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): May 29, 2019

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.)**

509 Madison Avenue, Suite 1608, New York, New York 10022
(Address of principal executive offices) (Zip Code)

Registrant's telephone number, including area code: (212) 980-9155

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))

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. ☐
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 Market

Item 7.01 Regulation FD Disclosure.

Tonix Pharmaceuticals Holding Corp. (the "Company") presented a poster (the "Poster Presentation") entitled "Steady-State Pharmacokinetic Properties of a Sublingual Formulation of Cyclobenzaprine (CBP) HCl (TNX-102 SL*): Comparison to Simulations of Oral Immediate Release CBP" on May 29, 2019 at the American Society of Clinical Psychopharmacology (ASCP) 2019 Annual Meeting. Copies of the Poster Presentation and the press release that discusses this matter are filed as Exhibits 99.01 and 99.02, respectively, to, and incorporated by reference in, this report.

The information in this Item 7.01 of this Current Report on Form 8-K, including Exhibit 99.01 and Exhibit 99.02 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 it 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 9.01 Financial Statements and Exhibits.

(d)

Exhibit No.	Description.
99.01	Poster Presentation
99.02	Press Release dated May 30, 2019, issued by the Company

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: May 30, 2019

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

Exhibit 99.01

Steady-State Pharmacokinetic Properties of a Sublingual Formulation of Cyclobenzaprine (CBP) HCl (TNX-102 SL*): Comparison to Simulations of Oral Immediate Release CBP

W29

Gregory Sullivan[1], Regina Kiu[1], Helen Stillwell[1], Bernd Meibohm[2], and Seth Lederman[1]

[1]Tonix Pharmaceuticals Inc, [2]U Tennessee Health Sciences Center

*TNX-102 SL is an investigational new drug and has not been approved for any indication

INTRODUCTION

TNX-102 SL is a sublingual (SL) formulation of cyclobenzaprine (CBP) designed for bedtime dosing that is being developed for the indications of posttraumatic stress disorder (PTSD), fibromyalgia (FM) and agitation in Alzheimer's Disease (AAD). Each is a central nervous system (CNS) condition in which sleep disturbances in slow wave and/or rapid eye movement sleep play central roles in the illness expression, and TNX-102 SL targets improvement in sleep quality.

CBP is a tricyclic molecule that has a unique receptor binding profile with high potency binding to serotonin-2A (5-HT_{2A}), α_1-adrenoceptor, H_1-histaminergic, and M_1-muscarinic cholinergic receptors. CBP acts as an antagonist on these receptors, and each have been suggested to have roles for enhancing different aspects of sleep quality. The primary metabolite, norcyclobenzaprine (nCBP), which has: (1) a long half-life of several days, (2) a binding profile similar to but less potent than CBP for these four receptors, except that nCBP has more potent binding and inhibitory activity at the norepinephrine transporter (NET), which enhances sympathetic activation and is disruptive to sleep quality. **Table 1.** shows the binding affinities (K_i) of CBP and nCBP on human receptors and transporters.

Table 1. Affinity (K_i in nM) of CBP and nCBP on Human Neuroreceptors & Transporters

	H_1	5-HT_{2A}	α_{1A}	α_{1B}	M_1	SERT	NET
Cyclobenzaprine (CBP)	1.2	5.4	7.1	8	8.4	29	89
Norcyclobenzaprine (nCBP)	17.8	55	82	71	355	461	12.8

TNX-102 SL was formulated to allow rapid sublingual absorption for bedtime dosing, which results in a CBP pharmacokinetic (PK) plasma concentration curve with maximal levels achieved during middle of the sleep phase and falling levels by the end. The oral mucosal absorption of TNX-102 SL largely bypasses hepatic first pass metabolism, resulting in a substantially lower levels of the long-lived and undesirable metabolite, nCBP. Previous Phase 1 PK studies comparing TNX-102 SL with immediate release (IR) oral CBP show TNX-102 SL provides sublingual transmucosal absorption, rapid systemic exposure, avoidance of first-pass metabolism, and lower exposure to the long-lived active major metabolite, nCBP, shown in Table 2.

Table 2. TNX-102 SL vs. Oral IR CBP: Single-Dose Pharmacokinetic Parameters

Parameter	TNX-102 SL 2.8 mg	Oral IR CBP 5 mg	TNX-102 SL Compared To Oral IR
	Cyclobenzaprine		
Absorption Lag Time	0.250 hr (15 min)	0.622 hr (37 min)	12x Faster
Relative Bioavailability	154%		54% higher
C_{max}	3.41 ng/mL	4.26 ng/mL	20% lower
$AUC_{0-\infty}$	57.4 ng•hr/mL	69.3 ng•hr/mL	17% lower
	Norcyclobenzaprine		
C_{max}	8.61 ng/mL	1.71 ng/mL	53% lower
$AUC_{0-\infty}$	90.5 ng•hr/mL	56.6 ng•hr/mL	48% lower
	Cyclobenzaprine/Norcyclobenzaprine		
Ratio $AUC_{0-\infty}$	1.58	1.18	89% higher

In this pivotal multi-dose bridging PK and safety study (TNX-CT-F106/P106[1]), TNX-102 SL 5.6 mg (2 x 2.8 mg tablets) was compared with AMRIX® (CBP extended release [ER] capsules) 30 mg at steady-state (ss) for CBP and major metabolite nCBP, and the overall profiles of metabolites were compared. Additionally, the PK profile of TNX-102 SL 5.6 mg at SS from this bridging study is also contrasted with a simulated SS PK profile of oral IR CBP 10 mg as modeled from a prior single dose PK study that included oral IR CBP arm (TNX-CT-F104) to assess the potential PK and pharmacodynamic (PD) differentiation of TNX-102 SL from the sustained release of CBP.

METHODS

Study P106 was a single center, comparative PK, open-label, randomized, multiple dose, 2-arm, parallel study conducted to support the registration of TNX-102 SL 5.6 mg. This pivotal exposure/safety bridging study compared the PK of TNX-102 SL at its potential therapeutic daily dose, 5.6 mg (2 x 2.8 mg tablets) with the reference listed drug [RLD], AMRIX ER capsules at the maximum recommended daily dose, 30 mg. F106 compared (1) the rate and extent of absorption, (2) the safety and tolerability, (3) the systemic exposure, and (4) the metabolic profile of the two drug products after reaching steady state exposure in 60 healthy adult volunteers under fasting conditions. The study included a 30-day screening period, 21-day confinement period with a 20-day daily dosing to reach steady state PK blood collections up-to 648 hours (27 days) post-last dose. Fifty-six (56) healthy volunteers ages ≥18 and ≤35 years were assigned to one of the two treatments:

Treatment A (N=26): 2 x TNX-102 SL (cyclobenzaprine HCl sublingual tablets) 2.8 mg once daily for 20 consecutive days

Treatment B (N=18): 1 x cyclobenzaprine HCl ER capsule (AMRIX) 30 mg once daily for 20 consecutive days.

Four (4) volunteers ages ≥65 and ≤75 years old were enrolled and assigned to **Treatment A**, but PK parameters for these four were analyzed separately and are not included in the displayed tables and figures.

The following PK parameters were calculated by standard non-compartmental methods for CBP and nCBP:

Day 1: AUC_{0-12}, C_{max}, T_{max}

Day 20: $AUC_{0-\tau,ss}$, $AUC_{0-\infty,ss}$, $C_{max,ss}$, $C_{min,ss}$, Residual area, T_{max}, $T_{1/2}$, K_{el}, F(%)

Safety variables include adverse events, clinical lab tests, vital signs, electrocardiogram and physical exam

The F106 TNX-102 SL 5.6 mg PK data also served as the comparator with PK simulations of steady state oral IR CBP 10 mg. The concentration-time profiles of CBP and nCBP during multiple dosing at steady state for oral IR CBP 10 mg once daily dosing were simulated from single-dose oral IR CBP 5 mg data from previous Phase 1 study TNX-CT-F104 (T104F) using nonparametric superposition methodology under the assumption of linear pharmacokinetics for CBP and nCBP. The adequacy of this methodology and assumption is supported by the good agreement between the predicted CBP and nCBP concentrations during multiple dosing of TNX-102 SL 5.6 mg once daily based on the TNX-102 SL 2.8 mg single dose administration in study F104 and the actually observed concentration time profiles in study F106 (see Figure 3).

RESULTS

F106 Pharmacokinetic Results:

PK results show that following the first dose of TNX-102 SL 5.6 mg on Day 1 and the last dose on Day 20, exposure levels of CBP and its long-lasting major metabolite, nCBP, from TNX-102 SL 5.6 mg were less than AMRIX ER 30 mg.

Table 3 and **Table 4** show Day 1 PK parameters for CBP for nCBP, respectively. For TNX-102 SL 5.6 mg and AMRIX ER 30 mg.

Table 3. Day 1 Plasma Cyclobenzaprine PK Parameters for TNX-102 SL 5.6 mg vs. AMRIX 30 mg

	TNX-102 SL 5.6 mg (2 x 2.8 mg Tablets) (Treatment A)				AMRIX 30 mg (1 x 30 mg ER Capsule) (Treatment B)			
Parameter (unit)	N	Mean	SD	CV%	N	Mean	SD	CV%
AUC_{0-12} (h*pg/mL)	26	69115	28441	38.3	30	181587	90115	37.5
C_{max} (pg/mL)	26	5178	2122	26.5	30	13895	1268	26.5
Parameter (unit)	N	Median	Min	Max	N	Median	Min	Max
T_{max} (h)	26	5.8	2.0	9.0	30	8.0	5.0	14.0

Table 4. Day 1 Plasma Norcyclobenzaprine PK Parameters for TNX-102 SL 5.6 mg vs. AMRIX 30 mg

	TNX-102 SL 5.6 mg (2 x 2.8 mg Tablets) (Treatment A)				AMRIX 30 mg (1 x 30 mg ER Capsule) (Treatment B)			
Parameter (unit)	N	Mean	SD	CV%	N	Mean	SD	CV%
AUC_{0-12} (h*pg/mL)	26	224386	56206	25.1	30	725838	204044	28.2
C_{max} (pg/mL)	26	1351	330	24.8	30	4094	1259	28.4
Parameter (unit)	N	Median	Min	Max	N	Median	Min	Max
T_{max} (h)	26	14.0	8.0	25.9	30	21.9	11.0	24.0

Table 5 and **Table 6** show Day 20 steady state PK parameters for CBP for nCBP, respectively, for TNX-102 SL 5.6 mg and AMRIX ER 30 mg.

Table 5. Day 20 Plasma Cyclobenzaprine PK Parameters for TNX-102 SL 5.6 mg vs. AMRIX 30 mg

	TNX-102 SL 5.6 mg (2 x 2.8 mg Tablets) (Treatment A)				AMRIX® 1 x 30 mg ER Capsule (Treatment B)			
Parameter (unit)	N	Mean	SD	CV%	N	Mean	SD	CV%
AUC_{0-12} (h*pg/mL)	26	174712	101480	58.1	30	603739	204457	30.5
$AUC_{0-\tau}$ (h*pg/mL)	26	498862	492177	98.7	30	2864790	909970	44.5
$AUC_{0-\infty}$ (h*pg/mL)	26	508080	494876	97.4	30	2872903	910721	43.9
Residual Area (%)	26	2.37	1.03	43.4	30	8.46	0.22	48.7
$C_{max,ss}$ (pg/mL)	26	11286	5659	50.5	30	39603	11863	30.0
$C_{min,ss}$ (pg/mL)	26	4910	3511	71.9	30	20604	7498	36.2
F(%)	26	92.9	25.2	24.9	30	70.4	12.5	17.5
K_{el} (1/h)	26	0.018	0.005	24.4	30	0.021	0.005	25.4
$T_{1/2}$ (h)	26	40.3	10.4	25.7	30	35.6	8.3	23.2
Parameter (unit)	N	Median	Min	Max	N	Median	Min	Max
T_{max} (h)	26	5.0	2.0	9.0	30	7.0	5.0	9.0

Table 6. Day 20 Plasma Norcyclobenzaprine PK Parameters for TNX-102 SL 5.6 mg vs. AMRIX 30 mg

	TNX-102 SL 2 x 2.8 mg Tablets (Treatment A)				AMRIX 1 x 30 mg ER Capsule (Treatment B)			
Parameter (unit)	N	Mean	SD	CV%	N	Mean	SD	CV%
AUC_{0-12} (h*pg/mL)	26	204981	92984	45.4	30	812043	336032	41.3
$AUC_{0-\tau}$ (h*pg/mL)	26	1273439	980658	77.1	30	5709126	3986758	71.8
$AUC_{0-\infty}$ (h*pg/mL)	26	1285031	980324	77.7	30	4722586	3396773	71.8
Residual Area (%)	26	8.95	0.59	81.5	30	8.26	0.20	77.5
$C_{max,ss}$ (pg/mL)	26	10170	4681	46.4	30	40218	16984	42.8
$C_{min,ss}$ (pg/mL)	26	6858	3282	46.1	30	27708	12236	44.2
F(%)	26	80.9	9.2	22.4	30	37.1	9.7	26.8
K_{el} (1/h)	26	0.011	0.003	27.6	30	0.012	0.004	31.1
$T_{1/2}$ (h)	26	60.7	18.1	27.1	30	64.8	17.4	27.2
Parameter (unit)	N	Median	Min	Max	N	Median	Min	Max
$T_{max,ss}$ (h)	26	8.0	6.0	12.0	30	8.8	5.0	24.1

Figure 1 shows Day 1 and Day 20 mean concentration-time profiles for plasma cyclobenzaprine for TNX-102 SL 5.6 mg and AMRIX ER 30 mg. **Figure 2** shows Day 1 and 20 day 20 mean concentration-time profiles for norcyclobenzaprine for TNX-102 SL 5.6 mg and AMRIX ER 30 mg.

Figure 1. Day 1 & Day 20 Cyclobenzaprine Levels for TNX-102 SL 5.6 mg vs. AMRIX 30 mg

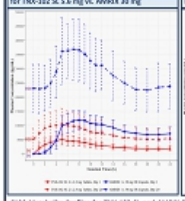
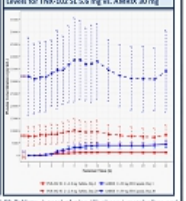


Figure 2. Day 1 & Day 20 Norcyclobenzaprine Levels for TNX-102 SL 5.6 mg vs. AMRIX 30 mg



F106 Metabolite Profiles for TNX-102 SL and AMRIX ER Tablets: A total of nine (9) phase I metabolites and eight (8) phase II metabolites were detected in human plasma dosed with CBP. All metabolites detected in the human plasma dosed with TNX-102 SL 5.6 mg were also detected in the samples dosed with AMRIX 30 mg ER capsules. Higher responses of the phase I and II (glucuronide conjugates) metabolites were detected in the volunteers dosed with AMRIX 30 mg ER capsules. In light of these results, it is reasonable to assume that the metabolic profiles are comparable in human plasma for both TNX-102 SL 5.6 mg and AMRIX 30 mg ER capsules. Moreover, the volunteers dosed with AMRIX 30 mg ER capsules were exposed to a higher concentration of each metabolite compared to the volunteers dosed with TNX-102 SL 5.6 mg.

F106 Safety Results and Prior Phase 1 Safety Results from Same Center:

In F106 the most commonly reported treatment-emergent adverse event (TEAE) reported by 38 subjects receiving 28 days of TNX-102 SL 5.6 mg were hypoesthesia oral (HO) (58.3%), product taste abnormal (43.3%), somnolence (33.3%), heart rate increase (30.0%), constipation (26.7%), blood pressure increase (16.7%), nausea (13.3%), paresthesia oral (10.0%), and dry mouth (10.0%). HO and/or paresthesia oral (PO) was reported 3 days or less in 28.7%, and 7 days or more in 23.8% of subjects receiving TNX-102 SL 5.6 mg (**Figure 6**). All reports were rated as mild and were transient (lasting an average of ~60 minutes or less).

For the 30 receiving 20 days of AMRIX 30 mg, most commonly reported TEAEs were heart rate increase (53.3%), constipation (40%), dry mouth (36.7%), headache (36.7%), blood pressure increase (13.3%), palpitations 13.3%, abnormal dreams and nightmares increase (13.3%), dizziness (10.0%) and product taste abnormal (10.0%).

No deaths, serious, or significant AEs were reported during the study.

Figure 3. Day 20 Concentration-Time Profiles of TNX-102 SL 5.6 mg Observed (F106) vs. Simulated (F104)

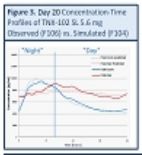


Figure 3 shows the observed concentration-time profile for TNX-102 SL 5.6 mg from study F106 for CBP and nCBP and the simulated profile based on single dose data for TNX-102 SL 2.8 mg from F104. The good agreement between the observed and predicted data support the adequacy of this methodology and assumption employed (nonparametric superposition methodology under assumption of linear pharmacokinetics for CBP and nCBP).

Figures 4 & 5 below show the Day 20 concentration-time profiles of CBP and nCBP for TNX-102 SL 5.6 mg and simulated oral IR CBP at 5 mg & 10 mg, respectively. During sleep hours (0-8 hrs post-dose), CBP steady state levels and AUC are higher than nCBP post TNX-102 SL, unlike the pattern observed with simulated oral IR CBP, with nCBP higher.

Figure 4. Day 20 Concentration-Time Profiles for TNX-102 SL 5.6 mg vs. Simulated Oral IR Cyclobenzaprine 5 mg

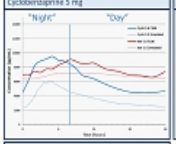


Figure 5. Day 20 Concentration-Time Profiles for TNX-102 SL 5.6 mg vs. Simulated Oral IR Cyclobenzaprine 10 mg

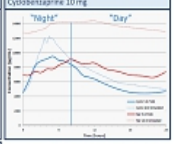


Figure 6. AEs of Hypoesthesia Oral and/or Paresthesia Oral in F106: No. of Days Reported

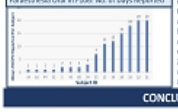


Figure 6 shows the total number of days per subject receiving TNX-102 SL 5.6 mg in which either hypoesthesia oral (HO) or paresthesia oral (PO) was reported. Of the 30 subjects, 30% reported at least occurrence on one day, and all occurrences were temporally related to dosing. Of the 30, 23.3% reported occurrence on a total of 7-20 days, and 23.7% reported occurrence on 3 or less days. All were rated as mild, lasted an average of 60 minutes, and never were associated with an oral lesion.

CONCLUSION

- At steady-state, exposure to CBP and nCBP from TNX-102 SL were less than from AMRIX.
- Overall, TNX-102 SL 5.6 mg or AMRIX 30 mg taken for 20 days were well tolerated.
- For the intended therapeutic daily bedtime administration, these data show during typical sleep hours (0-8 hours post-dose), CBP steady-state levels and AUC are higher than nCBP post TNX-102 SL administration, which optimizes the effects of CBP on the sleeping brain.
- In contrast, a simulation of oral IR CBP 5 mg (Figure 4) and 10 mg (Figure 5) predicts nCBP steady-state levels and AUC higher than CBP during sleep.
- The dynamic changes in CBP over 24 hours are believed to optimize the effects on the brain, and these changes are magnified in the higher predicted occupancy of relevant receptors, 5-HT_{2A}, α_1, H_1 and M_1, during the typical sleep hours.
- In contrast, nCBP has a higher affinity for the norepinephrine (NE) transporter, which would be expected to impair sleep due to higher synaptic NE availability during period in which optimal sleep quality is associated with lower NE activity. Therefore, TNX-102 SL is predicted to be differentiated from oral IR CBP due to lower nCBP levels than CBP during the typical sleep hours.
- Together these data support the use of TNX-102 SL as a potential chronic bedtime treatment for PTSD, FM and AAD.

www.tonixpharma.com

Presented at the American Society of Clinical Psychopharmacology (ASCP) in Scottsdale, AZ May 28-31, 2019; Poster Session I – Poster #I W29, 11:15 AM – 1 PM EDT, Palomino

Exhibit 99.02

Tonix Pharmaceuticals Presented Results from Pharmacokinetic Analyses of TNX-102 SL in a Poster Presentation at the American Society of Clinical Psychopharmacology

NEW YORK, May 30, 2019 (GLOBE NEWSWIRE) -- Tonix Pharmaceuticals Holding Corp. (Nasdaq: TNXP) (Tonix or the Company) a clinical-stage biopharmaceutical company focused on developing small molecules and biologics to treat psychiatric, pain and addiction conditions as well as to improve biodefense, presented a poster at the American Society of Clinical Psychopharmacology (ASCP) 2019 Annual Meeting held May 28-31, 2019, in Scottsdale, Ariz. The poster, titled "Steady-State Pharmacokinetic Properties of a Sublingual Formulation of Cyclobenzaprine (CBP) HCl (TNX-102 SL*): Comparison to Simulations of Oral Immediate Release CBP" includes pharmacokinetic, or PK, analyses of TNX-102 SL. The poster can be found on the Scientific Presentations page of Tonix's website.

The poster presentation reports PK results of TNX-102 SL, a sublingual form of cyclobenzaprine (CBP), studied in a comparative PK, open-label, randomized, parallel, two-arm, multiple-dose bridging study, with the reference listed drug AMRIX® (cyclobenzaprine HCl extended release capsules). TNX-102 SL is being developed as a potential treatment for posttraumatic stress disorder (PTSD), fibromyalgia (FM) and agitation in Alzheimer's disease (AAD), which are central nervous system (CNS) conditions in which sleep disturbances are believed to play essential roles in the illness expression.

Gregory M. Sullivan, M.D., Chief Medical Officer, Tonix Pharmaceuticals Holdings Corp., commented, "We believe this study serves to bridge TNX-102 SL to the safety findings and relevant labeling information of AMRIX, qualifying it for the 505(b)(2) regulatory approval pathway, which is intended to streamline the U.S. Food and Drug Administration (FDA) approval of pharmaceutical products that incorporate already-approved pharmacological agents."

Dr. Sullivan added, "Designing a drug begins with the active ingredient, but formulation is key to improving characteristics such as how much of the administered drug gets to the target organ and how quickly it gets there. For TNX-102 SL the target organ is the brain, and CBP is known to efficiently pass from the blood stream to the brain. We believe that data from this PK study confirms that TNX-102 SL as a sublingual tablet delivers CBP dynamically into the blood stream with reduced formation of nCBP, which are properties that we consider optimized for a sleep quality drug. We believe these data support the use of TNX-102 SL as a potential chronic bedtime treatment for PTSD, FM and AAD."

About Tonix Pharmaceuticals Holding Corp.

Tonix is a clinical-stage biopharmaceutical company focused on discovering and developing pharmaceutical products to treat psychiatric, pain and addiction conditions, and biological products to improve biodefense through potential medical counter-measures. Tonix's lead program is for the development of Tonmya** (TNX-102 SL), which is in Phase 3 development as a bedtime treatment for PTSD. TNX-102 SL for the treatment of PTSD has U.S. Food and Drug Administration (FDA) Breakthrough Therapy designation. Tonix is also developing TNX-102 SL as a bedtime treatment for fibromyalgia and agitation in Alzheimer's disease under separate Investigational New Drug applications (INDs) to support potential pivotal efficacy studies. The fibromyalgia program is in Phase 3 development and the agitation in Alzheimer's program is Phase 2 ready. The agitation in Alzheimer's disease IND has been designated a Fast Track development program by the FDA. TNX-1300*** (T172R/G173Q double-mutant cocaine esterase 200 mg, *i.v.* solution) is being developed under an IND and is in Phase 2 development for the treatment of cocaine intoxication. TNX-1300 (formerly known as RBP-8000) is a recombinant protein enzyme produced through rDNA technology in a non-disease-producing strain of *E. coli* bacteria. TNX-1300 for cocaine intoxication has FDA Breakthrough Therapy designation. TNX-601 (tianeptine oxalate) is in the pre-IND application stage, also for the treatment of PTSD but by a different mechanism from TNX-102 SL and designed for daytime dosing. TNX-601 is also in development for a potential indication - neurocognitive dysfunction associated with corticosteroid use. A Phase 1 clinical formulation selection pharmacokinetic study of TNX-601 will be conducted outside of the U.S. in 2019. TNX-801 (live virus vaccine for percutaneous (scarification) administration) is a potential smallpox-preventing vaccine based on a live synthetic version of horsepox virus, currently in the pre-IND application stage.

** TNX-102 SL (cyclobenzaprine HCl sublingual tablets) is an investigational new drug and has not been approved for any indication.*

***Tonmya has been conditionally accepted by the U.S. Food and Drug Administration (FDA) as the proposed trade name for TNX-102 SL for the treatment of PTSD.*

****TNX-1300* (T172R/G173Q double-mutant cocaine esterase 200 mg, *i.v.* solution) *is an investigational new biologic and has not been approved for any indication.*

This press release and further information about Tonix can be found atwww.tonixpharma.com.

Forward Looking Statements

Certain statements in this press release are forward-looking within the meaning of the Private Securities Litigation Reform Act of 1995. These statements may be identified by the use of forward-looking words such as “anticipate,” “believe,” “forecast,” “estimate,” “expect,” and “intend,” among others. These forward-looking statements are based on Tonix's current expectations and actual results could differ materially. 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 failure to obtain FDA clearances or approvals and noncompliance with FDA regulations; 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 forth in the Annual Report on Form 10-K for the year ended December 31, 2018, as filed with the Securities and Exchange Commission (the “SEC”) on March 18, 2019, 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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