.	n.d.	6.56E-08	7.78E-10

Favorable (green) or problematic (red) features

n.d. not detected
n.t. not tested

Conclusions

- We generated four potent, high affinity, human/cyno cross-reactive, fully human antagonistic anti-BTLA mAbs. Three antagonists, 13-F7-A, 16-H16-A, and 12-I8-A, had reduced FcgRI binding and no binding to FcgRIIB compared to clinical candidate JS004, and thus may have improved pharmacokinetics and reduced chance of FcR-dependent adverse events (e.g. cytokine release syndrome or other immune-mediated toxicities).
- Targeting BTLA is ideal since its ligand HVEM is expressed and/or upregulated in the TME of many cancers including melanoma, NSCLC, CRC, gastric cancer, glioblastoma, and prostate cancer and generally corelates with reduced overall survival[1,3].
- Targeting BTLA offers promising opportunities for cancer immunotherapy and may demonstrate strong synergy when combined with other checkpoint antagonists, potentially overcoming resistance mechanisms and improving clinical outcomes.

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AACR Annual Meeting, April 17-22, 2026, San Diego, CA

Exhibit 99.03

Pharmacokinetics of TNX-1700 in Non-Human Primates and Human FcRn/Serum Albumin Transgenic Mice

Abstract 7940

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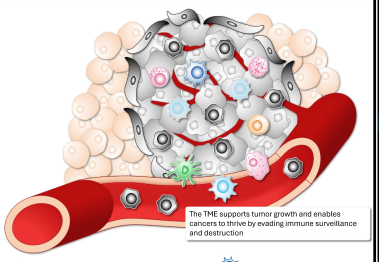
Abstract

Introduction: TNX-1700 is a novel recombinant fusion molecule of human Trefoil Factor-2 (TFF2) protein and human serum albumin (HSA) that is being investigated as a potential therapeutic for gastric cancer. In syngeneic mouse models of gastric and colorectal cancer, TNX-1700 functions as a CXCR4 partial agonist that activates antitumor immunity in the tumor microenvironment by modulating myeloid cell trafficking to reduce tumor-induced granulopoiesis and accumulation of immunosuppressive neutrophils[1]. TNX-1700 is engineered to extend plasma half-life, enhance systemic exposure, and improve cancer immunotherapy. The HSA domain in TNX-1700 provides a long circulatory half-life (>14 days) and multiple ligand-binding sites. Approved albumin-linked drugs include detemir (Levemir®), liraglutide (Victoza®), and albiglutide (Eperzan®/Tanzeum®) for diabetes, and nanoparticle albumin-bound paclitaxel (nab-paclitaxel) for cancer therapy. TNX-1700 represents a next-generation application of the albumin platform in immuno-oncology.

Methods: The pharmacokinetics (PK) of TNX-1700 were evaluated in non-human primates (NHP; cynomolgus macaques) and double-transgenic mice expressing human neonatal Fc receptor (FcRn) and human serum albumin (HSA). Animals received a single dose of 1 mg/kg or 3 mg/kg TNX-1700, administered intravenously (IV) to NHPs or intraperitoneally (IP) to FcRn/HSA mice. For comparison, untagged human TFF2 (molar equivalent to TNX-1700) was also administered into FcRn/HSA mice. Serial blood samples were collected over 0-35 days and analyzed using the Boster PicoKine™ Human TFF2 ELISA kit. Pharmacokinetic parameters were determined by non-compartmental analysis.

Introduction

Tumors Create a Toxic, Immunosuppressive Microenvironment (TME)



- *Healthy cell*
- *Malignant cell*
- *Myeloid-derived suppressor cell (MDSC)*
- *Cancer-associated fibroblast*
- *Exhausted CD8 T cell*
- *Cytotoxic CD8 T cell*
- *CD4 T cell*
- *Dendritic cell (DC)*
- *B cell*
- *Natural Killer (NK) cell*
- *Macrophage*
- *Neutrophil*

- Tumors are surrounded by endothelial and stroma cells, and invading immune cells, both innate and adaptive[2,3]
- Complex regulatory network supports tumor growth, enabling cancers to thrive by evading immune surveillance and destruction[3,4]
- The TME sabotages tumor-killing cytotoxic CD8 T cells[2]
- Myeloid-derived suppressor cells (MDSCs) interfere with anticancer immunity[3,4]

Study Design for a Pharmacokinetic Study in Humanized Mice and Non-Human Primates

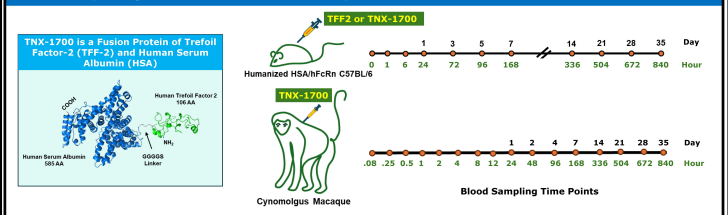


Figure 1: Study design adopted for single dose pharmacokinetics (PK) of TNX-1700 in double-transgenic mice expressing human neonatal Fc receptor (FcRn) and human serum albumin (HSA), and non-human primates (NHP; cynomolgus macaques). TNX-1700 was administered intraperitoneally (IP) to FcRn/HSA mice. For comparison, untagged human TFF2 (molar equivalent to TNX-1700) was also administered into FcRn/HSA mice. TNX-1700 was administered intravenously (IV) to NHPs.

Results

Pharmacokinetic Profile of TFF2 and TNX-1700 in Humanized FcRn/HSA C57BL/6 Mice

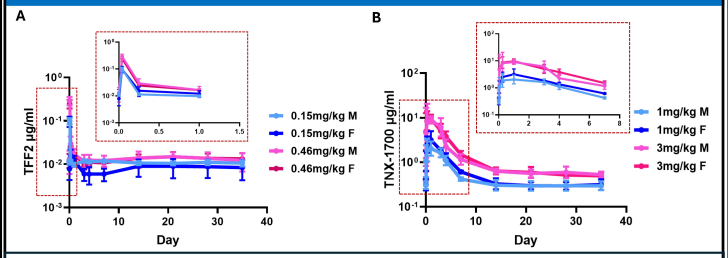


Figure 2: PK profile of TFF2 and TNX-1700 in humanized mice. A. TFF2 dosed via IP at 0.15 or 0.46 mg/kg. B. TNX-1700 dosed via IP at 1 or 3 mg/kg.

Pharmacokinetic Profile of TNX-1700 in Cynomolgus Macaques

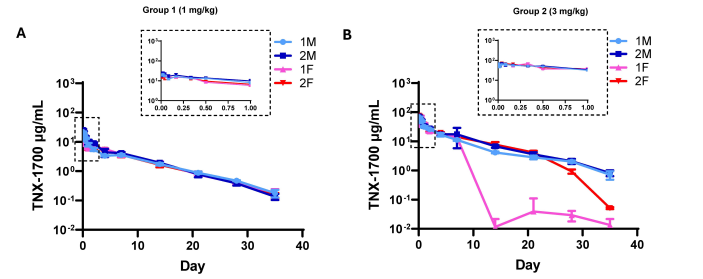


Figure 3: PK profile of TNX-1700 in NHPs. A. Group 1 was dosed via IV at 1 mg/kg TNX-1700. B. Group 2 was dosed via IV at 3 mg/kg TNX-1700.

Antibody Response in NHPs

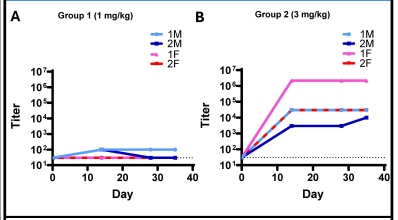


Figure 4: Anti-TNX-1700 IgG ELISA responses. A. Group 1 represents the anti-TNX-1700 profile in NHPs dosed with 1 mg/kg of TNX-1700. B. Group 2 represents the anti-TNX-1700 profile in NHPs dosed with 3 mg/kg of TNX-1700.

Pharmacokinetics of TFF2 and TNX-1700 in Human FcRn/HSA Mice and Non-human Primates

Human FcRn/HSA Mice, Single Dose, IP

TFF2	0.15 mg/kg	0.46 mg/kg
C_{max}	0.10 µg/mL	0.27 µg/mL
AUC_{0-t}	8.2 h*µg/mL	12.0 h*µg/mL

TNX-1700	1.0 mg/kg	3.0 mg/kg
C_{max}	3.0 µg/mL	10.3 µg/mL
AUC_{0-t}	1642 h*µg/mL	2318 h*µg/mL

NHP, Single Dose, IV

TNX-1700	1.0 mg/kg	3.0 mg/kg
C_{max}	21.3 mg/mL	71.4 mg/mL
AUC_{0-t}	1868 h*mg/mL	6484 h*mg/mL
$T_{1/2}$	7.1 Days	7.0 Days

All animals survived without clinical signs or >10% body-weight loss. Comparable PK profiles were observed across species and doses. In cynomolgus macaques, mean terminal half-life (t½) was 7.1 days (%CV = 9.65), clearance (CL) 13.3 mL/day (%CV = 14.3), and volume of distribution (Vz) 135.2 mL (%CV = 18.3). Allometric scaling predicted in humans a t½ of 14.2 days (%CV = 12.9), CL = 105.2 mL/day (%CV = 26.4), and Vz = 2,158 mL (%CV = 34.0). Results from the humanized murine studies provided evidence that untagged human TFF2 is rapidly cleared and that fusion with HSA significantly increased the PK profile similar to that observed in NHPs and to levels supportive for clinical candidates.

Conclusions

TNX-1700 exhibited dose-independent, linear pharmacokinetics with low inter-animal variability, and exposure was consistent across doses and species. Although its half-life is shorter and clearance higher than IgG-based biologics, TNX-1700 substantially extends the half-life of TFF2 and achieves durable systemic exposure, supporting its potential as a therapeutic candidate for gastric cancer.

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