LONGBOARD PHARMACEUTICALS

Corporate Presentation - The Potential of LP352

December 2022

Forward-Looking Statements

This presentation contains forward-looking statements about Longboard Pharmaceuticals, Inc. ("we," "Longboard" or the "Company"), including statements regarding: our future results of operations and financial position; business strategy; the timing, costs, conduct and results of our preclinical studies and clinical trials for our product candidates, such as our expectations regarding our PACIFIC Study and data from our Phase 1 Open-Label PK/PD study; the timing and likelihood of regulatory filings and approvals for our product candidates, such as our pre-IND meeting for LP659; our intellectual property; our ability to obtain regulatory approval and commercialize our product candidates; the potential of LP352, including to limit adverse events associated with currently available non-selective ASMs, make a difference across a range of DEEs, and be a best-in-class ASM, including through BID dosing; and other statements that are not historical facts, including statements that may include words such as "will", "may", "can", "intend", "plan", "expect", "believe", "potential" and similar words.

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Certain information contained in this presentation and statements made orally during this presentation relate to or are based on studies, research, publications, surveys and other data obtained from third-party sources and Longboard's own internal estimates and research. While Longboard believes these third-party studies, research, publications, surveys and other data to be reliable as of the date of this presentation, it has not been independently verified, and makes no representations as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources.

This presentation discusses product candidates that have not yet been approved for marketing by the U.S. Food and Drug Administration.

Longboard's Pipeline of Next Generation GPCR Programs

Program	MOA	Therapeutic Area	Preclinical	Ph I	Ph II	Ph III	Anticipated Milestones
LP352	5-HT2C Superagonist	DEEs and other refractory epilepsies					Ph 1 data at medical mtg. - H1 2022 Ph 1 CSF PK/PD qEEG data - Q4 2022 Ph 1b/2a PACIFIC Study Data - H2 2023
LP659	S1P Receptor Modulator	Multiple neurological diseases					Pre-IND Meeting – Q4 2022

- *We hold rights to other product candidates, including LP143 and nelotanserin, through the Arena License Agreement*
- *We are eligible to receive royalties of 9.5% - 18.5% on sales of lorcaserin if approved for commercialization**

** Through the Royalty Purchase Agreement*

Definitions: DEEs=developmental and epileptic encephalopathies; S1P = sphingosine 1-phosphate; PK=pharmacokinetics; PD=pharmacodynamics; EEG = electroencephalogram

LP352 has the Potential to Make a Difference Across a Range of DEEs

LP352

Greater Selectivity and Specificity

The product of

20 years of world-class GPCR research and optimization

Penetrates the brain in a dose-dependent, consistent and sustained manner

5-HT2 proof-of-concept observed across multiple DEEs and seizures types, however there are safety and dosing considerations with other compounds:

- LP352 is the ***only*** 5-HT2C agonist being **dose optimized** to address this patient population

LP352 demonstrated **predictive efficacy in several pre-clinical seizure models**:

- Multiple zebrafish and rodent models

Demonstrated consistent CNS engagement through:

- Transient prolactin increases
- Sustained qEEG activity

Ph 1 data support potential best-in-class profile:

- SAD/MAD
- CSF/EEG

Enrolling the Ph 1b/2a **PACIFIC study** in patients 12-65 years old with DEE diagnosis

- **No echocardiograms**
- Evaluating broad range of seizure types across DEEs

Strong IP protection through 2041*

*Composition of matter through 2036 with potential for PTE & PTA (2041)
Definitions: DEEs=developmental and epileptic encephalopathies; CSF = cerebrospinal fluid; EEG = electroencephalogram

LONGBOARD PHARMACEUTICALS

The Potential of 5-HT2C Superagonist LP352

A potential best-in-class serotonin receptor agonist anti-seizure product candidate that is designed to be highly selective and being dose-optimized to treat a broad range of DEEs effectively and safely

The Potential of LP352

Greater Selectivity and Specificity

- 5-HT2 agonist designed to only bind to the 5-HT2C receptor*
- 5-HT2 agonist that has no detected activity at receptors associated with significant adverse side effects: 5-HT2B (valvular heart disease and PAH) & 5-HT2A (psychiatric: insomnia, hallucinations, euphoria)

Preclinical Validation

- Reduces seizure activity in model of neuronal hyperexcitability in zebrafish
- Reduced epileptiform activity in fish and rodent models of disinhibition
- Reduced duration and number of epileptiform events in zebrafish model of Dravet Syndrome

Clinical Validation SAD/MAD

- In general, favorable safety and tolerability observed. Adverse events generally consistent with expected effects of serotonergic medications
- No observed food effect
- Potential prolactin biomarker which increased in a dose dependent and transient manner

Clinical Validation CSF/EEG **

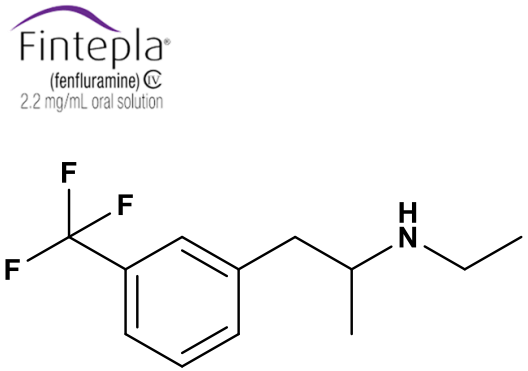
- Favorable safety and tolerability results observed, adverse events generally consistent with previous clinical studies
- Plasma and CSF PK concentration increased in a dose dependent and consistent manner
- Demonstrated effects on qEEG activity within first few dose(s)
- Demonstrated sustained dose-dependent effects on qEEG activity after continuous dosing, thus indicating receptor engagement

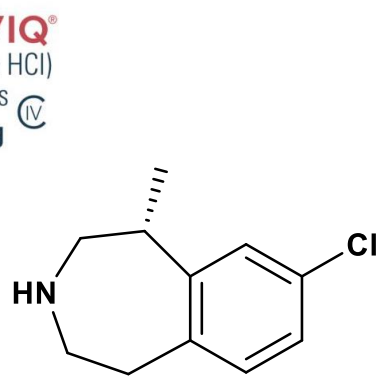
*Radioligand binding assays assessing >150 targets showed significant affinity only to 5-HT2C receptors
** Based on first two cohorts
Definitions: PAH = pulmonary arterial hypertension

5-HT2 Evolution in Rare Epilepsies

Weight Loss Drugs Repurposed

Compound:	**FINTEPLA® DS** (fenfluramine, ZX008)	**Lorcaserin** (Marketed as BELVIQ)
History:	• Approved for weight loss in 1973, became popular in 1990s in **Fen-Phen** (never approved in combo) • Withdrawn due to significant **cardiac toxicity (1997)** • **Repurposed for DEEs at lower dose**	• Designed to avoid the cardiac effects seen with fenfluramine • Approved for weight loss in 2012 • No significant difference in major adverse cardiovascular outcomes versus placebo[1] • Withdrawn from market 2020; increased occurrence of **cancer in safety clinical trial**
Current Status:	• Approved for the treatment of seizures with Dravet & LGS (REMS required with echocardiograms), Ph 3 CDD	• FDA Expanded Access Program in Dravet, Ph 3 Dravet



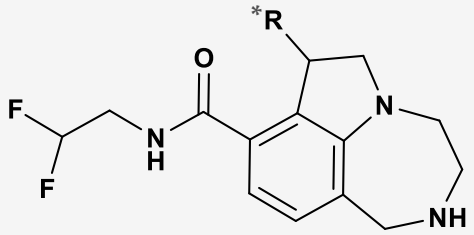


Designed & Being Dose-Optimized for DEEs

LP352

- Designed to be a next-generation selective 5-HT2C superagonist
- Dose optimization for DEEs
- BID formulation work ongoing, expected for Ph 3
- **No echocardiograms** in PACIFIC study

- Ph 1b/2a clinical trial in multiple DEEs



*R=undisclosed
1 = CAMELLIA-TIMI 61

LP352 Designed to be a Next Generation 5-HT2C with Greater Selectivity and Specificity

	Serotonin Receptor Subtype	EC_{50}, nM	Ki, nM	Selectivity 5-HT2C vs 5-HT2B	Selectivity 5-HT2C vs 5-HT2A	Potential Adverse Events Per Receptor Subtype
LP352 5-HT2C Superagonist	**5-HT2C**	**~120**	**~50**	**>200x**	**>200x**	CNS, GI
	5-HT2B	>10,000	>10,000			n/a
	5-HT2A	>10,000	>10,000			n/a
Nordexfenfluramine (an active metabolite of fenfluramine) [1]	5-HT2C	72.4	10.4	0.94x	11.5x	CNS, GI
	5-HT2B	25.7	9.8			Cardiac, Pulmonary
	5-HT2A	1778	120.2			Psychiatric
Lorcaserin[2]	5-HT2C	39	13	11.3x	7.1x	CNS, GI
	5-HT2B	2380	147			n/a
	5-HT2A	553	92			Psychiatric

LP352 selectivity may limit off-target effects associated with currently available non-selective ASMs

1 Third party study previously commissioned by Arena, 2 BELVIQ FDA approved prescribing information 06/2012; Note: The above table is for illustrative purposes only and is not a head-to-head comparison. Differences exist between in vitro study designs and methodologies, and caution should be exercised when comparing data across studies
Definitions: CNS= Central nervous system ; GI = Gastrointestinal; ASM = anti-seizure medication

LP352 Inhibited Seizure Activity in Multiple Preclinical Models

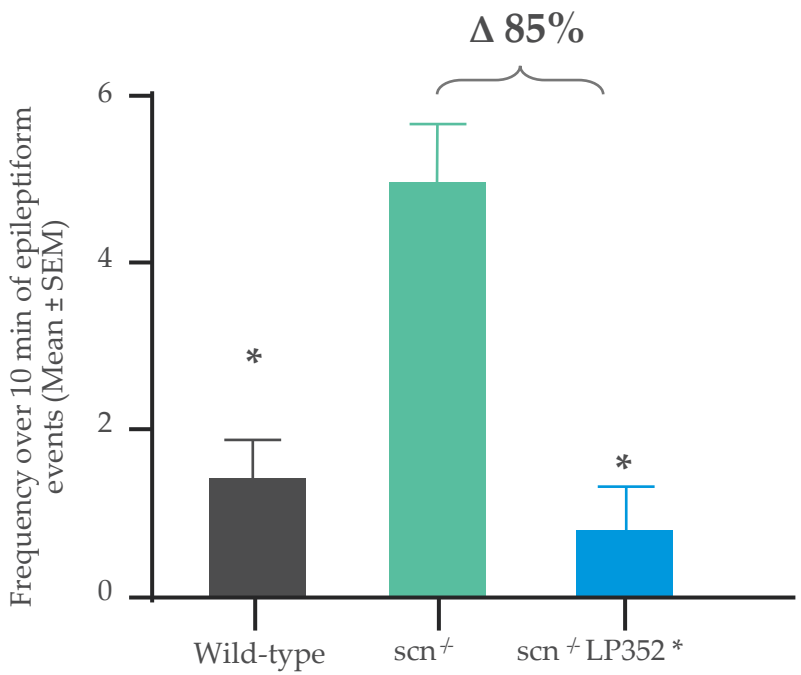
	Corneal Kindling	Pentylenetetrazol (PTZ) (i.v.)	Scn1a$^{A1783V/WT}$ Transgene	*scn1lab* Transgene	Ethyl ketopentenoate (EKP)	Kainic acid (KA)
Model	Partial (focal) limbic seizures	Acute seizure	Genetic model of Dravet Syndrome	Genetic model of Dravet Syndrome	Generalized seizure	Acute and chronic seizure
Species	mouse	mouse	mouse	zebrafish	zebrafish	zebrafish
Activity	-	+	-	+	+	+

Potential ASMs are assayed in multiple relevant preclinical models based on the compound's MOA. Models are conducted utilizing wide range panels that typically produce a mix of positive and negative results. The above are a subset of preclinical assays conducted with LP352. Preclinical models are not necessarily predictive of clinical efficacy or regulatory approval.

Definitions: ASM = anti-seizure medication; MOA = mechanism of action

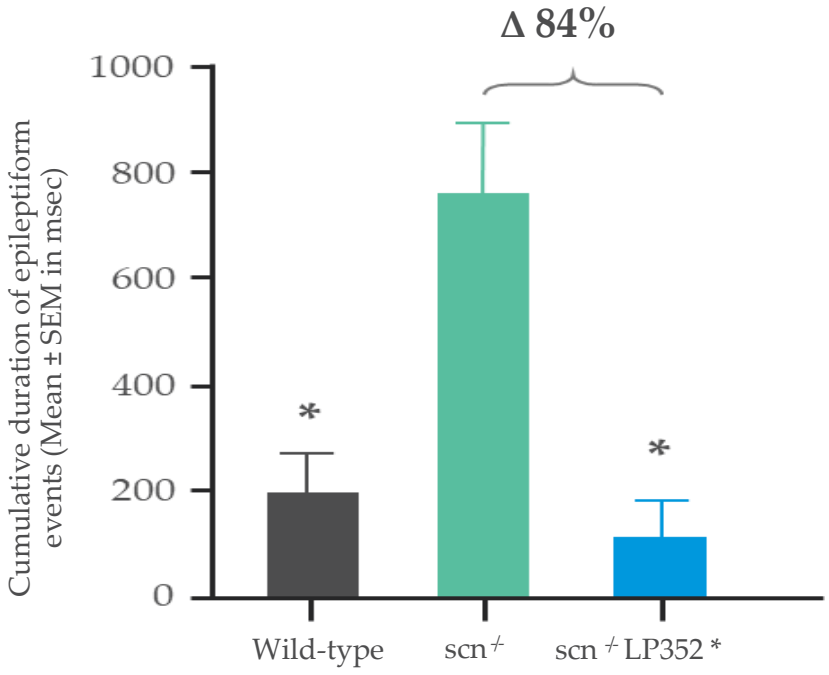
LP352 Significantly Reduced Epileptiform Frequency & Duration in the Zebrafish *scn1lab* Model of Dravet Syndrome

FREQUENCY OF EPILEPTIFORM EVENTS



LP352 demonstrated 85% reduction of epileptiform events

DURATION OF EPILEPTIFORM ACTIVITY



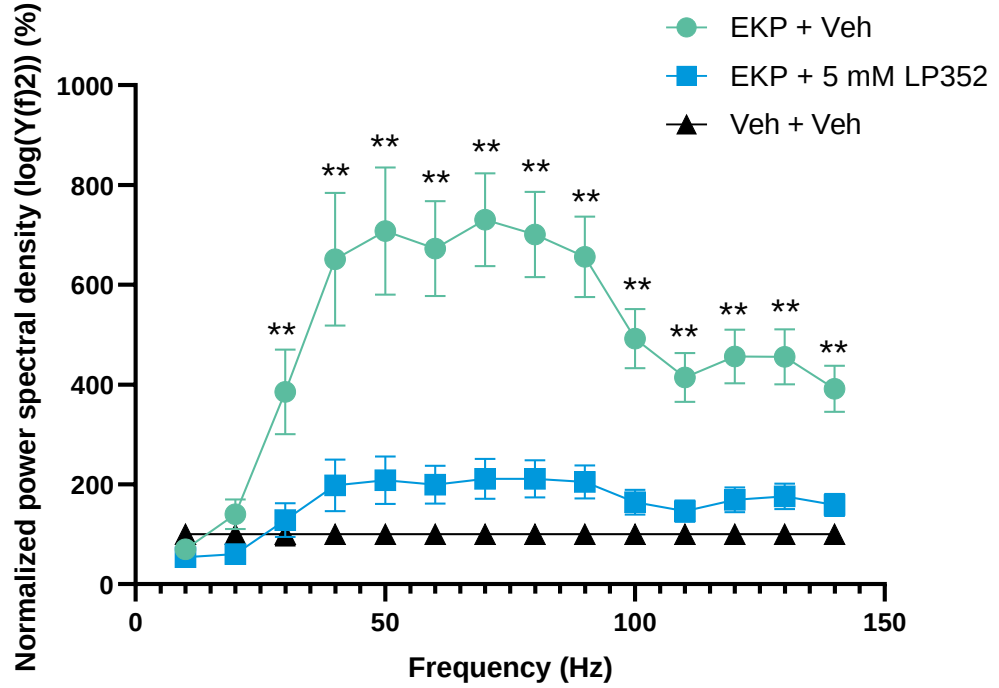
LP352 demonstrated 84% reduction of epileptiform duration

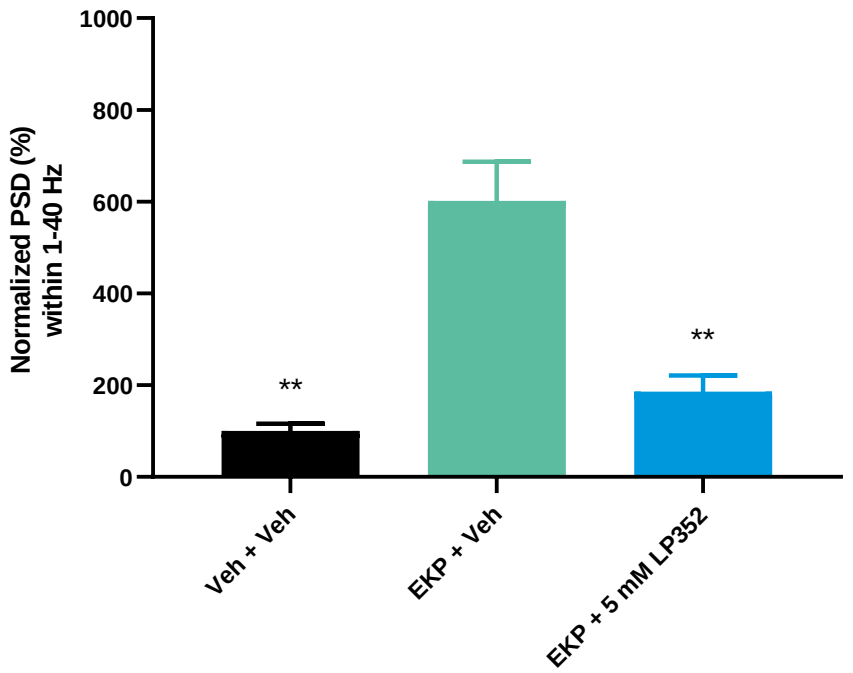
*LP352 30mM

N=17-23/group, *p<0.001

LP352 Significantly Improved Seizure Activity in the Zebrafish EKP Epilepsy Model

Epileptiform brain activity (LFPs) in the EKP model





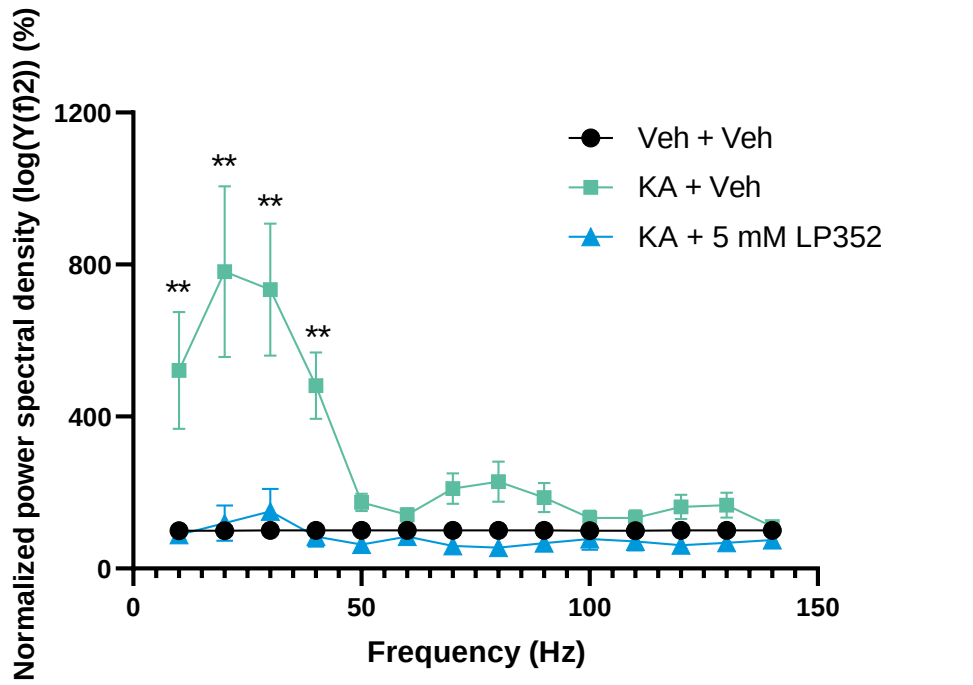
LP352 demonstrated ~69% reduction in seizure activity

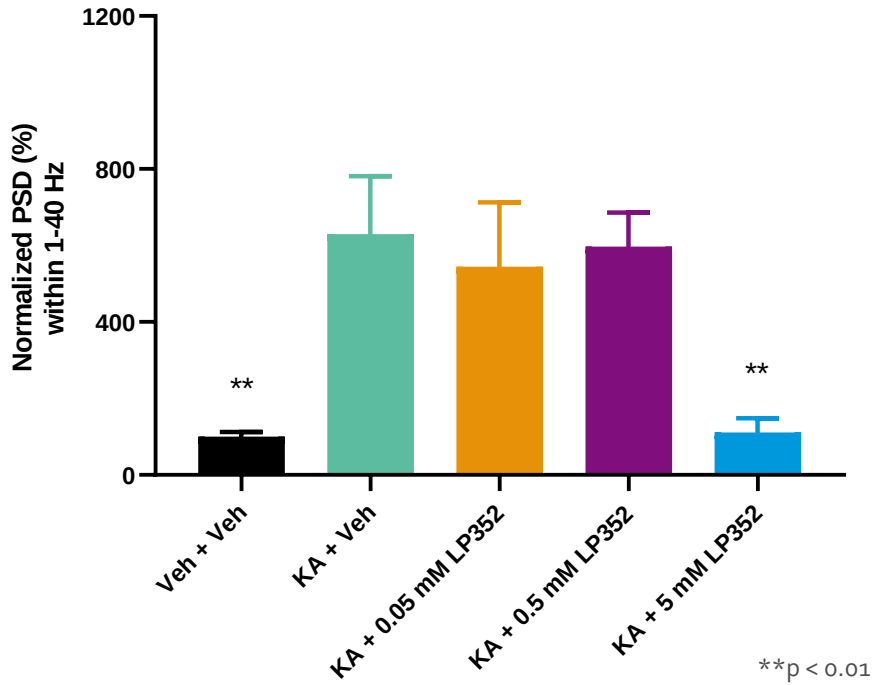
Definitions: LFP = local field potential; EKP= Ethyl ketopentenoate; Veh = Vehicle

N=18-20/group, **p < 0.0001

LP352 Significantly Reduced Seizure Activity in the Zebrafish Kainic Acid Epilepsy Model

LP352 Normalized Power Spectral Density





LP352 demonstrated ~82% reduction of seizure activity

Definitions: KA= Kainic Acid; Veh = Vehicle

N=12-14/group

LP352 Demonstrated Dose-Dependent Improvement in Time to Clonic Seizures in PTZ Mouse Model

Effect of LP352 on the Threshold for Seizures Induced by the Timed Intravenous Infusion of PTZ in Male Mice

Compound	Animal Weight (Grams, Mean ± S.E.M.)	Time of Test	PTZ Dose (mg/kg, Mean ± S.E.M)	
			First Twitch	Clonus
Vehicle Control	32.7 ± 0.7	0.5 hr	25.1 ± 1.5	26.4 ± 1.6
LP352 3mg/kg	31.4 ± 0.4	0.5 hr	26.5 ± 0.8	30.3 ± 1.1
LP352 10mg/kg	31.8 ± 0.4	0.5 hr	28.7 ± 0.7	32.3 ± 1.2**

**$p < 0.01$

LP352 Ph 1 Multiple Ascending Dose (MAD) Results

Favorable Safety & Tolerability Results Observed

Treatment-Emergent Adverse Events by Preferred Term Occurring in ≥ 2 Subjects in Any Treatment Group – MAD (Safety Set)

Preferred Term n(%) E	LP352 (TID) Cohort 1 3 mg (N=6)	LP352 (TID) Cohort 2 6 mg (N=6)	LP352 (TID) Cohort 3 12 mg (N=7)	LP352 (TID) Cohort 4 18 mg (N=6)	Pooled Placebo (N=8)
Subjects with at least 1 TEAE	5 (83.3) 9	6 (100) 29	6 (85.7) 39	6 (100) 55	4 (50.0) 8
Headache	2 (33.3) 2	2 (33.3) 4	2 (28.6) 5	4 (66.7) 5	1 (12.5) 1
Somnolence	1 (16.7) 1	1 (16.7) 1	4 (57.1) 4	3 (50.0) 5	0
Dizziness	0	3 (50.0) 3	2 (28.6) 3	2 (33.3) 2	0
Micturition Urgency	1 (16.7) 1	0	1 (14.3) 1	5 (83.3) 5	0
Dizziness Postural	0	0	1 (14.3) 1	5 (83.3) 5	0
Diarrhoea	1 (16.7) 1	4 (66.7) 4	1 (14.3) 1	0	0
Orthostatic Hypotension	0	0	2 (28.6) 3	4 (66.7) 4	0
Constipation	1 (16.7) 1	1 (16.7) 1	2 (28.6) 3	1 (16.7) 1	1 (12.5) 1
Nausea	1 (16.7) 1	0	1 (14.3) 1	2 (33.3) 2	1 (12.5) 1
Paraesthesia	0	1 (16.7) 1	2 (28.6) 3	1 (16.7) 1	0
Chills	0	0	1 (14.3) 1	3 (50.0) 5	0
Anxiety	0	2 (33.3) 2	0	2 (33.3) 2	0
Orthostatic HR Response Increased	0	0	0	3 (50.0) 5	1 (12.5) 1
Dysmenorrhoea	1 (16.7) 1	0	0	2 (33.3) 2	1 (12.5) 1
Fatigue	0	2 (33.3) 2	0	0	0
Vessel Puncture Site Bruise	0	0	0	2 (33.3) 2	0
Hypotension	0	2 (33.3) 2	0	0	0

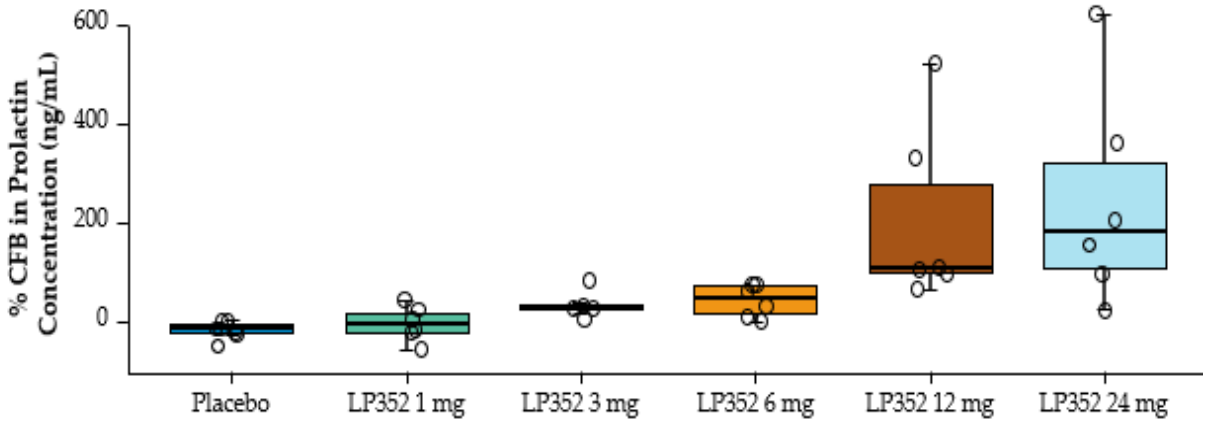
- Majority of AEs were mild to moderate (most common was headache)
- AEs generally consistent with expected CNS effects and expected effects of serotonergic drugs
- At the maximum planned dose, a single SAE of anxiety was reported two days after last dose of study drug and subsequently resolved

LP352 Ph 1 Single Ascending Dose (SAD) Results

Favorable Pharmacokinetics and Pharmacodynamics Results Observed

Single Ascending Dose & Single-Dose Food Effect (N=40)

Percent Change from Baseline in 2-Hour Prolactin Concentration Across All Dose Groups Under Fasted Conditions



LP352 Demonstrated No Meaningful Food Effect

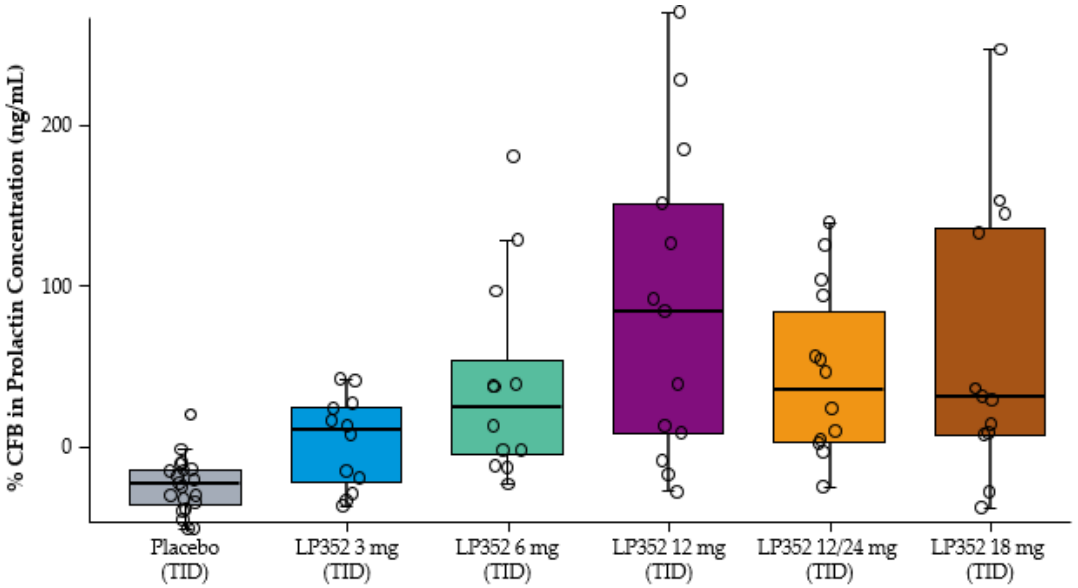
Parameter (Unit)	Geometric Mean (95% CI)			
	n	Fed	n	Fasted
C_{max} (ng/mL)	6	8.8 [5.1, 15.3]	6	8.5 [4.9, 14.6]
$AUC_{0\text{-last}}$ (h*ng/mL)	6	61.9 [33.4, 114.7]	6	49.2 [26.6, 91.2]
$AUC_{0\text{-inf}}$ (h*ng/mL)	6	63.6 [34.7, 116.6]	6	51.0 [27.8, 93.4]

LP352 Ph 1 MAD Results

Favorable Pharmacokinetics and Pharmacodynamics Results Observed

Multiple Ascending Dose & Dose Titration (N=43)

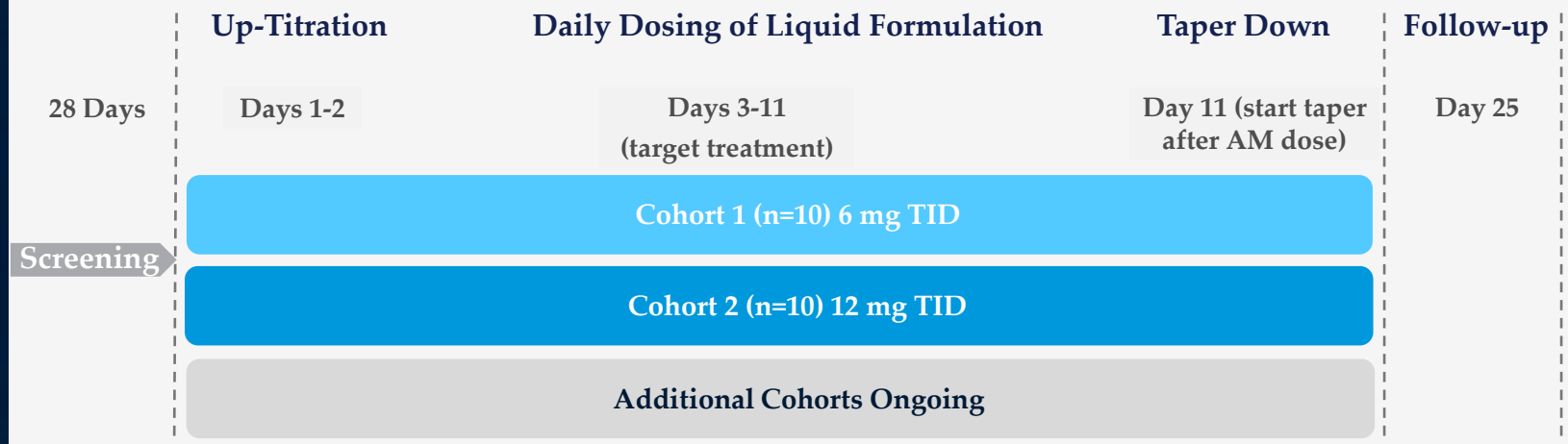
Pharmacodynamics: Boxplot of Percent Change From Baseline in Prolactin Concentration with Dose on Day 1 at 2 Hrs - MAD and Dose Titration (Safety Population)



Key Summary of LP352 Pharmacokinetic Parameters by Cohort (Day 14) - MAD and Dose Titration (PK Analysis Population)

Parameter (Unit)	MAD 12 mg (N = 7)
C_{max} (ng/mL) (Mean)	44.9
T_{max} (h) (Median)	1.3
$AUC_{0\text{-}inf}$ (h*ng/mL) (Mean)	330
$T_{1/2}$ (h) (Mean)	6.0

LP352 102: A Phase 1 PK/PD CNS Study in Adult Healthy Volunteers



A Phase 1, Open-label Study to Assess Central Nervous System Pharmacokinetics (PK) and Pharmacodynamics (PD) of Orally Administered LP352

Key Study Objectives:

- Characterize the plasma and CSF PK
- Characterize the safety and tolerability of the doses with titration and taper
- Assess the PK-PD relationships between plasma and CSF exposure and PD endpoints of safety and efficacy, including qEEG endpoints as indicators of CNS target engagement

Plasma:

- Samples Days 1-11 (and taper)
- PK parameters: Cmax, Tmax and AUCtau

CSF:

- Samples Day 11
- PK parameters: Cmax, Tmax and AUCtau

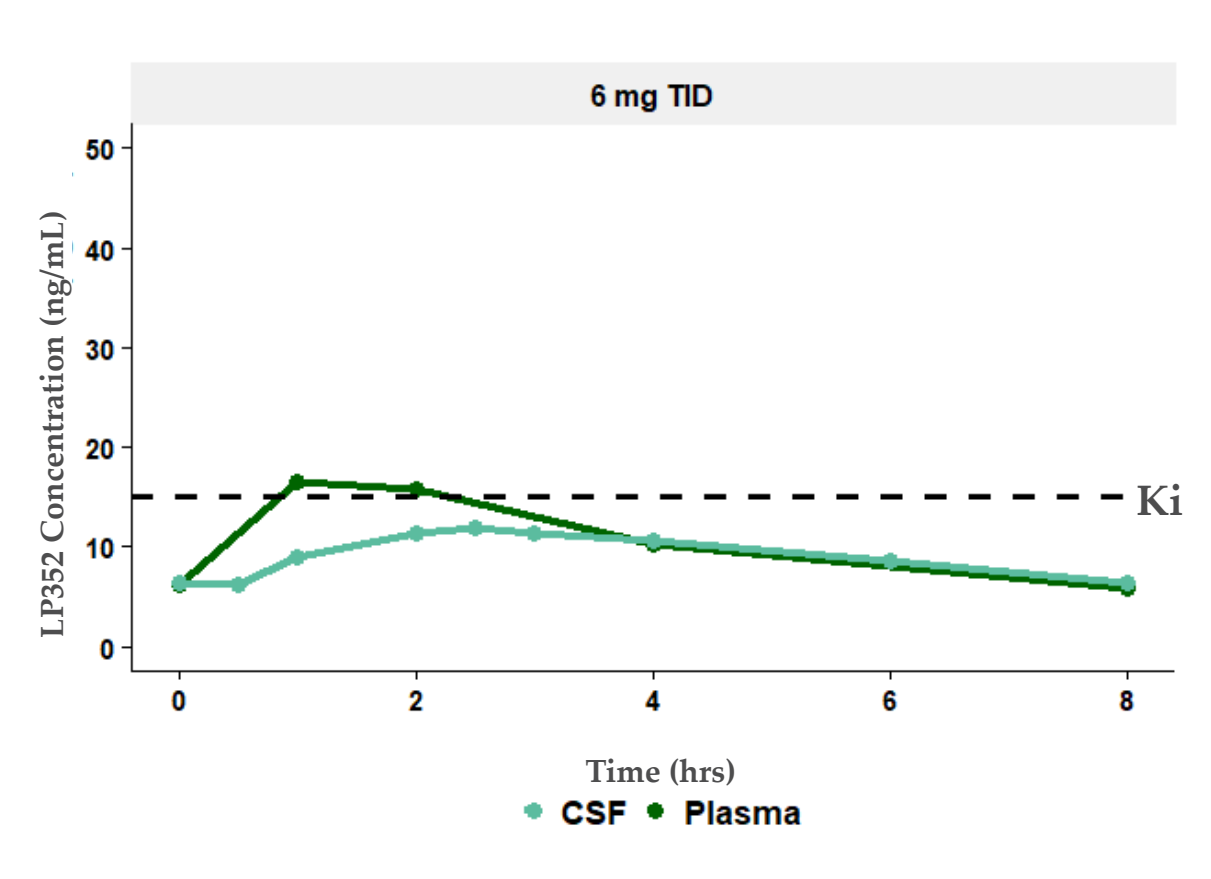
EEG:

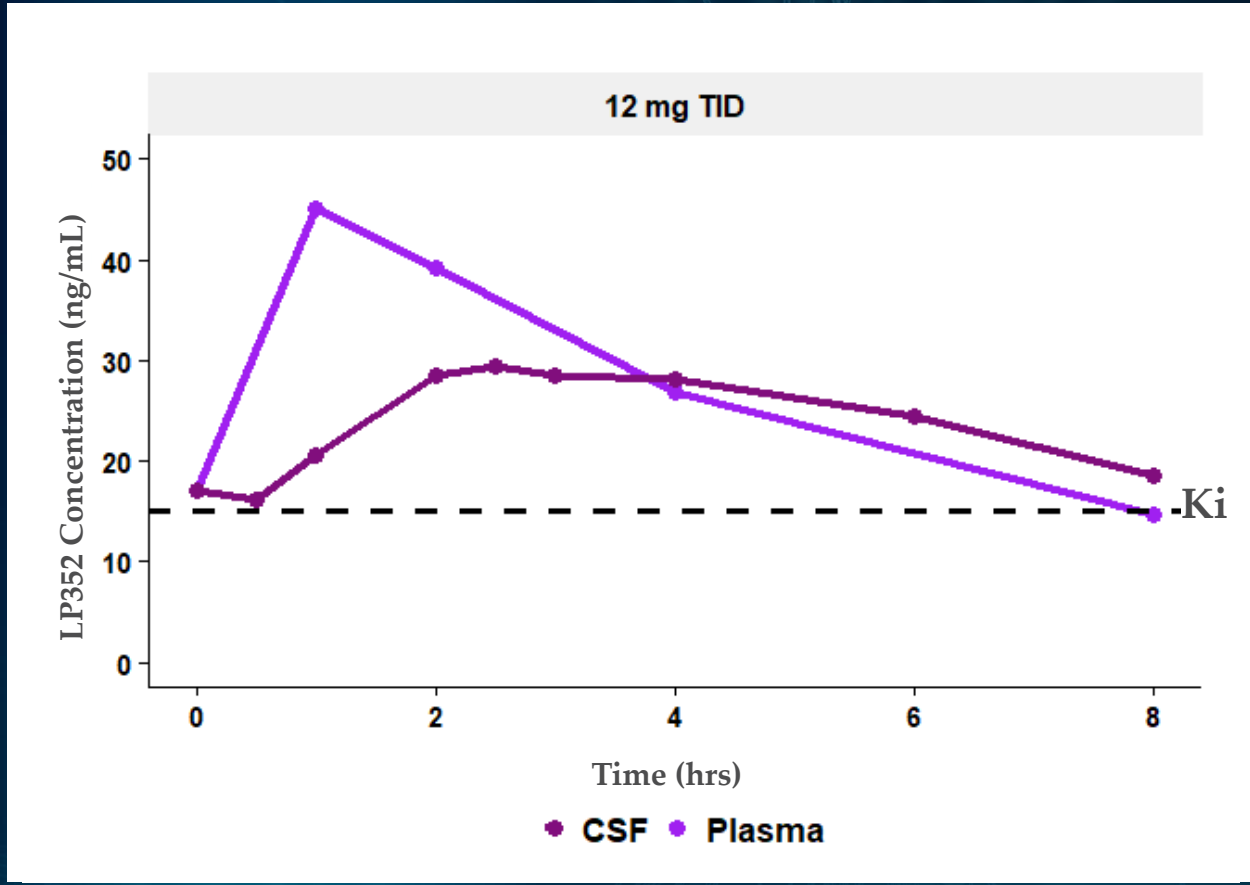
- Serial EEGs Days -1, 1, 3 & 10 (trough Day 16)
- EEG parameters: Five-minute resting EEG with eyes closed and five-minute resting EEG with eyes open performed with the participant seated comfortably in a sound-attenuated room
- Resting EEG evaluated by spectral and coherence analysis, including spectral amplitudes and coherences in clinical frequency bands

Topline data from the 102 study are presented in this deck

Steady State Plasma & CSF Concentrations for LP352 (6mg & 12mg)

12 mg TID Exceeded Ki Value for 5-HT2C Activity throughout Dosing Interval*





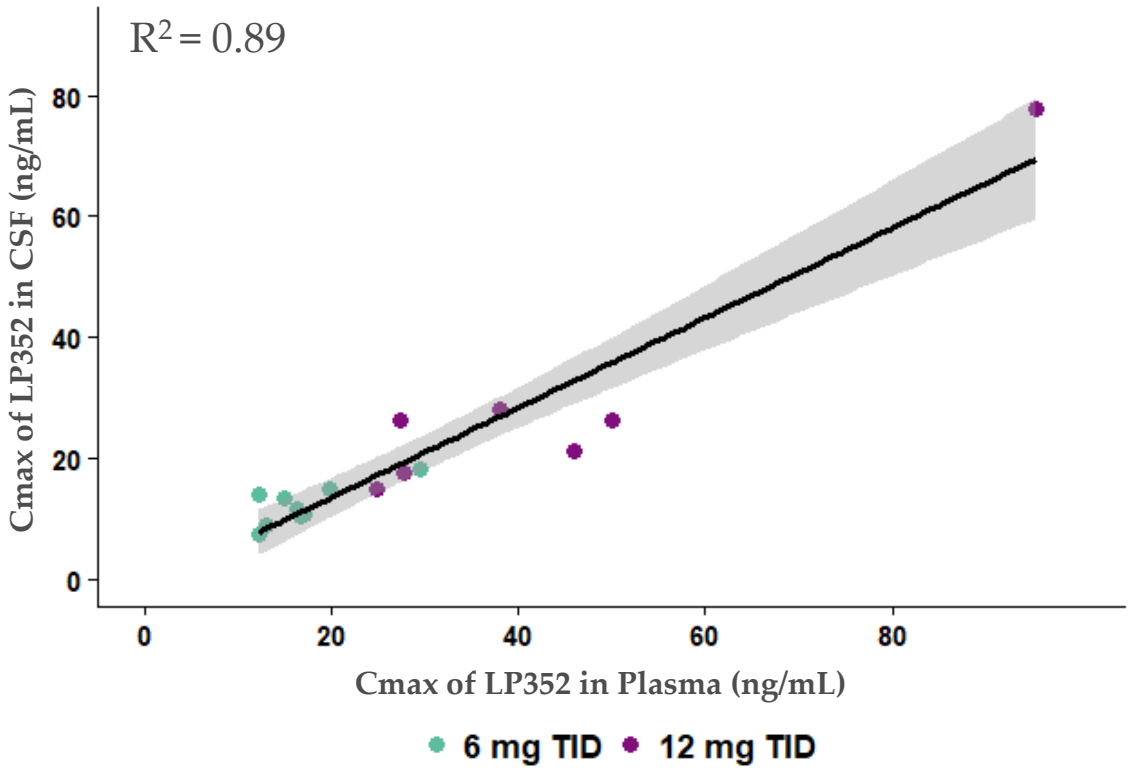
The vast majority of participants in the 12 mg TID cohort achieved plasma and CSF levels above the relevant Ki throughout the dosing period.

*Topline data from 102 study

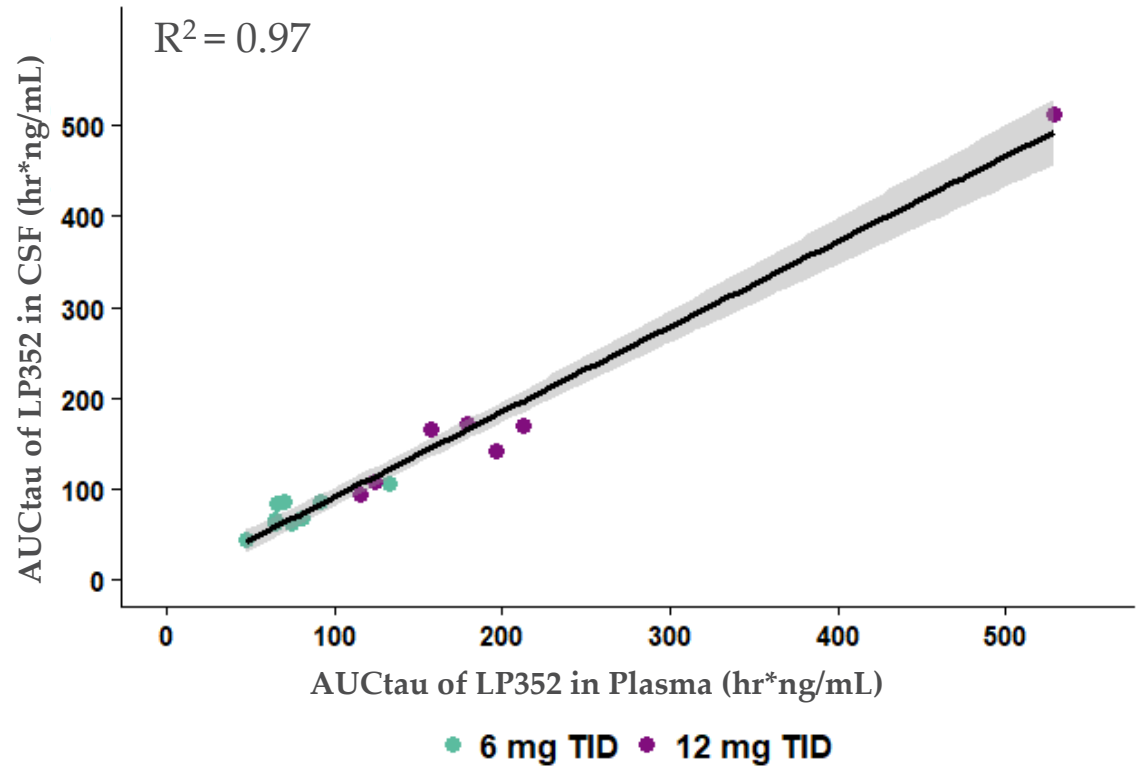
Cmax and AUC CSF vs. Plasma Correlations

Strong Correlation Between Plasma and CSF PK Parameters*

CSF vs. LP352 Cmax



CSF vs. LP352 AUCtau

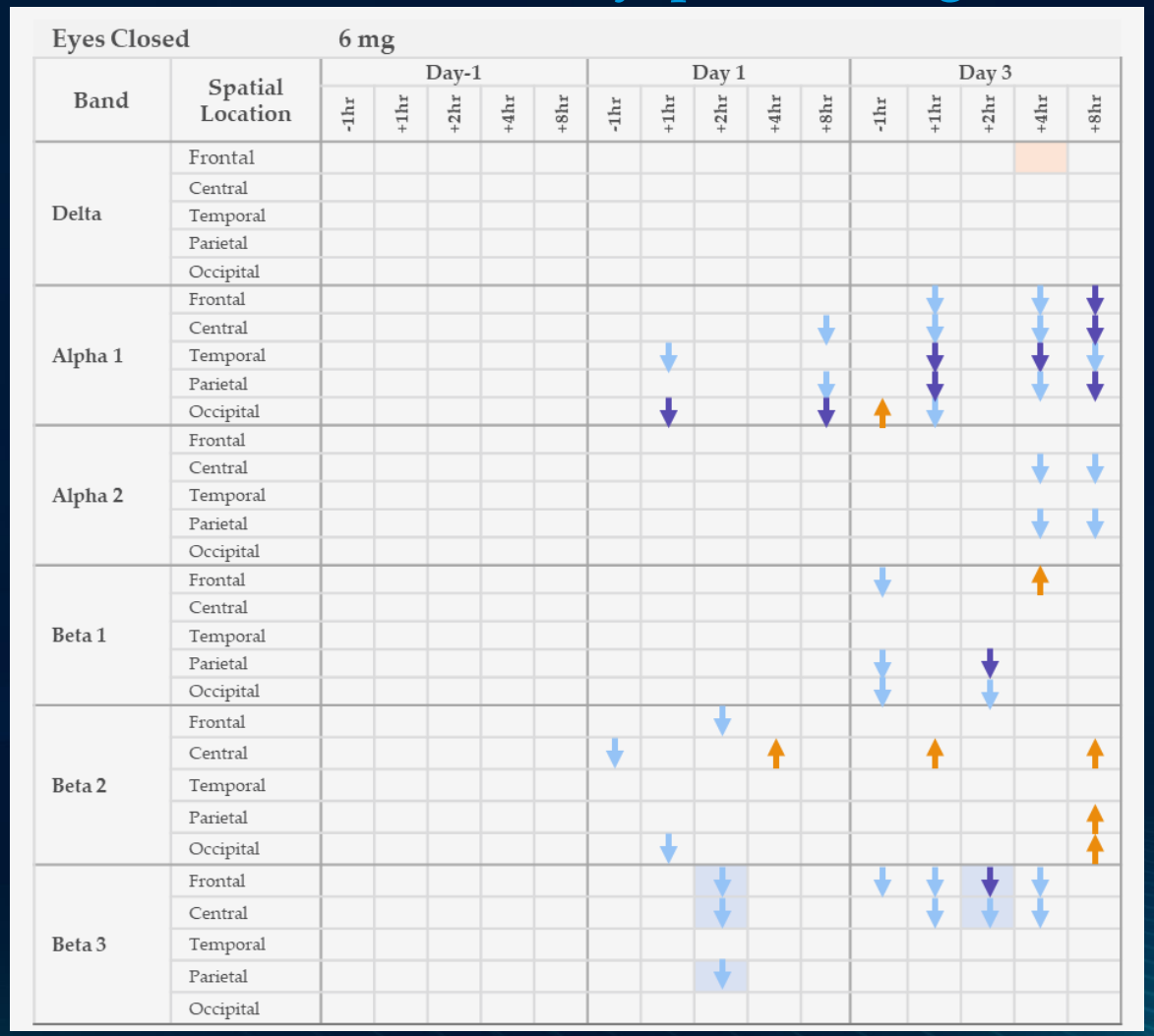


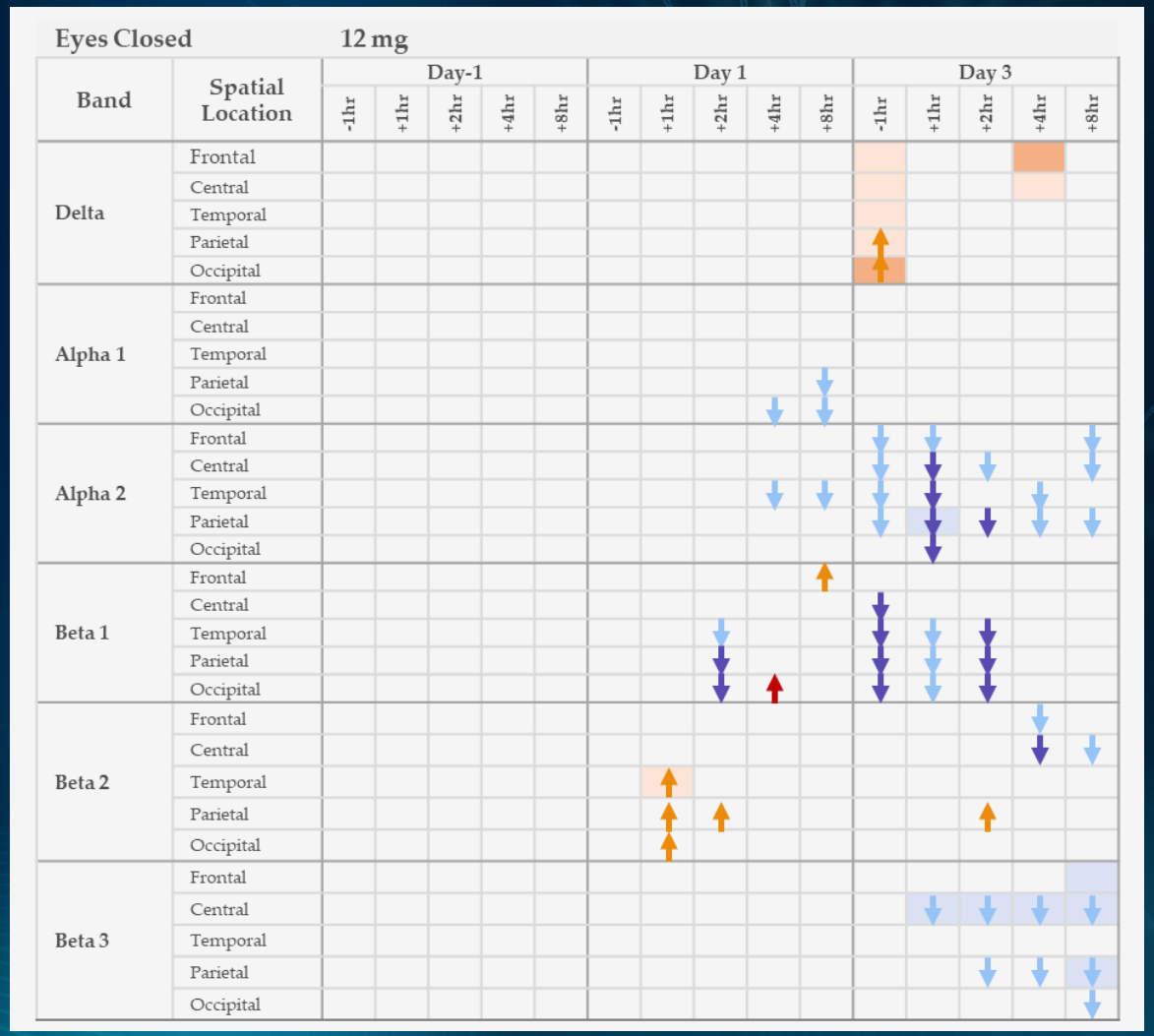
*Topline data from 102 study

5-Minute Resting qEEG Spectral Amplitudes in Clinical Frequency Bands

(Days -1,1 and 3)

LP352 Demonstrated Early qEEG Changes*





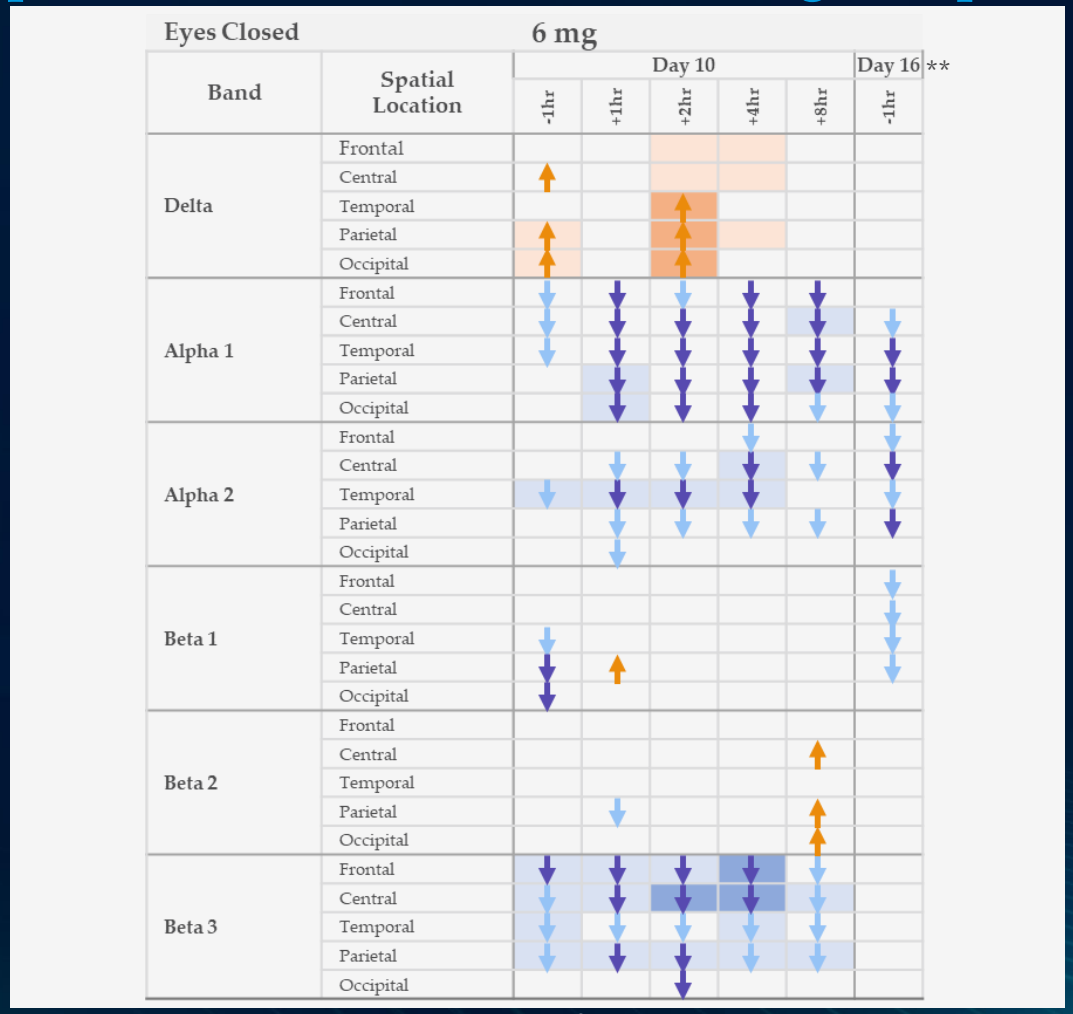
*Topline data from 102 study

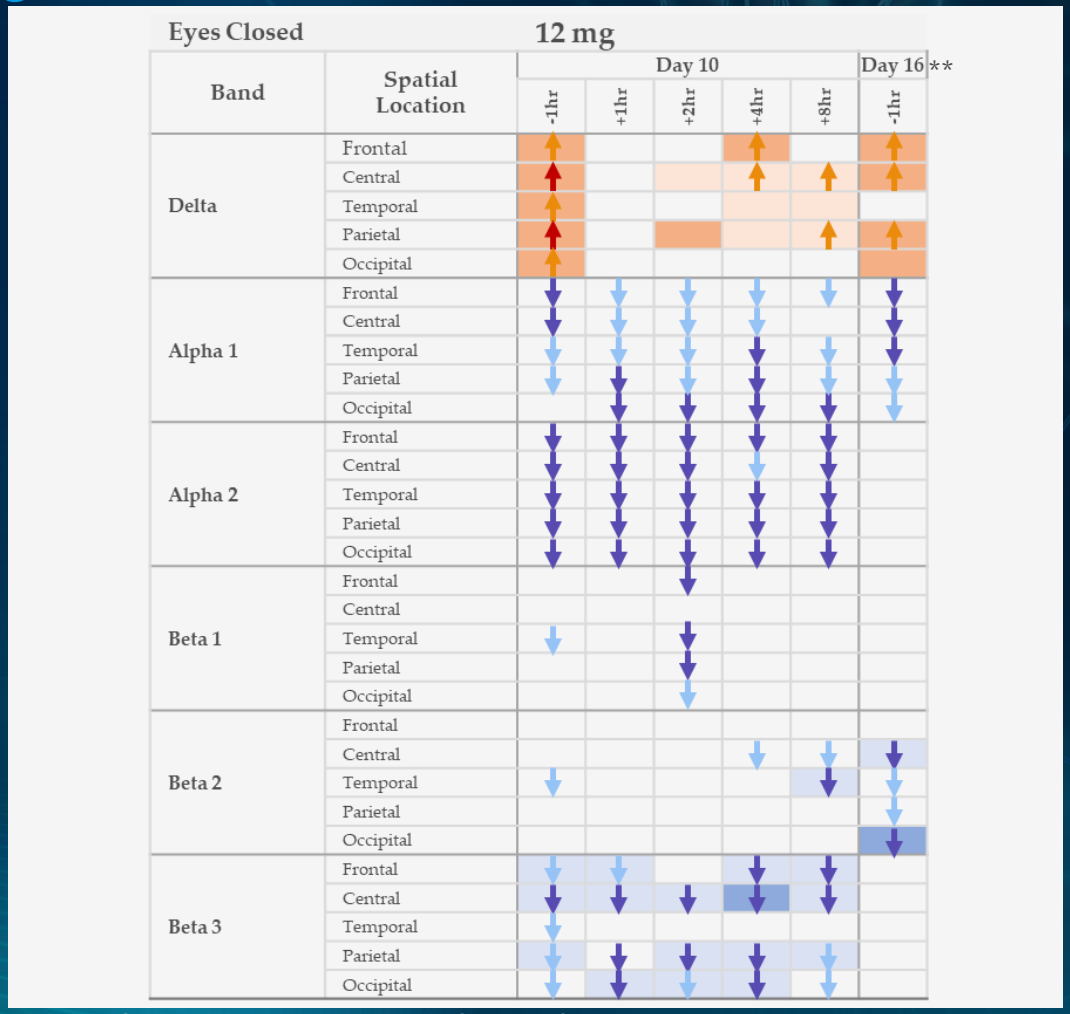
Small and large salient contrasts (≥10%, ≥15%) are indicated by light and heavy arrows (blue=decrease, red=increase) respectively. Small and large salient Cohen's d values (≥0.5, ≥0.8) are indicated by light and dark shading (blue=decrease, orange=increase) respectively.

5-Minute Resting qEEG Spectral Amplitudes in Clinical Frequency Bands

(Days 10 and 16)

LP352 Demonstrated Sustained Effects on qEEG Activity After Continuous Dosing in a Dose-Dependent Manner, Thus Indicating Receptor Engagement*





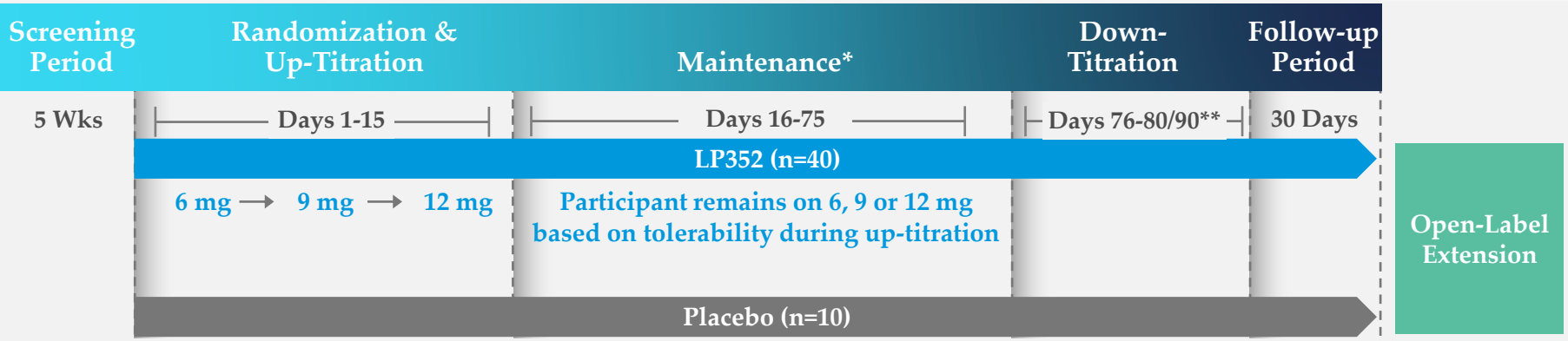
*Topline data from 102 study ** On Day 16, all participants in Cohort 1 and 2 receive one final dose which was less than the full dose of 6 mg and 12 mg, respectively. Small and large salient contrasts (≥10%, ≥15%) are indicated by light and heavy arrows (blue=decrease, red=increase) respectively. Small and large salient Cohen's d values (≥0.5, ≥0.8) are indicated by light and dark shading (blue=decrease, orange=increase) respectively

LP352-102 Phase 1 Study: Key Takeaways To Date

- First known study of its kind for a 5HT2 agonist (e.g. fenfluramine or lorcaserin)
- Favorable safety and tolerability results observed, with AEs generally consistent with previous clinical studies
- Plasma and CSF PK concentration increased in a dose-dependent and consistent manner
- Demonstrated effects on qEEG activity within first few dose(s)
- Demonstrated sustained dose-dependent effects on qEEG activity after continuous dosing, thus indicating receptor engagement
- In summary, we believe the data suggest that LP352 engaged neurotransmitter systems and altered the EEG spectrum

LP352 Ph 1b/2a PACIFIC Study in Patients with DEEs

PACIFIC
Developmental and Epileptic Encephalopathies Study



A double-blind, placebo-controlled study to assess the safety, tolerability, pharmacokinetics of LP352 and

Key Efficacy Signals:

- **EVALUATE reduction in seizures across a broad group of epilepsies**
- **IDENTIFY potential indications for pivotal studies**
- **ANALYZE concentration response to understand dosing in different seizure types and disorders**

Basic Information:

Sites: ~30 sites

Ages: ≥ 12 to ≤65 yrs old

Key Inclusion Criteria:

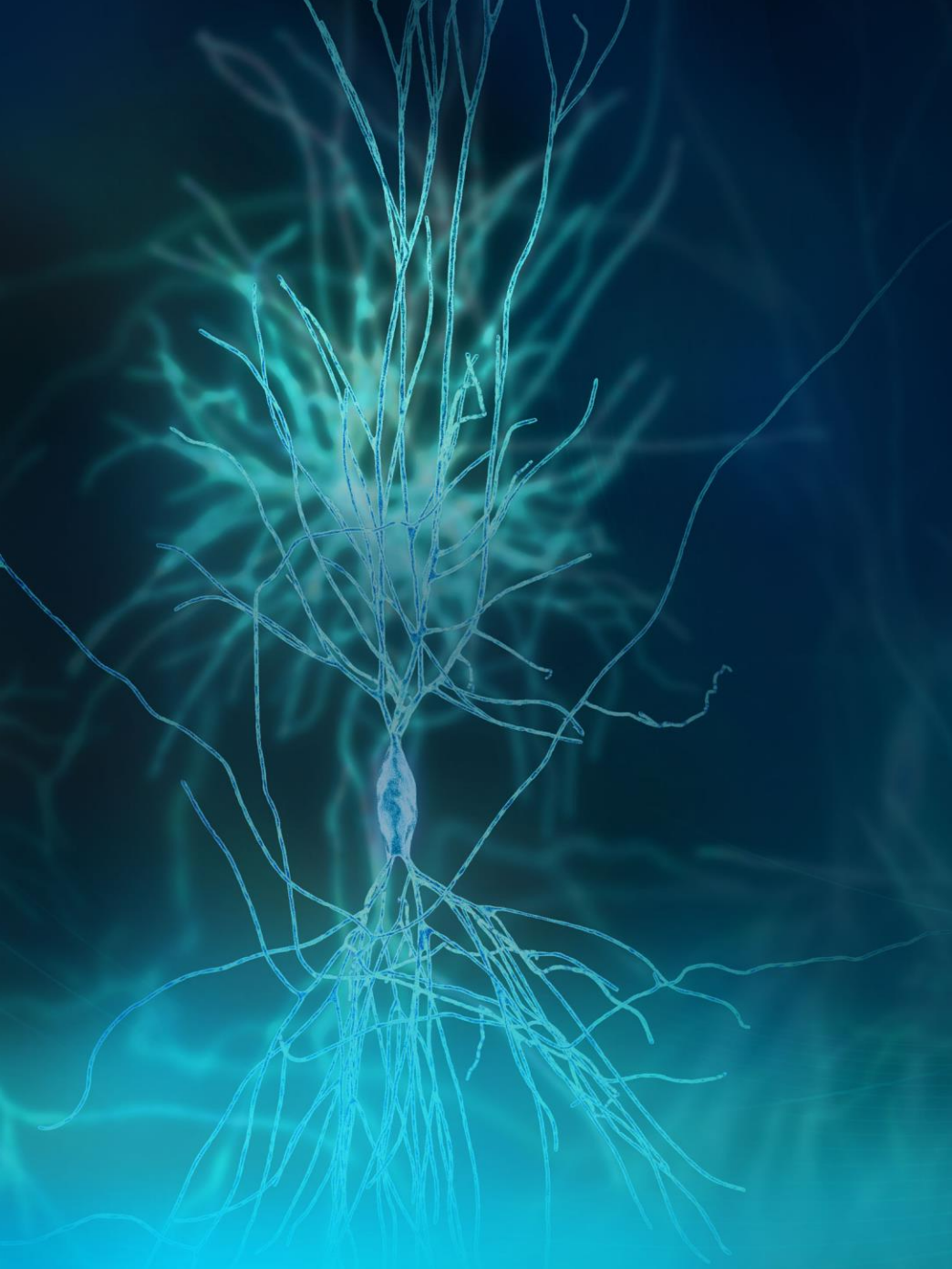
- Developmental and epileptic encephalopathies (DEEs) with ≥ 4 motor seizures per month in 3 mos. prior to screening and ≥ 4 motor seizures in the month of screening
- DEEs (multiple syndromes) may include Dravet syndrome, Lennox-Gastaut syndrome, Tuberous Sclerosis complex, CDKL5 deficiency disorder, SCN2A-related disorders, among others

Key Exclusion Criteria:

- Use of fenfluramine & lorcaserin

PACIFIC Study Initiated March 2022; Data Expected 2H 2023

* Maintenance Dose of LP352 (TID): 6 mg, 9 mg, 12 mg or placebo TID
** Up to a 15-day down-titration/taper period (reducing dose every 5 days) depending on the last maintenance dose



Thank you!

Nasdaq: LBPH