



High-Impact Neuromedicines for Real-World Care

R&D Day
October 27, 2026



| Heather Turner Chief Executive Officer

| Disclaimer

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These and other risks are described more fully in our filings with the Securities and Exchange Commission (“SEC”), including the “Risk Factors” section in the Company’s Form 10-Q for the quarter ended June 30, 2026, filed with the SEC on August 11, 2026. All forward-looking statements contained in this presentation speak only as of the date on which they were made. Except to the extent required by law, we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

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| Our Mission

To boldly advance
high-impact therapies
that help people with
brain disorders live life
more fully.

| Today's Speakers



Heather Turner
Chief Executive Officer



Susan G. Kozauer, MD
Chief Medical Officer



Gad Soffer
Chief Operating and Business Officer



Kaya Pai Panandiker
Chief Commercial Officer



Roger McIntyre, MD
Prof. of Psychiatry and Prof.
of Pharmacology
& Toxicology Univ. of Toronto



Joe Miller
Chief Financial Officer



| Today's Focus



- LB Pharma and the LB-102 Opportunity
- LB-102 Clinical Data in Schizophrenia and Observations we are Exploring
- New Preclinical Data Supporting Potential Differentiation
- Planning for Potential Commercialization in Schizophrenia
- Looking Ahead to Pivotal Data and Summary
- Q&A

LB Today: Late-Stage Development Pipeline with Multiple Clinical Milestones and Runway Expected Beyond 2Q 2029

Psychosis Related Disorders

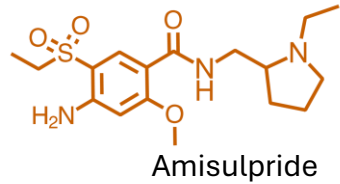
Indication	Product	Route of Administration	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones
Schizophrenia	LB-102	Oral	<div><div></div></div>				Topline data expected 1H 2027 Pre-NDA meeting expected 2H 2027
	LB-102	Long acting injectable	<div><div></div></div>				LAI Formulation development in 2026

Mood Disorders

Indication	Product	Route of Administration	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones
Bipolar 1 Depression	LB-102	Oral	<div><div></div></div>				Topline data expected 1Q 2028
Adjunctive MDD	LB-102	Oral	<div><div></div></div>				Topline data expected 1H 2029

\$327.8 million¹ as of June 30, 2026, exclusive of recent \$150 million private placement²

LB-102 is a Structural Modification of Amisulpride, a Generic AP Available Ex-US



Advantages

- Selectively inhibits D2, D3, and 5HT7 receptors with few off-target effects¹
- Among the highest effect sizes (0.73) compared with approved APs²
- Favorable tolerability profile with one of the lowest all-cause discontinuation rates^{2,3}

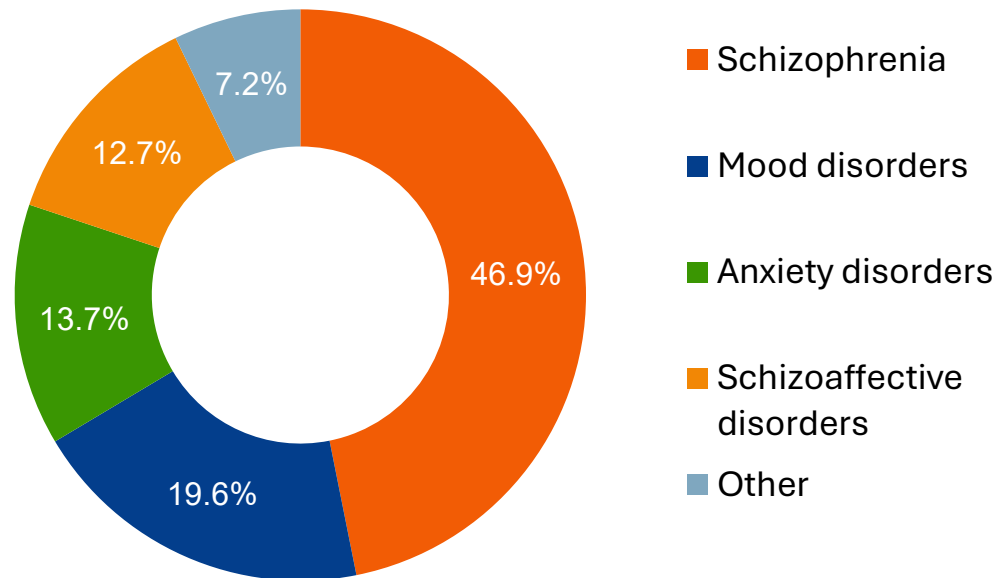
Limitations

- Poor blood-brain barrier (BBB) penetration
- Requires high doses for schizophrenia (400-800 mg) increasing systemic exposure
- Twice daily (BID) dosing creates compliance challenges

| Amisulpride is Widely Prescribed, Highlighting its Established Clinical Utility

2023 EUROPEAN PRESCRIPTIONS BY DIAGNOSIS¹

~2M MONTHLY PRESCRIPTIONS
across 16 European countries



Approved ex-U.S.² for:

- **Schizophrenia**
- **Negative symptoms of schizophrenia**
- **Dysthymia (persistent depressive disorder)**

| LB-102 Was Purpose-Built to Address Amisulpride's Limitations

Designed to improve BBB penetration of amisulpride

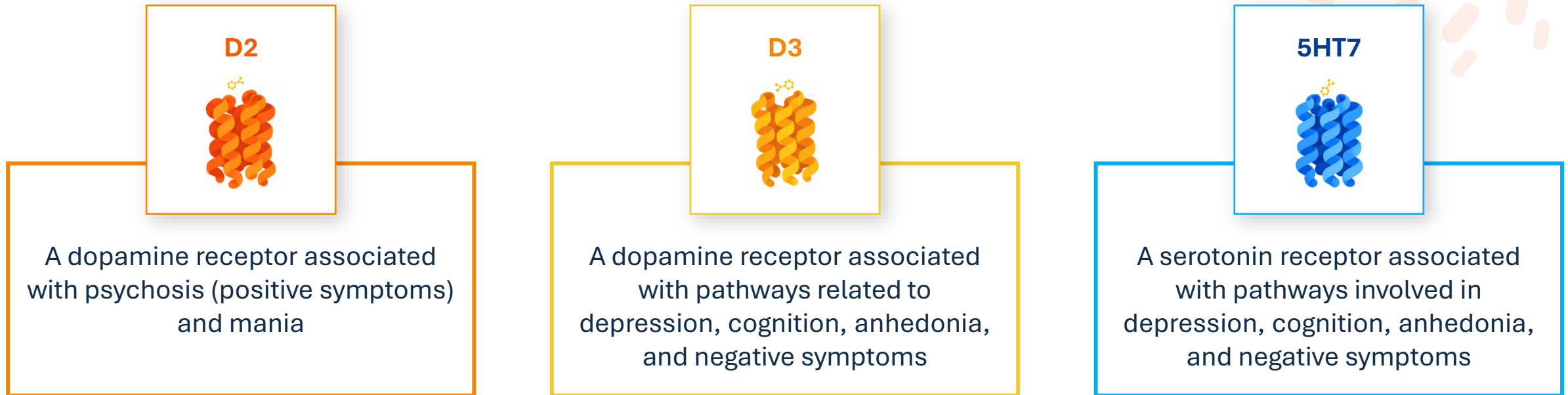


Potential advantages of LB-102 resulting from methylation of amisulpride

- ✓ Improved potency, lowering required dose and reducing systemic exposure – 50 mg LB-102 $\approx$ 400 mg amisulpride¹
- ✓ Enabled convenient once-daily dosing
- ✓ NCE status and wholly-owned issued composition of matter IP
- ✓ Potential for improved tolerability (e.g., lower EPS) consistent with NOVA-1 clinical experience
- ✓ Retained CNS receptor binding profile including lack of off-target effects²

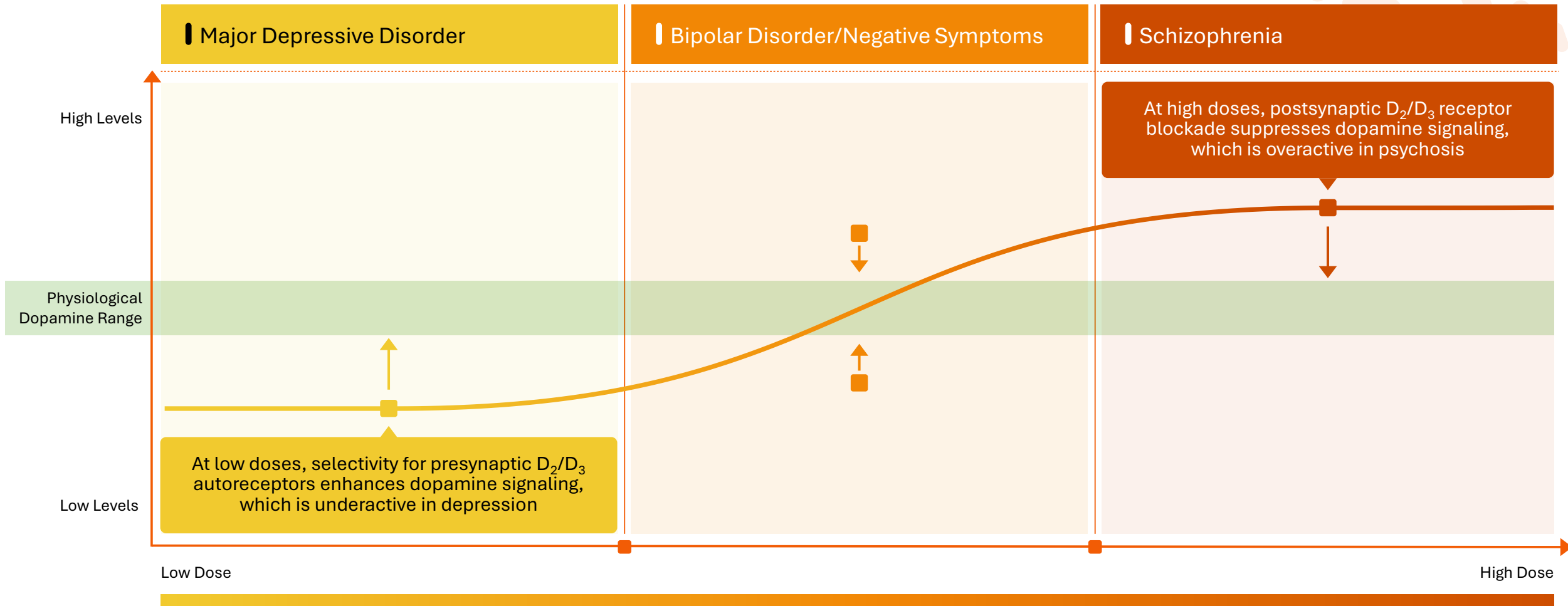
LB-102's Mechanism, NOVA-1 Data, and the Heritage of Amisulpride Support Clinical Development in a Wide Range of Indications

LB-102 and amisulpride have a distinct CNS receptor binding profile among antipsychotics



- Selective binding profile with few off-target effects contributes to the favorable tolerability profile seen in our NOVA-1 study
- Potential to preferentially engage presynaptic autoreceptors implicated in mood, anhedonia and cognition at lower doses¹

Distinct Mechanism of LB-102 and Amisulpride Provides Opportunity in a Range of Indications



LB-102 has Potential to be the Branded AP of Choice in Schizophrenia

Potential first benzamide in the U.S. for the treatment of schizophrenia



Compelling Evidence of Clinical Activity in NOVA-1

- Effect observed as early as week 1
- Clinically meaningful PANSS reduction across all doses at each week
- Strong treatment effect

Potential for a Class Leading Safety Profile

- Low EPS (including akathisia), QTc prolongation, negligible sedation
- No expected food effect or DDIs
- Few GI side effects

Potential for Differentiated Effects in Negative and Cognitive Symptoms

- Significant effect on negative symptoms at 50 mg dose
- Robust, dose dependent treatment effect on cognition

Simple once-daily dosing, starting therapeutic dose without titration, if approved

Our Goal Is to Transform What Is Possible for People With Schizophrenia

Too Few People With Schizophrenia Achieve Long-Lasting Relief

33%

Long-lasting symptomatic remission

27%

Long-lasting adequate quality of life

13%

Long-lasting functional remission

Novick D, et al. *Schizophr Res*. 2009



| Susan G. Kozauer, MD Chief Medical Officer

LB-102 is Under Development in a Range of Neuropsychiatric Disorders With Several Clinical Trials Completed To Date



Completed clinical trials

- ❖ Phase 1 SAD / MAD trial in healthy volunteers
- ❖ Phase 1b PET receptor occupancy trial
- ❖ Phase 2 trial (NOVA-1) in patients experiencing an acute exacerbation of schizophrenia



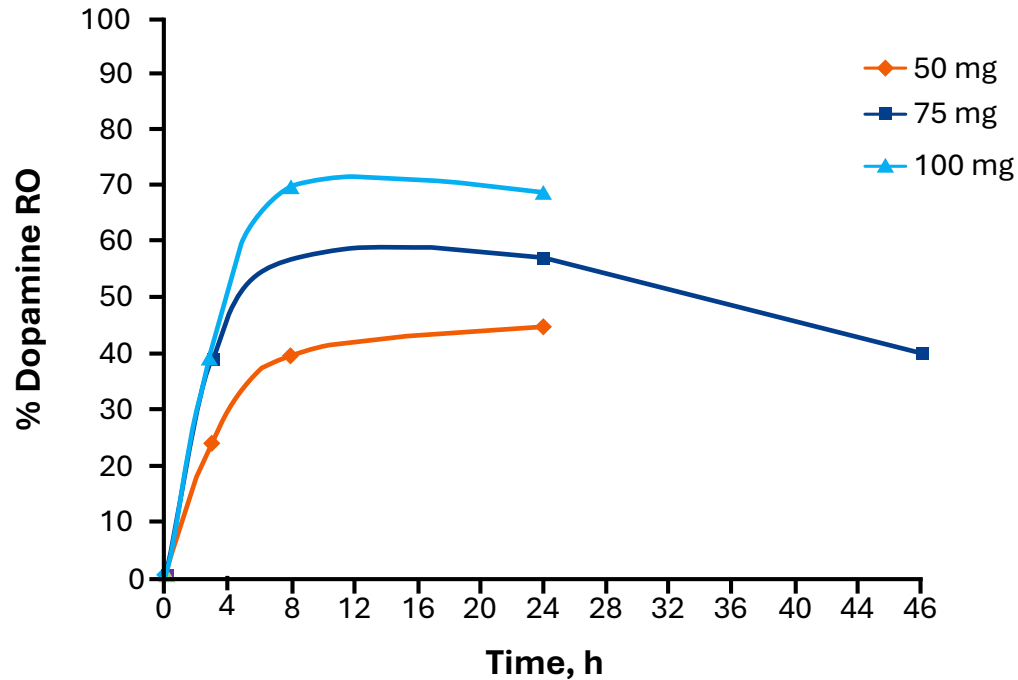
Ongoing/planned clinical trials

- ❖ Phase 3 trial (NOVA-2) in patients experiencing an acute exacerbation of schizophrenia
- ❖ Open Label 52-Week Safety-Tolerability (NOVA-3) in patients with stable schizophrenia
- ❖ Phase 2 trial (ILLUMINATE-1) in bipolar 1 depression
- ❖ Phase 2 trial in adjunctive MDD

LB-102: Striatal D₂ Receptor Occupancy

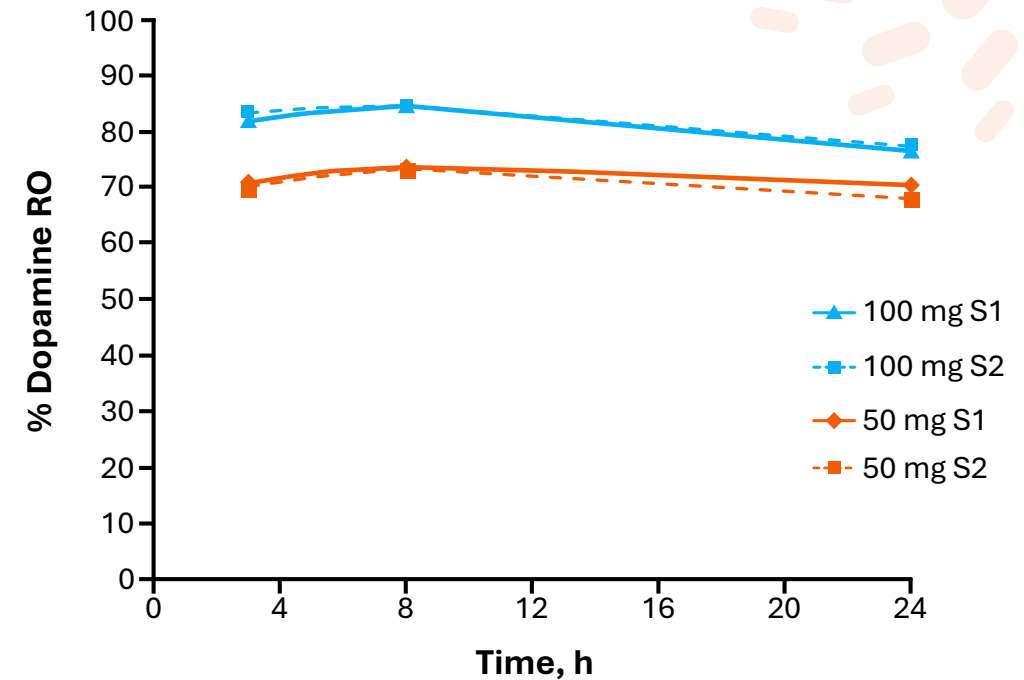
Results of a Phase 1b PET study in healthy volunteers

Average % dopamine receptor occupancy after single dose



- Receptor occupancy was ~40% nearly 2 days after a single dose despite mean plasma concentration <10 ng/mL¹

Average % dopamine receptor occupancy at steady state



- LB-102 50 mg demonstrates similar level of receptor occupancy (~70%) to amisulpride 300-400 mg/day^{1,2}
- Little variability (~10%) over 24 hours – supports once-daily dosage



Schizophrenia is a Complex Brain Disorder with Three Symptom Domains

Positive symptoms

Symptoms that affect a person's perception of reality, such as hallucinations, delusions or disorganized behavior.

Negative symptoms

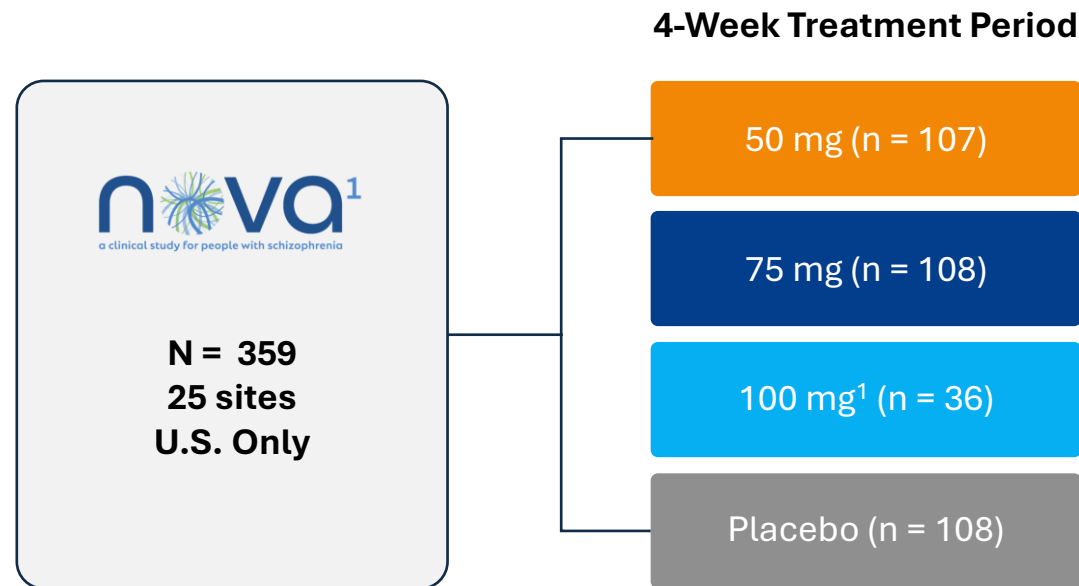
A reduction in emotional expression, motivation, pleasure, joy or social engagement.

Cognitive symptoms

Can include difficulties with memory, attention, executive function or processing information.

Successful NOVA-1 Acute Schizophrenia Trial Has Potential to Be One of Two Pivotal Trials to Support FDA Approval

NOVA -1 Trial Design



Primary Endpoint:

PANSS Δ from baseline at day 28

Secondary Endpoints:

CGI-S, PANSS positive and negative subscales, Marder factor

Exploratory Endpoint: Cognition

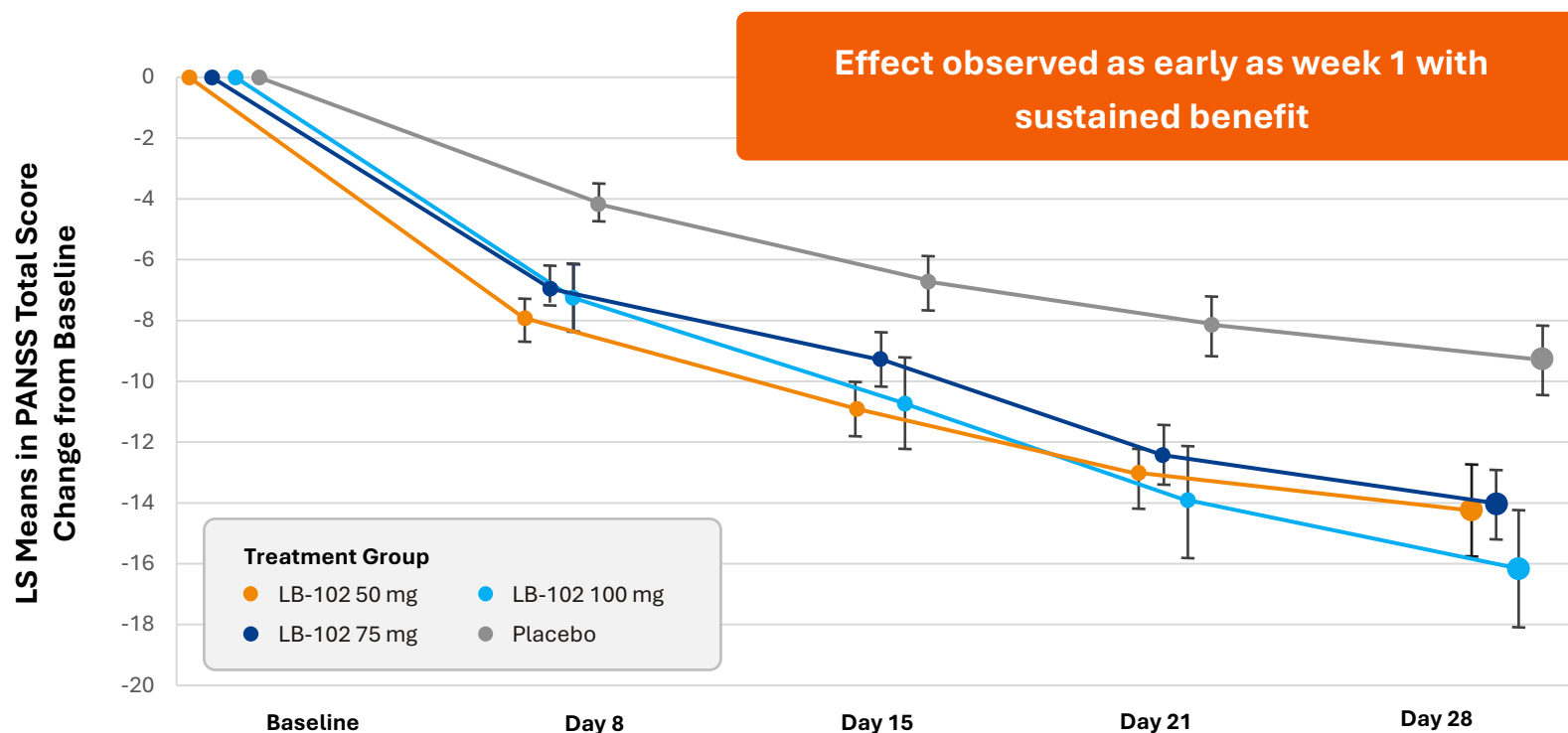
Designed trial to be potentially pivotal with large sample size, robust statistical analyses, conservative imputation of missing data

FDA noted, in writing, that our NOVA-1 trial appeared to have many of the characteristics of an adequate and well-controlled trial – opportunity to seek approval with one additional successful registrational trial



Statistically Superior Clinical Activity to Placebo at All Three Doses

Clinically meaningful PANSS reduction in 4-week NOVA-1 schizophrenia trial (baseline PANSS of 94)



PANSS Δ from baseline (Δ vs. Placebo at Day 28)

- 50 mg**
-14.3 (-5.0 vs. Pbo)
($p=0.0009$)
- 75 mg**
-14.0 (-4.7 vs. Pbo)
($p=0.0022$)
- 100 mg**
-16.1 (-6.8 vs. Pbo)
($p=0.0017$)
- Placebo**
-9.3

Compelling Treatment Effect at Doses Planned for Phase 3

	Dose	Effect Size (completers)	Effect Size (MMRM)
LB-102 (NOVA-1)	50 mg	0.61	0.50
	100 mg	0.83	0.64

- ✓ Results for LB-102 are near the top of effect size (ES) reported for approved first-line APs^{1,2}
- ✓ ES for 100 mg dose are in the range of those previously reported for amisulpride

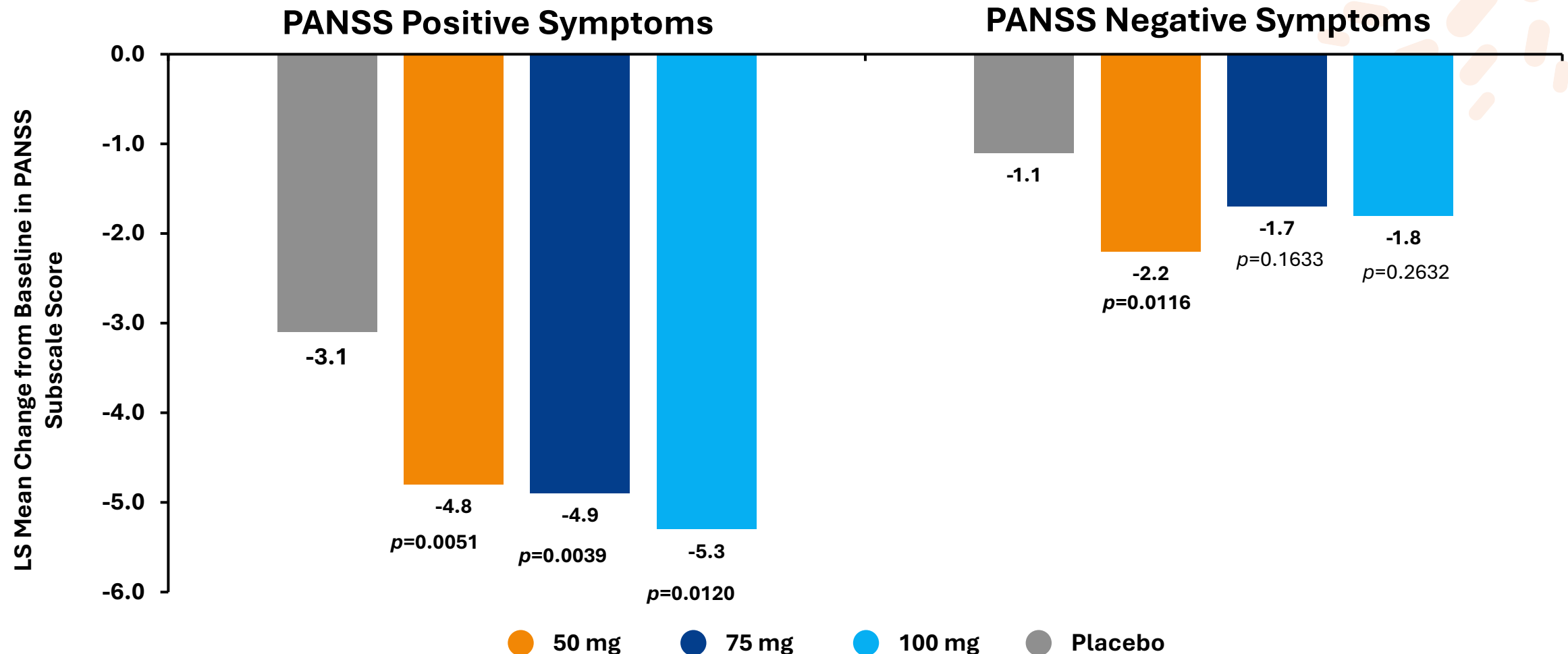
Results demonstrate statistical robustness of NOVA-1 trial and provide confidence heading into Phase 3

Effect size is calculated by taking the difference in average PANSS change between two groups (an active treatment arm and placebo) and dividing it by the pooled standard deviation. Completer analysis includes observed data from patients who received the protocol specified four weeks of treatment; MMRM refers to Mixed Model for Repeated Measures. MMRM analysis includes observed and imputed data from all patients with at least one post baseline PANSS assessment; 100 mg was exploratory dose. Descriptive comparison of point estimates from separate trials. Not based on a head-to-head comparison. Differences in study populations, designs, assessment time points, and statistical methods may affect comparability

1. Huhn M, et al. *Lancet*. 2019; 2. Fabiano, N, et al, *European Neuropsychopharmacology*. 2025

Improvements Were Demonstrated in NOVA-1 at Week 4 on Both Positive and Negative Symptoms

Observed effect on negative symptoms at low dose consistent with bimodal mechanism



| Robust, Dose-Dependent Effect on Cognition in NOVA-1, a Key Unmet Need

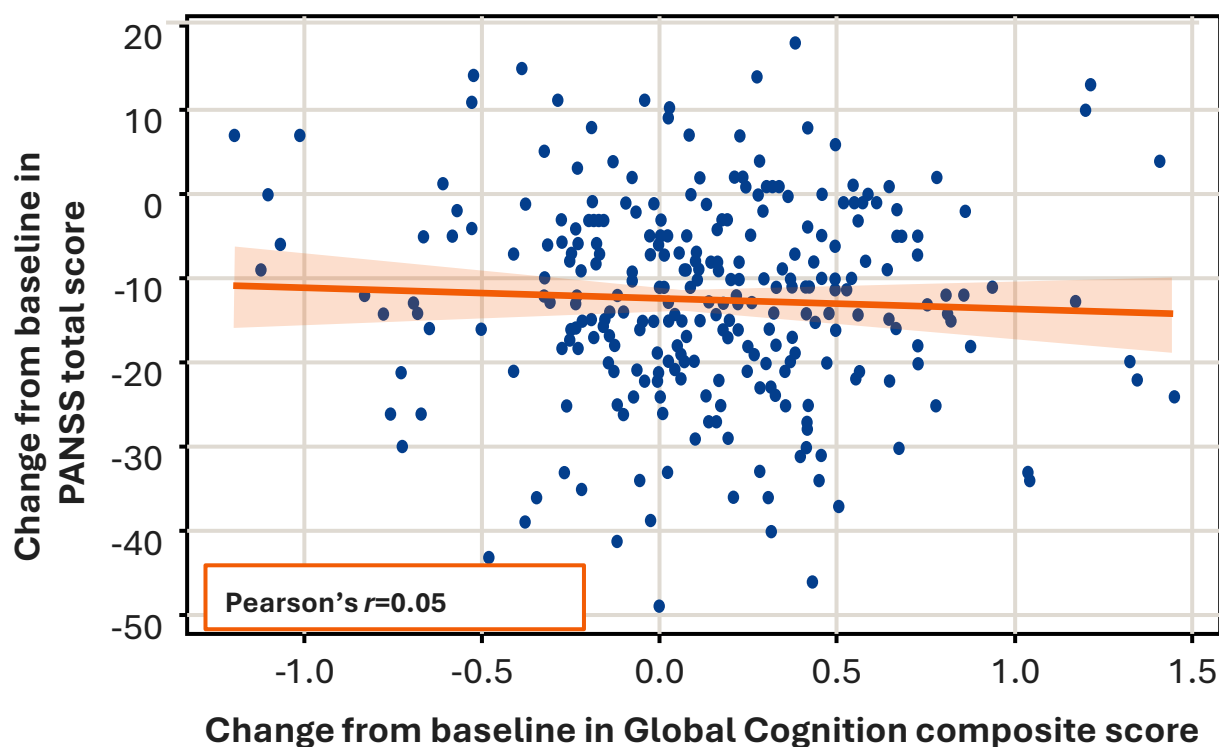
Global composite effect¹: psychomotor function, memory, attention, working memory and executive function

	Dose	Effect Size	p-value
LB-102 (NOVA-1)	50 mg (n=90)	0.26	0.0476
	75 mg (n=83)	0.41	0.0027
	100 mg (n=23)	0.66	0.0018

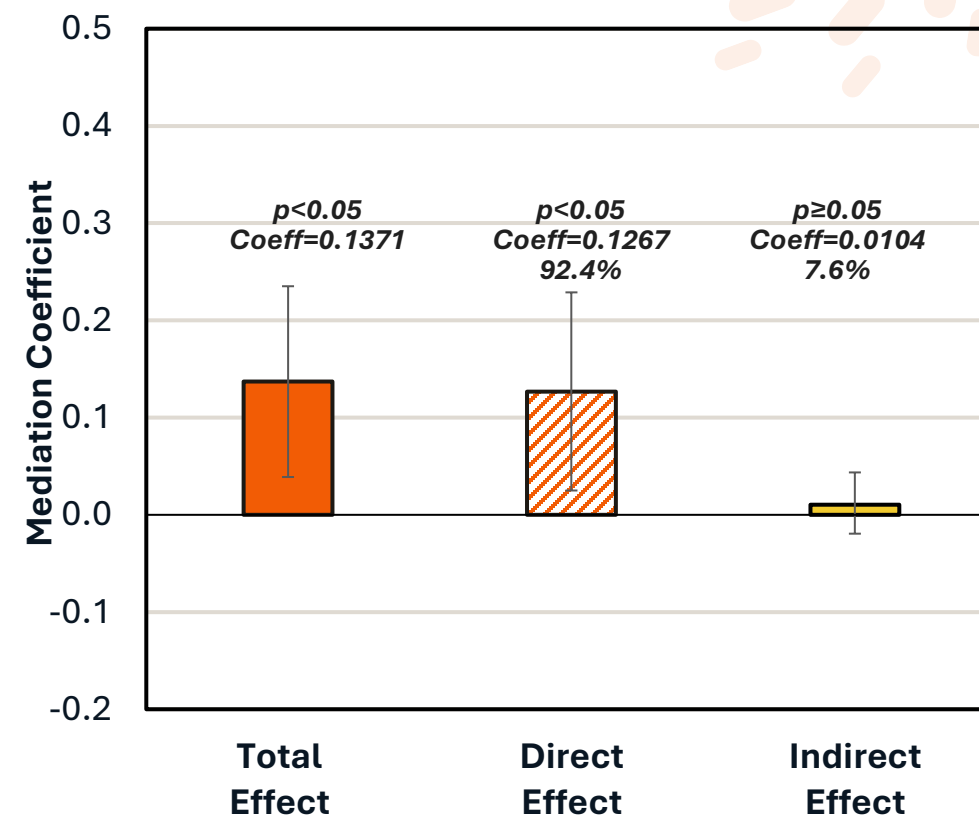
- Significant, dose-dependent improvement in cognition consistent with LB-102 mechanism
- Magnitude of observed benefit is considered clinically meaningful
- High rate of satisfactory completion of tests demonstrates reliability of data
- Broad patient population; effect observed without enriching analysis for severe cognitive impairment at baseline
- Cognitive deficits are highly prevalent with significant unmet need spanning schizophrenia, bipolar depression and MDD²

Improvement in Cognitive Performance was Primarily a Direct Effect of LB-102

Bivariate Analysis of the Association Between Treatment-Related Changes in PANSS Total Score and Global Cognition Composite Score for LB- 102 in NOVA-1



Mediation Analysis of the Effect of LB-102 on Global Cognition Composite Score



Favorable Adverse Event (AE) Profile in NOVA-1

Most AEs were mild or moderate in severity and similar to placebo

Adverse Events Reported in ≥5% of Patients Number of subjects (% of treatment group)	50 mg (n=107)	75 mg (n=108)	100 mg (n=36)	Placebo (n=108)
Insomnia	27 (25.2%)	23 (21.3%)	14 (38.9%)	24 (22.2%)
Headache	12 (11.2%)	9 (8.3%)	2 (5.6%)	10 (9.3%)
Anxiety	10 (9.3%)	9 (8.3%)	4 (11.1%)	9 (8.3%)
Agitation	11 (10.3%)	6 (5.6%)	4 (11.1%)	10 (9.3%)
Weight increase	13 (12.1%)	8 (7.4%)	3 (8.3%)	4 (3.7%)
Hyperprolactinemia	11 (10.2%)	8 (7.5%)	6 (16.6%)	0
Blood creatine phosphokinase increased	4 (3.7%)	1 (0.9%)	2 (5.6%)	3 (2.8%)
Alanine aminotransferase increased	3 (2.8%)	1 (0.9%)	2 (5.6%)	1 (0.9%)
Somnolence	1 (0.9%)	4 (3.7%)	2 (5.6%)	0
Constipation	4 (3.7%)	1 (0.9%)	2 (5.6%)	0

- AEs leading to discontinuation were reported at the following rates: 50 mg (1.9%), 75 mg (2.8%), 100 mg (8.3%), Pbo (1.9%)
- Serious Adverse Events (SAE) occurred at the following rates: 50 mg (less than 1%), 75 mg (less than 1%), 100 mg (2.8%), Pbo (1.9%)
- Comorbid conditions at entry influenced reporting of TEAEs which were defined as any AE that began on or after the first dose, or any pre-existing condition that reappeared during treatment or follow up. As a result, AEs such as insomnia appear elevated
- Weight gain reflects any increase without a threshold. We observed ~1.6 kg placebo adjusted weight gain while preserving metabolic status¹

Potential for a Class Leading Rate of EPS (Including Akathisia)

Total EPS Observed in LB-102 NOVA-1 Trial
Number of subjects (% of treatment group)

Preferred Term	50 mg (n=107)	75 mg (n=108)	100 mg (n=36)	Placebo (n=108)
Dystonia	0	3 (2.8%)	1 (2.8%)	1 (0.9%)
Akathisia	1 (0.9%)	2 (1.9%)	0	1 (0.9%)
Extrapyramidal disorder	0	1 (0.9%)	1 (2.8%)	2 (1.9%)
Total EPS	1 (1.0%)	6 (5.6%)	2 (5.6%)	4 (3.7%)

- EPS is generally correlated with dopamine receptor occupancy (RO) rate
- EPS rates (including akathisia) observed with other D₂ antagonists and partial agonists can reach more than 30%
- EPS rates with LB-102 were low despite 70-80% RO

Low Rates of Other Adverse Events of Interest Support the Potential for a Class Leading Safety Profile



Prolactin-related AEs Low rate (~1-5.6%); AEs were mild to moderate and did not result in discontinuation



Sedation Single case of sedation among 251 patients exposed to LB-102 in NOVA-1 trial



QTc Prolongation Minimal QT prolongation (4.3-5.4 ms); no patients met pre-specified stopping criteria

LB-102 has Potential to be the Branded AP of Choice in Schizophrenia

Potential first benzamide in the U.S. for the treatment of schizophrenia



Compelling Evidence of Clinical Activity in NOVA-1

- Effect observed as early as week 1
- Clinically meaningful PANSS reduction across all doses at each week
- Strong treatment effect

Potential for a Class Leading Safety Profile

- Low EPS (including akathisia), QTc prolongation, negligible sedation
- No expected food effect or DDIs
- Few GI side effects

Potential for Differentiated Effects in Negative and Cognitive Symptoms

- Significant effect on negative symptoms at 50 mg dose
- Robust, dose dependent treatment effect on cognition

Simple once-daily dosing, starting therapeutic dose without titration, if approved

Efficacy, Tolerability and Simplicity of Use Enable Patients to Sustain Treatment



This matters because 80% of patients relapse within 5 years, and consequences can be serious:



Recovery can be slower and less complete, and illness can become more resistant to treatment



More frequent admissions to hospital and greater use of healthcare resources



Increased risk of self-harm and homelessness



Loss of self-esteem and social and vocational disruption

We are Exploring the Potential Mechanistic Underpinnings of LB-102's Observed Clinical Profile

LOWER EPS POTENTIAL

EPS rate similar to placebo in NOVA-1,
despite 70 – 80% RO

MECHANISTIC HYPOTHESIS

Regional selectivity

NEGATIVE SYMPTOM EFFECTS

Significant effect at 50 mg dose
in NOVA-1

MECHANISTIC HYPOTHESIS

Presynaptic autoreceptor
antagonism

D3 / 5HT-7 antagonism

| Roger McIntyre, MD

Professor of Psychiatry and Professor of Pharmacology
and Toxicology, University of Toronto

| Disclosure

Dr. McIntyre has been engaged by LB Pharma as a medical advisor and to speak at this program. Accordingly, Dr. McIntyre has a financial relationship with LB Pharma

The research studies presented in this presentation were conducted by LB Pharma; not by Dr. McIntyre

| Agenda

- Brain regions and their connection to schizophrenia symptoms
- Regional selectivity as a potential mechanism for low EPS with high D2 RO
- Understanding amisulpride's bimodal activity profile
- Effects of LB-102 on pre-synaptic autoreceptors
- Next steps

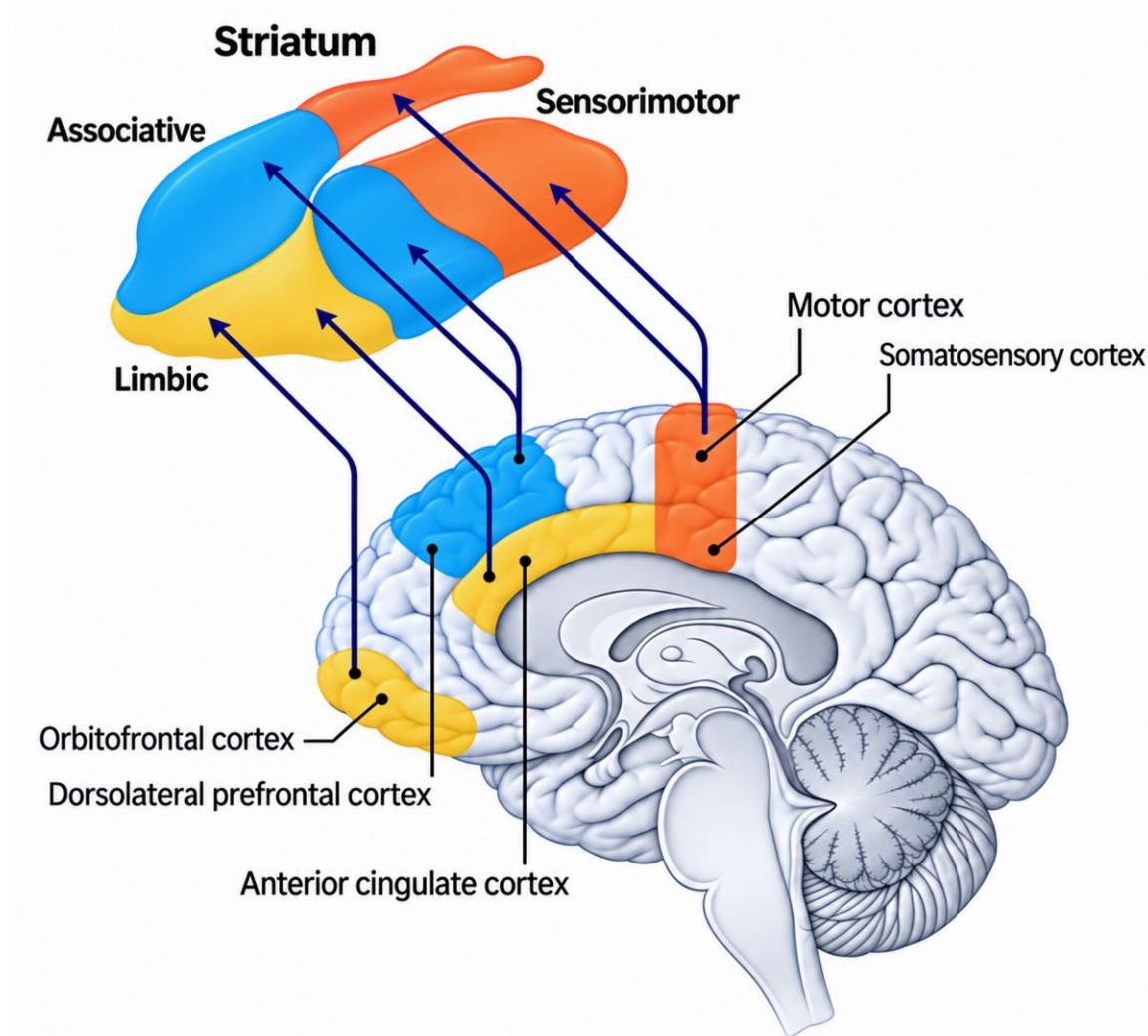


The Core Symptoms of Schizophrenia Can Be Mapped to Aberrant Dopamine Signaling in Specific Regions of the Brain

Positive Symptoms
(associative striatum)

Negative Symptoms
(Limbic Striatum:
Ventral Striatum &
Nucleus Accumbens)

Cognitive Symptoms
(Prefrontal Cortex)



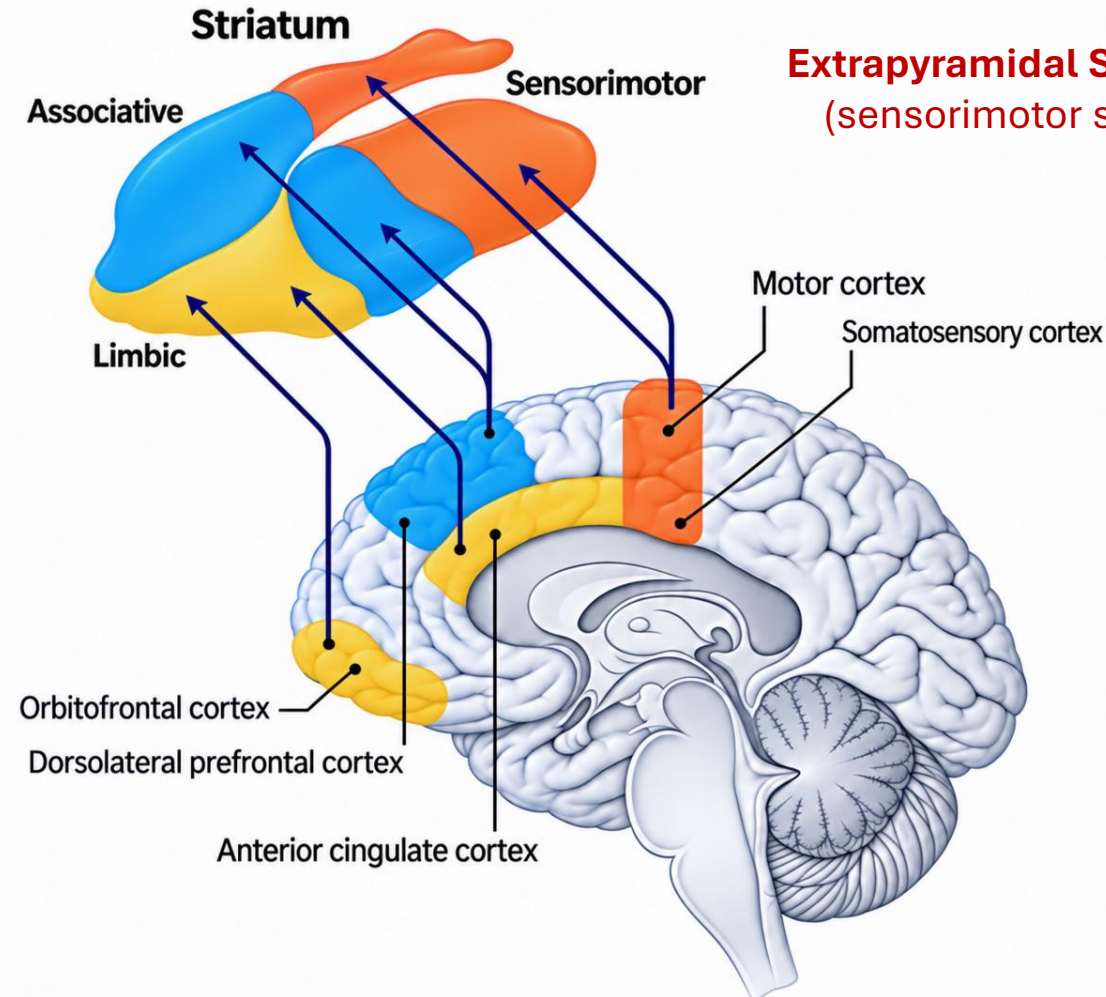
- **Hyper**dopaminergia in the associative striatum, is considered to be associated with positive symptoms¹
- **Hypo**dopaminergia and diminished dopaminergic reactivity in the limbic striatum and prefrontal cortex are postulated to play a role in negative and cognitive symptoms^{2,3}

Many Therapeutics That Address Positive Symptoms Also Impact Dopamine Signaling in Parts of the Brain that Can Cause Side Effects

Positive Symptoms
(associative striatum)

Negative Symptoms
(Limbic Striatum:
Ventral Striatum &
Nucleus Accumbens)

Cognitive Symptoms
(Prefrontal Cortex)



- Non-selective dopamine receptor antagonist antipsychotics inhibit dopamine signaling in the sensorimotor striatum^{1,2}
- This inhibition is considered to be related to extrapyramidal symptom (EPS) side effects including parkinsonism, akathisia, and tardive dyskinesia^{1,3}

To Better Understand LB-102's Low Rate of EPS Observed in NOVA-1 Despite High D₂ RO, Regional Selectivity in the CNS was Hypothesized to Play a Role

SCIENTIFIC QUESTION



Does LB-102 affect dopamine signaling in the associative vs sensorimotor striatum differently?

- If LB-102 acts predominantly in the associative striatum and motor regions are less affected, this could account for low EPS despite high D₂ RO
- Selectivity for the associative versus motor striatum could also potentially account for the NOVA-1 observation of clinical activity and a low rate of EPS



STUDY DESIGN

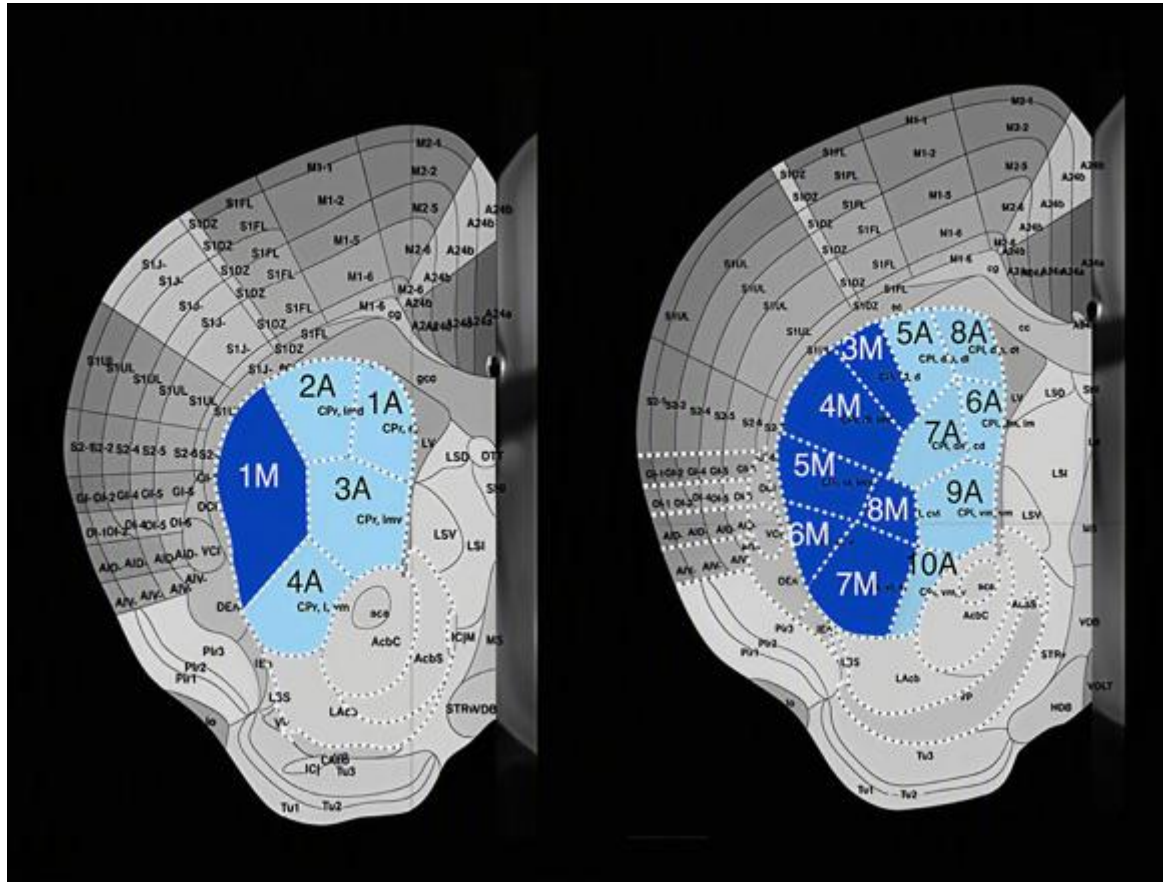


Assessed pattern of neuronal activity in whole mouse brain after administration of LB-102

- Measured drug-induced changes in c-Fos, a marker of neuronal activation, versus control
- Mapped areas of the brain where LB-102 is active onto an atlas of mouse brain regions
- Compared LB-102's regional activity profile with that of amisulpride, haloperidol, and cariprazine
- Calculated associative to motor striatal activation ratios for each agent

The Mouse Brain Atlas Divides the Brain Into Functional Units and Differentiates Between the Associative and the Motor Striatum

Subdivisions of the mouse caudoputamen, a part of the striatum, from the mouse brain atlas

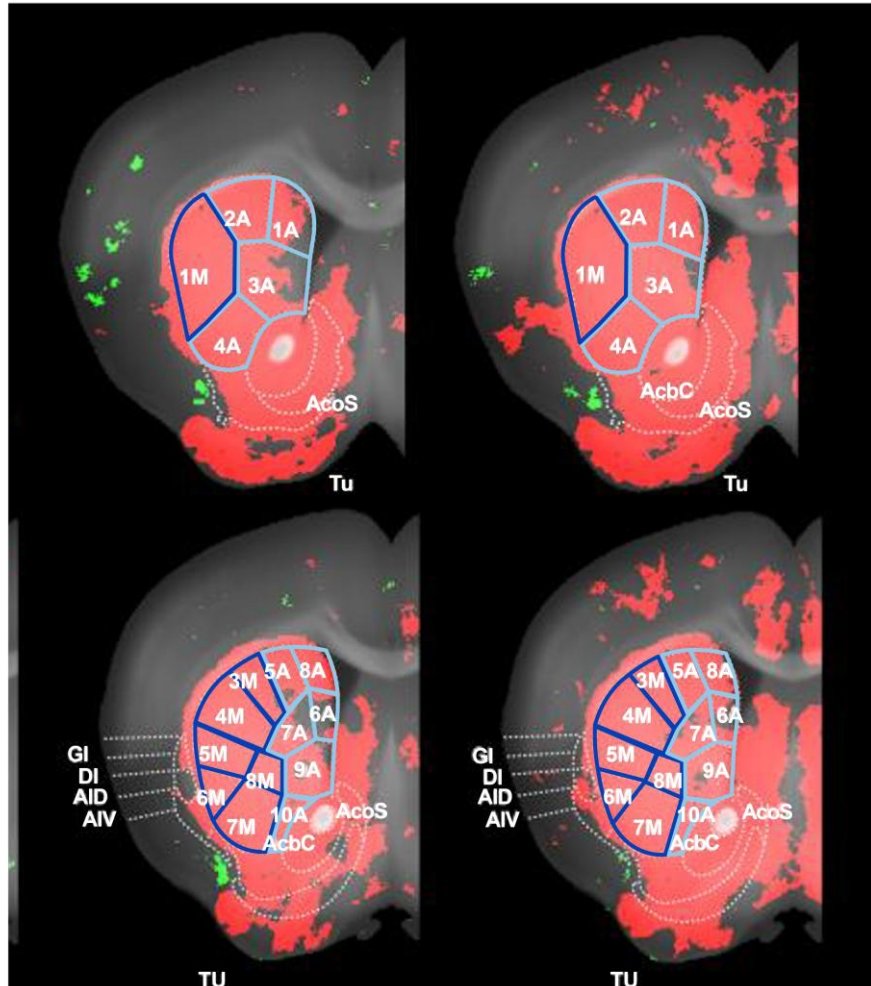


- Associative striatum inputs
- Motor striatum inputs

- Associative striatum inputs (light blue regions)
- Motor striatum inputs (dark blue regions)
- Activity mapping results displayed as color-coded spatial areas of statistically significant differences between active and control
- Observed differences are overlaid on the grayscale reference mouse brain
- Conserved cortico-striatal organization across species supports the translational relevance of mouse activity-mapping findings to human striatal biology^{1,2}

Haloperidol, an Antipsychotic Drug Associated with High Rates of EPS, Showed Clear Signs of Activity Throughout the Associative and Motor Striatum

Hal 0.5 mg/kg, sc Hal 1.0 mg/kg, sc



- This pattern is consistent with positive symptom efficacy as well as high rates of EPS^{1,2}
- The associative to motor striatal activation ratio for haloperidol is 0.45-0.59
- Clinically EPS rates for haloperidol have been reported as 44-56%¹

Red shows **increases** in neuronal activity (c-fos+ cell counts)

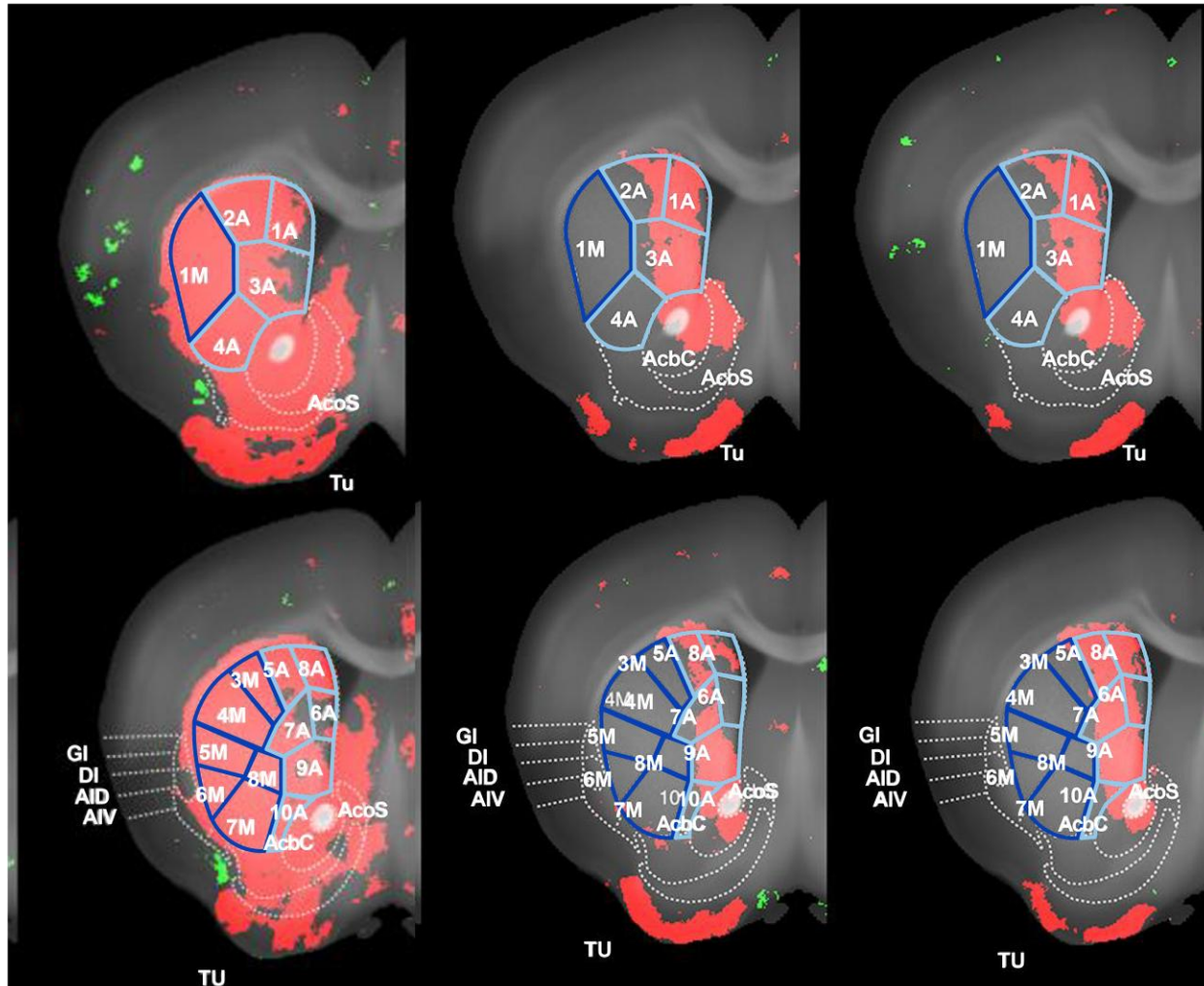
Green shows **decreases** in neuronal activity (c-fos+ cell counts)

□ Associative striatum inputs

□ Motor striatum inputs

LB-102 and Amisulpride Showed a Distinct, Motor Sparing Pattern of Activity Compared with Haloperidol

Hal 0.5mg/kg, sc Ami 30 mg/kg, sc LB 20 mg/kg, sc



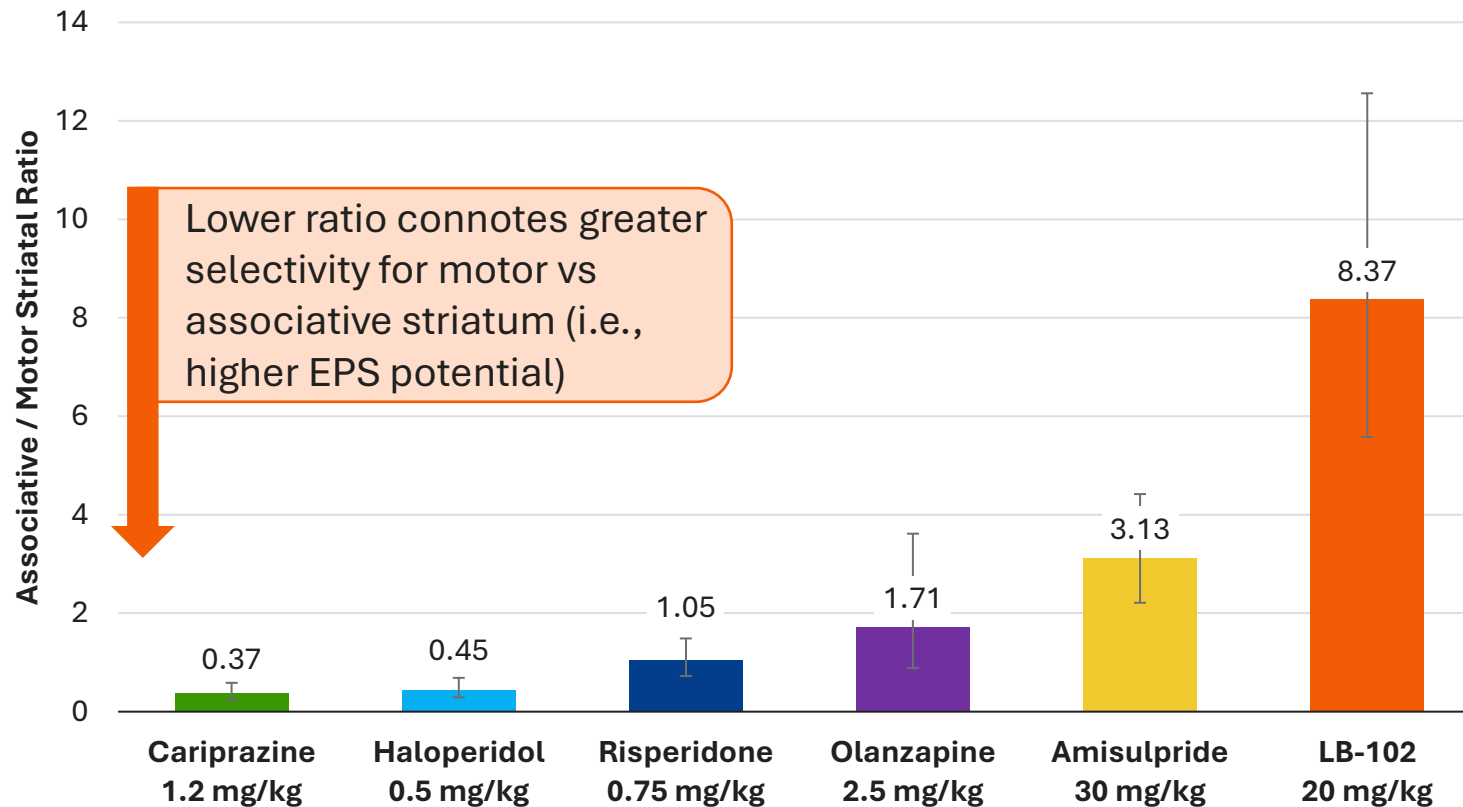
- LB-102 and amisulpride predominantly affect the associative striatum while sparing motor regions versus control
- This pattern is consistent with positive symptom activity and lower rates of EPS
- The associative to motor striatal activation ratios for LB-102 and amisulpride are 8.37 and 3.13, respectively, at these doses
 - Suggests LB-102 has a larger motor sparing effect than amisulpride in this experiment
 - Consistent with LB-102's lower rates of EPS compared with amisulpride

Red shows **increases** in neuronal activity (c-fos+ cell counts)
Green shows **decreases** in neuronal activity (c-fos+ cell counts)

□ Associative striatum inputs
□ Motor striatum inputs

Regional Selectivity is a Plausible Potential Mechanism for the Low Rate of EPS Observed with LB-102 in NOVA-1

Striatal Selectivity in Mouse Brain



The associative to motor striatal activation ratio of LB-102 was higher than that of cariprazine, haloperidol, risperidone, olanzapine, and amisulpride

The EPS Rate Observed in NOVA-1 Following Treatment with LB-102 Was Lower Than That Reported for Other D₂ Antagonists and Partial Agonists

Selected Antipsychotics	Clinical Rates of EPS (including akathisia)
Haloperidol	44 - 56% ¹
Risperidone	17 - 35% ²⁻⁴
Olanzapine	15 - 32% ⁵
Cariprazine	24 - 33% ^{6,7}
Amisulpride	11 - 23% ⁸
LB-102 (NOVA-1)	0.9 - 5.6%⁹

The EPS rate observed for LB-102 in NOVA-1 is lower than that reported for other D₂ antagonists

Summary and Next Steps (Regional Selectivity)

- Preclinical c-Fos activation study suggests LB-102 may have a regionally selective activity profile in the brain
 - Preferentially active in associative versus motor related areas of the striatum
 - Supports plausible potential mechanism for observed low rate of EPS despite high D2 RO
- LB-102 may operate in “zip codes” that offer potential for clinical activity while potentially minimizing motor related toxicity
- EPS (including akathisia) is one of the most common reasons for discontinuation of APs^{1,2}

Next steps include additional preclinical analyses of regional effects on c-Fos activity in response to LB-102 in the cortex, hippocampus and midbrain brain regions and with longer term dosing

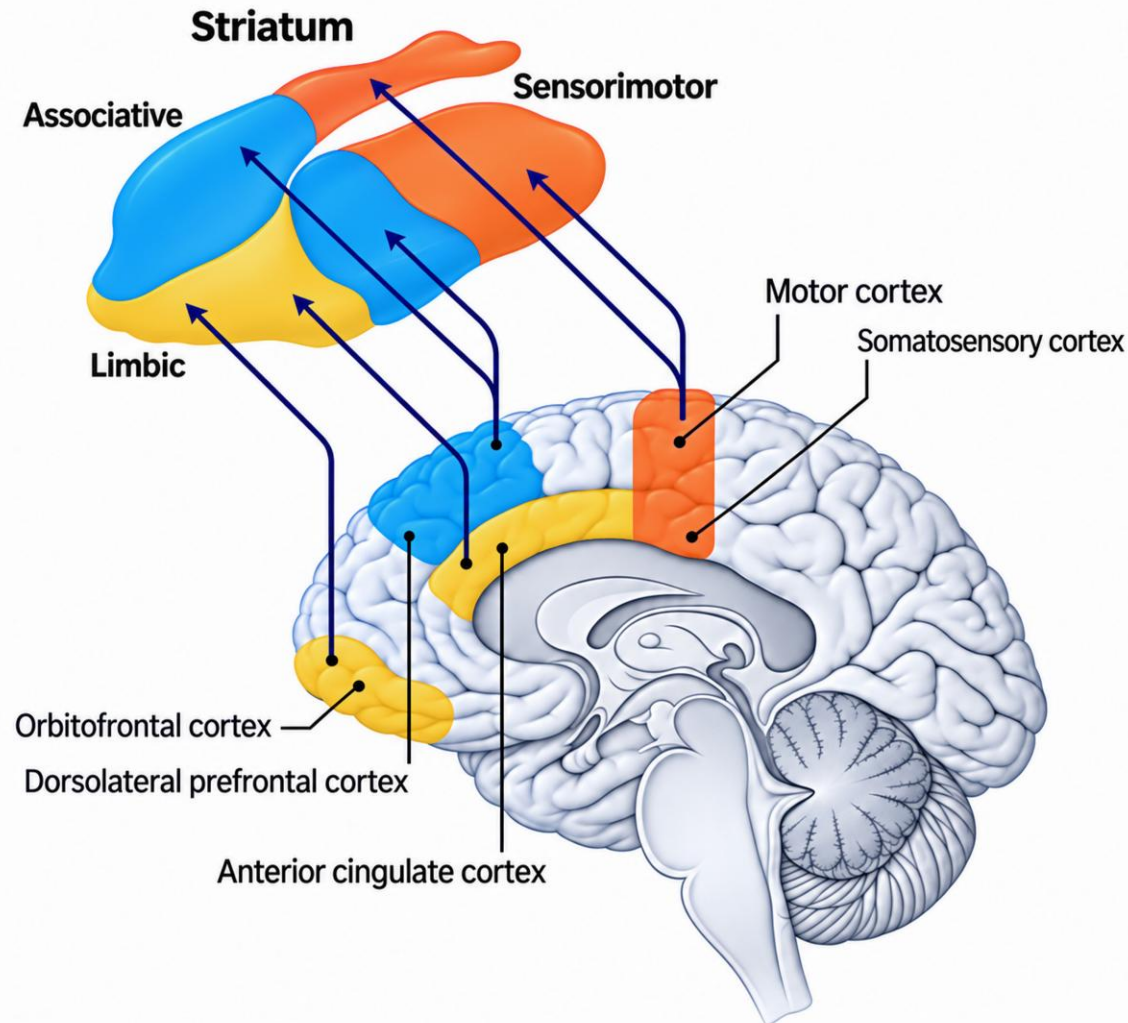
Could further support understanding of clinical effects in negative symptoms and cognition

Negative Symptoms is an Area of Unmet Need Where There are Currently No Approved Therapies in the U.S.

Positive Symptoms
(associative striatum)

Negative Symptoms
(Limbic Striatum:
Ventral Striatum &
Nucleus Accumbens)

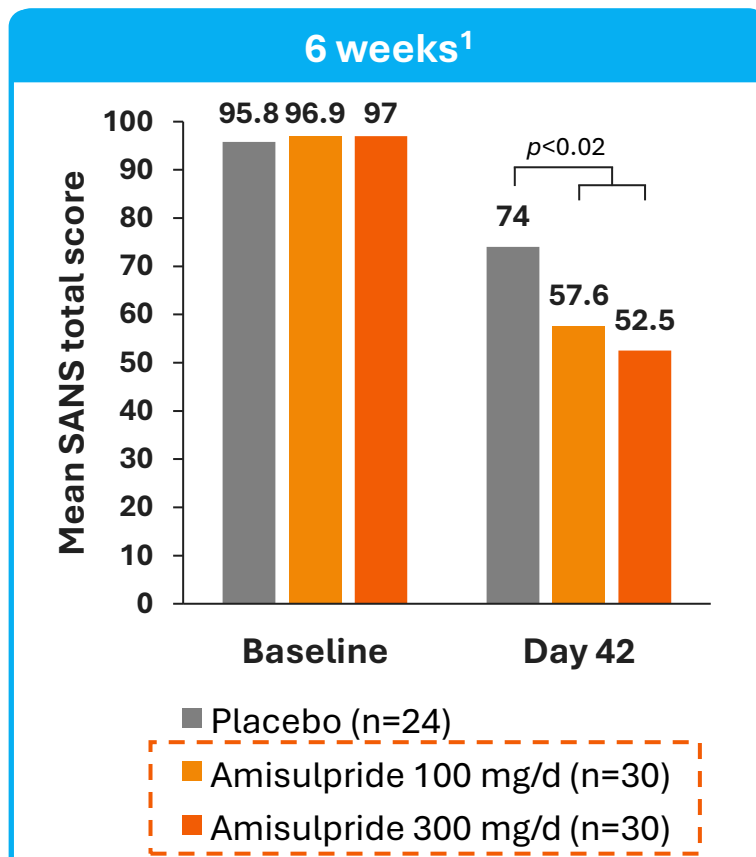
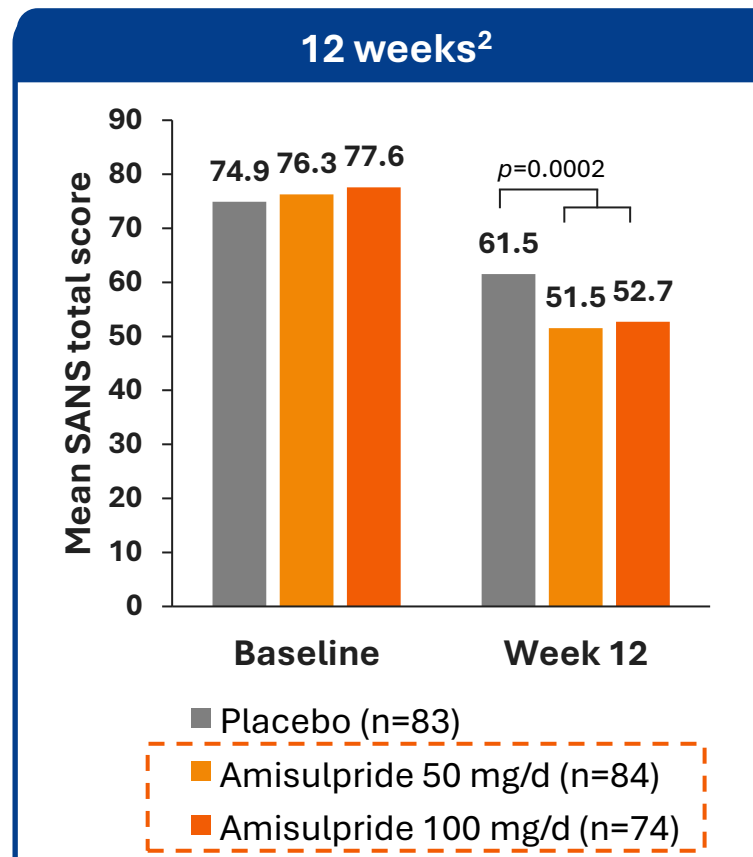
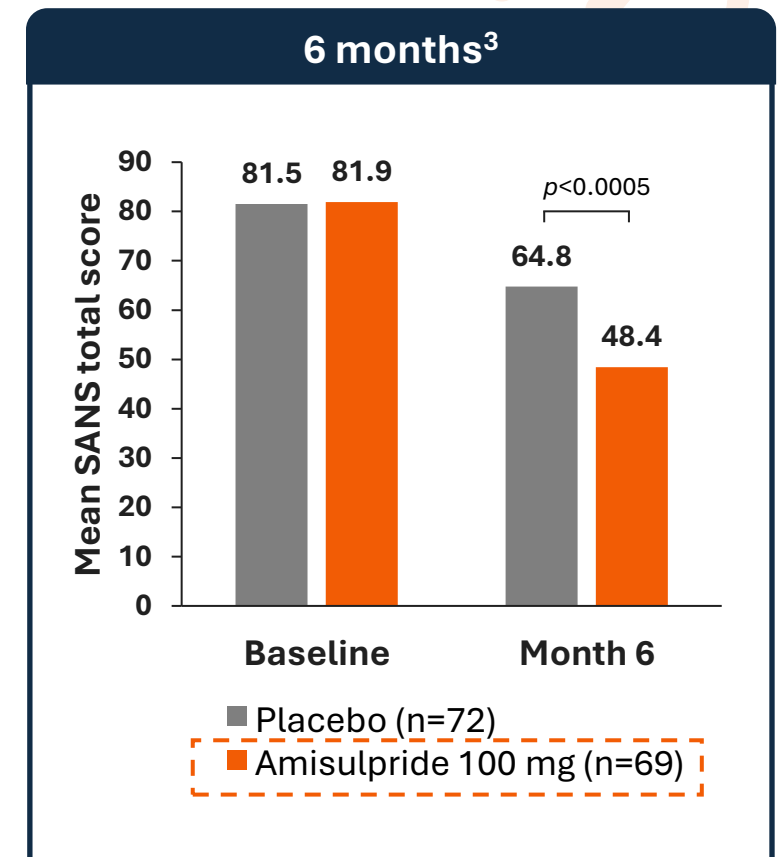
Cognitive Symptoms
(Prefrontal Cortex)



- **Hypo**dopaminergia and diminished dopaminergic reactivity in the limbic striatum and prefrontal cortex are postulated to play a role in negative and cognitive symptoms^{1,2}
- Historically, unique negative symptom activity and procognitive effects of amisulpride have hypothesized to be linked to limbic selectivity and low-dose D2/D3 autoreceptor antagonism preference

Multiple Sources Showed that Amisulpride Demonstrated Statistically Significant Benefit vs Pbo in Negative Symptoms of Schizophrenia, Specifically at Lower Doses

Low-dose amisulpride (50–300 mg/day) has reproducible efficacy in predominant/primary negative symptoms of schizophrenia in multiple randomized, double-blind, placebo-controlled trials¹⁻³

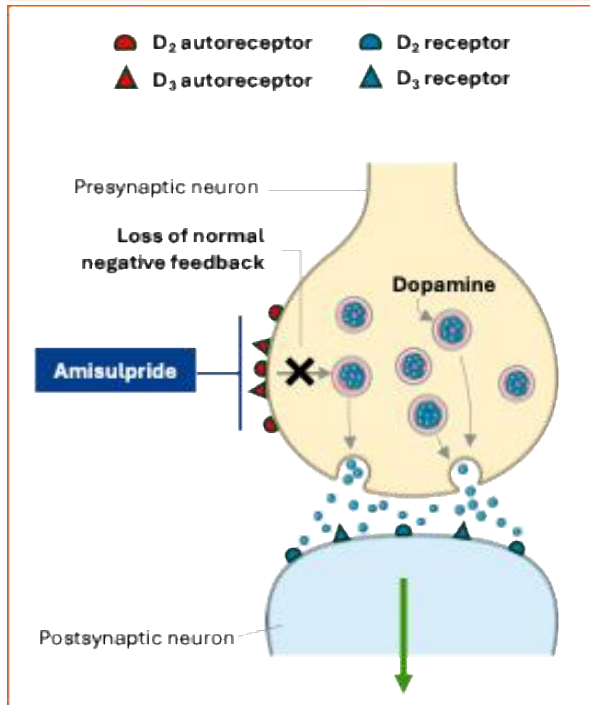


Underlying These Clinical Results is Preclinical Data That Suggests Amisulpride Has a Bimodal Mechanism of Action

LOWER DOSES

Presynaptic Autoreceptor Blockade

- Rationale for treating **negative** and **mood** symptoms¹⁻⁵

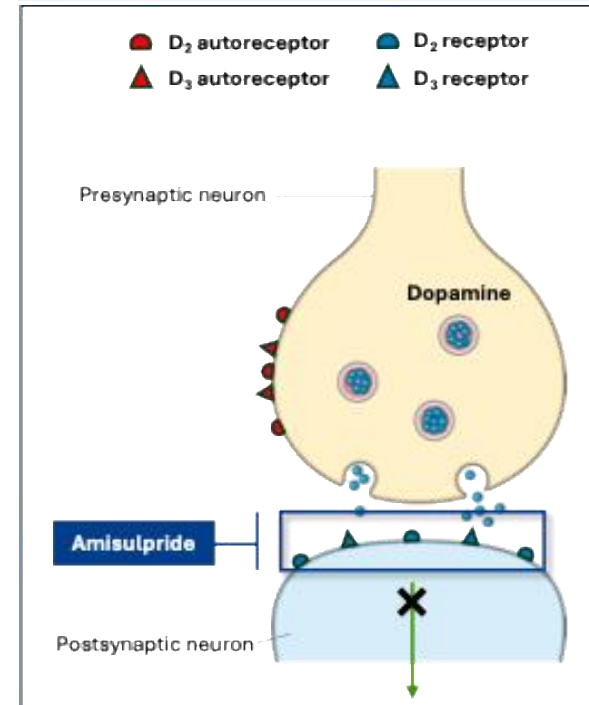


Enhanced
Dopamine
Neurotransmission

HIGHER DOSES

Postsynaptic Receptor Antagonism

- Rationale for treating **positive** symptoms¹⁻⁵



Reduced
Dopamine
Neurotransmission

LB-102 was Hypothesized to be Able to Antagonize Presynaptic Autoreceptors and Increase Dopamine Signaling like Amisulpride

SCIENTIFIC QUESTION



Can LB-102 antagonize pre-synaptic autoreceptors and trigger the release of dopamine in the nucleus accumbens?

- May support potential for bimodal mechanism of action similar to that of amisulpride
- May provide a mechanism to address the hypodopaminergic state associated with anhedonia and diminished motivation in negative symptoms of schizophrenia and depression



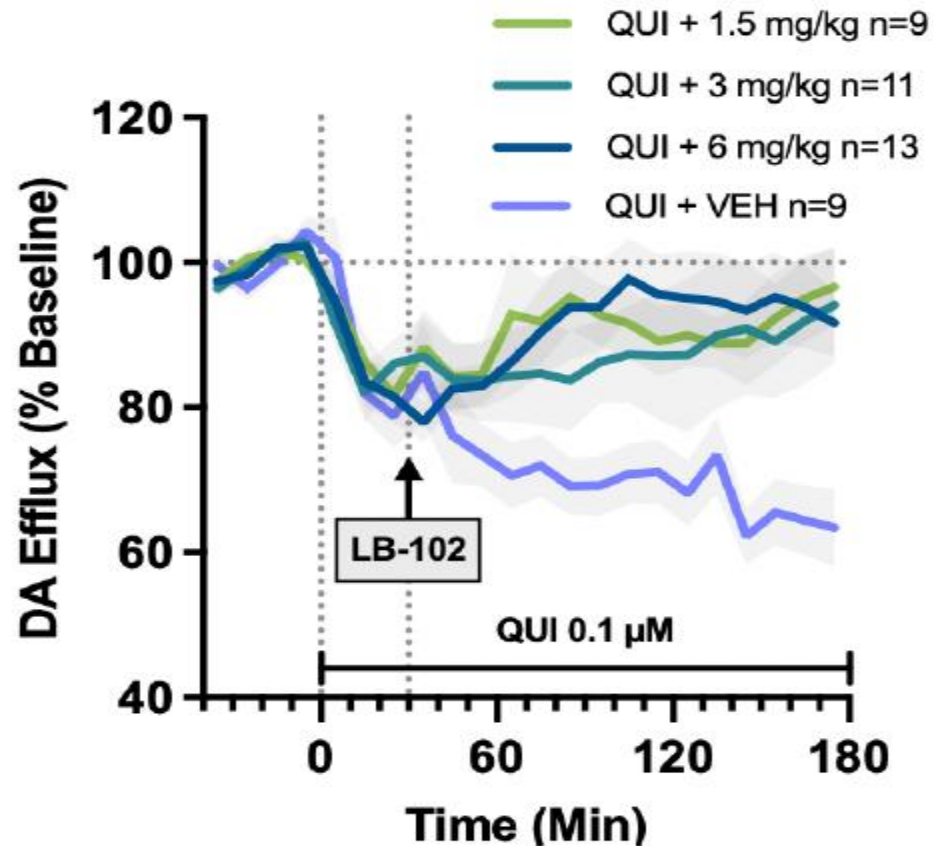
STUDY DESIGN



Assessed the ability of oral administration of LB-102 to trigger the release of dopamine in the nucleus accumbens of mice

- Surgically placed a microdialysis probe in the nucleus accumbens (NAc) of mice
- Infused quinpirole, an autoreceptor agonist, through the probe to suppress dopamine efflux
- Administered LB-102 orally
- Evaluated LB-102's ability to increase dopamine efflux in the nucleus accumbens

LB-102 Blocked Presynaptic D2 Autoreceptor Inhibitory Activity and Triggered the Release of Dopamine



- Local infusion of quinpirole in the NAc produced a marked and sustained decrease in dopamine efflux
- LB-102, administered 30 min after quinpirole, gradually reversed the quinpirole-reduced dopamine efflux toward baseline
- Informal behavioral observations suggested increased arousal and exploration in treated animals after administration of LB-102
- Results presented at ECNP (Oct 2026)

These data strengthen the scientific rationale for the clinical development of LB-102 in mood disorders
Suggests a potential mechanism for addressing key residual symptoms such as anhedonia

Summary and Next Steps (Limbic Selectivity and Bimodal Activity)

- Results of preclinical studies to date highlight the potential of LB-102 to modulate mesolimbic dopaminergic signaling and reward circuitry
- These data support the scientific rationale for the clinical development of LB-102 in negative symptoms, bipolar depression, and MDD
- Negative symptoms and anhedonia are significant unmet needs¹ and may overlap with cognitive impairment²

Next steps include: further preclinical evaluation of dynamics of the bimodal effects, deeper evaluation of the role of D₃ antagonism with respect to both depression / negative symptoms and cognition



Summary and Insights



| Kaya Pai Panandiker Chief Commercial Officer

Unmet Needs Persist Across Schizophrenia Treatment

“If you look at the percentage of people who are taking a traditional, either 1st or 2nd generation antipsychotic, a large percentage are not adequately controlled.”

U.S. psychiatrist, LB-102 primary market research, 2026

Schizophrenia affects millions of Americans¹

~3M U.S. adults have schizophrenia¹

Treatment requires balancing efficacy, tolerability and simple use²

74% of patients discontinued their initial treatment within 18 months²

Inadequate response drives switching³

58% of oral AP switches involved inadequate clinical response³

Negative symptoms remain common and debilitating⁴

~60% of patients experience negative symptoms⁴

Cognitive deficits negatively impact function⁵

~80% of patients have impaired cognition⁵

Treatment Trade-Offs Reinforce the Need for More Balanced Options

Drug Class	ESTABLISHED CLASSES · MULTIPLE AGENTS, DECADES OF USE		EMERGING
	D ₂ antagonists	D ₂ /D ₃ partial agonists	Muscarinic agonism
Established ✓	Established efficacy foundation for positive symptoms ¹	Introduced efficacy via partial dopamine agonism ³	Demonstrated efficacy through a non-D ₂ pathway ⁴
Trade-Offs ✗	EPS, sedation, weight and metabolic effects, prolactin related AEs, most agents require titration to reach target dose ^{1,2}	Akathisia, restlessness, insomnia, and activation, most agents require titration to reach target dose ³	GI side effects, twice-daily dosing, titration, and meal-timing requirements, requires titration to reach target dose ⁴

Efficacy is the entry ticket. Tolerability is what keeps patients on therapy.

“Historically, our medicines that had the highest efficacy had the highest side effect burden.”⁵
U.S. KOL · 2026



LB-102 May Address Key Treatment Priorities

Dimension	Why It Matters	LB-102 NOVA-1 Profile
Clinical Response	Control of positive symptoms is foundational; inadequate response can lead to treatment change ^{1,2}	✓
Tolerability	Sedation, EPS including akathisia, metabolic effects, prolactin-related effects, and GI symptoms can limit dose optimization and treatment continuity ¹	✓
Simple to Use	Titration, multiple daily doses, and meal-related instructions can add friction at initiation and may delay or prevent patients from reaching and maintaining an optimal effective dose ^{3,4}	✓

CLINICAL RESPONSE · U.S KOL

“So, tolerability looks promising, but you are not compromising on efficacy...**gentle giant.**”⁵

TOLERABILITY · U.S. PSYCHIATRIST

“The Low EPS. Low akathisia. Low weight gain. Lack of sedation. Low GI side effects. **Those are all important.**”⁵

SIMPLE TO USE · U.S. PSYCHIATRIST

“Non-adherence destabilizes the whole picture... and that’s partly motivated by the tolerability and dosing”⁵

U.S. payers cite relapse-related inpatient and emergency care, not pharmacy spend, as the primary cost drivers in schizophrenia

Commercialization Preparation Will Appropriately Scale With Milestones



TODAY

Foundation underway

- Market & competitive insights
- Positioning, evidence & access planning
- Scientific exchange & MSL deployment
- KOL & patient-advocacy engagement
- Lifecycle planning



PHASE 3 TOPLINE

Refine and scale

Data will guide final product positioning, evidence priorities, and the scale of potential launch preparation



POTENTIAL LAUNCH

Prepare for launch

- Commercial execution & field force
- Medical Affairs scale-up
- MSL expansion
- Supply readiness

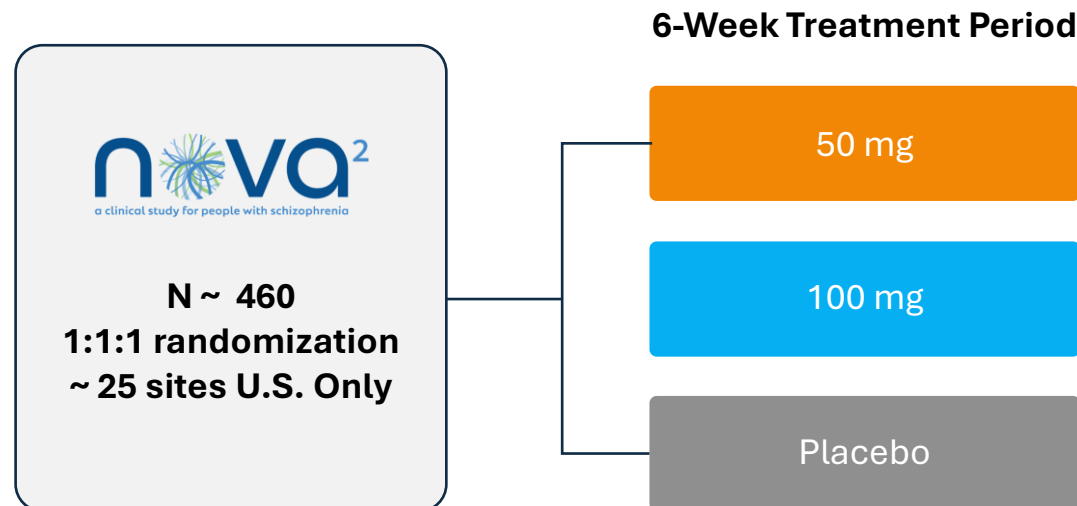
| Heather Turner Chief Executive Officer

| LB-102's Robust Registrational Program is Underway in Schizophrenia

Phase 3 NOVA-2 topline data read out expected in 1H 2027



Phase 3 Trial Design



Primary Endpoint:

PANSS Δ from baseline at day 42 (6 weeks)

Secondary Endpoints:

CGI-S, PANSS positive and negative subscales, Marder factors, cognition, Personal and Social Performance Scale (PSP)

Phase 3 (Inpatient)

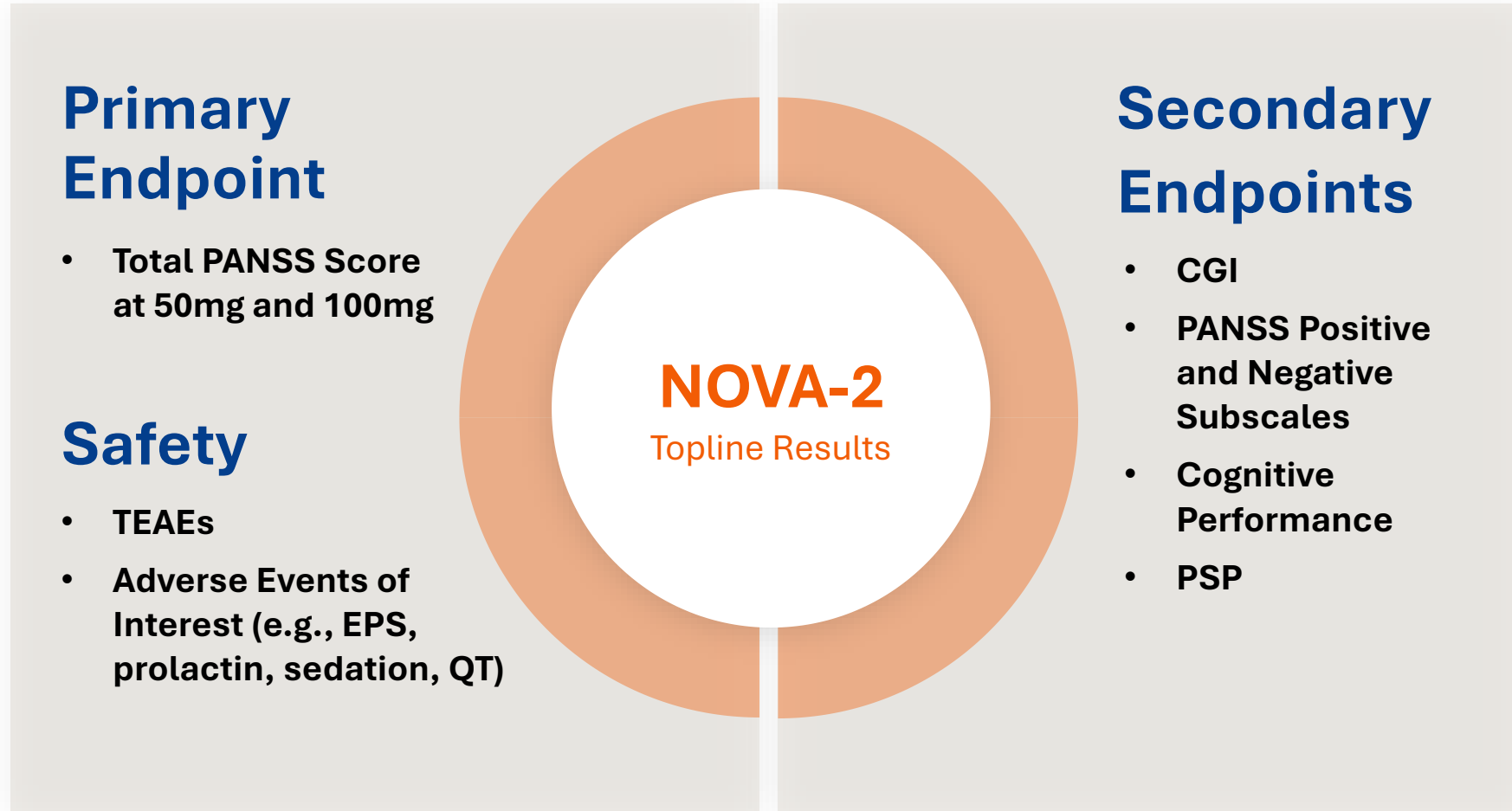
- Low execution risk: Similar in scope to NOVA-1
- Primary changes: 6-week duration; 3 versus 4 arms
- Doses selected to inform use in commercial setting

Open Label Extension (Outpatient)

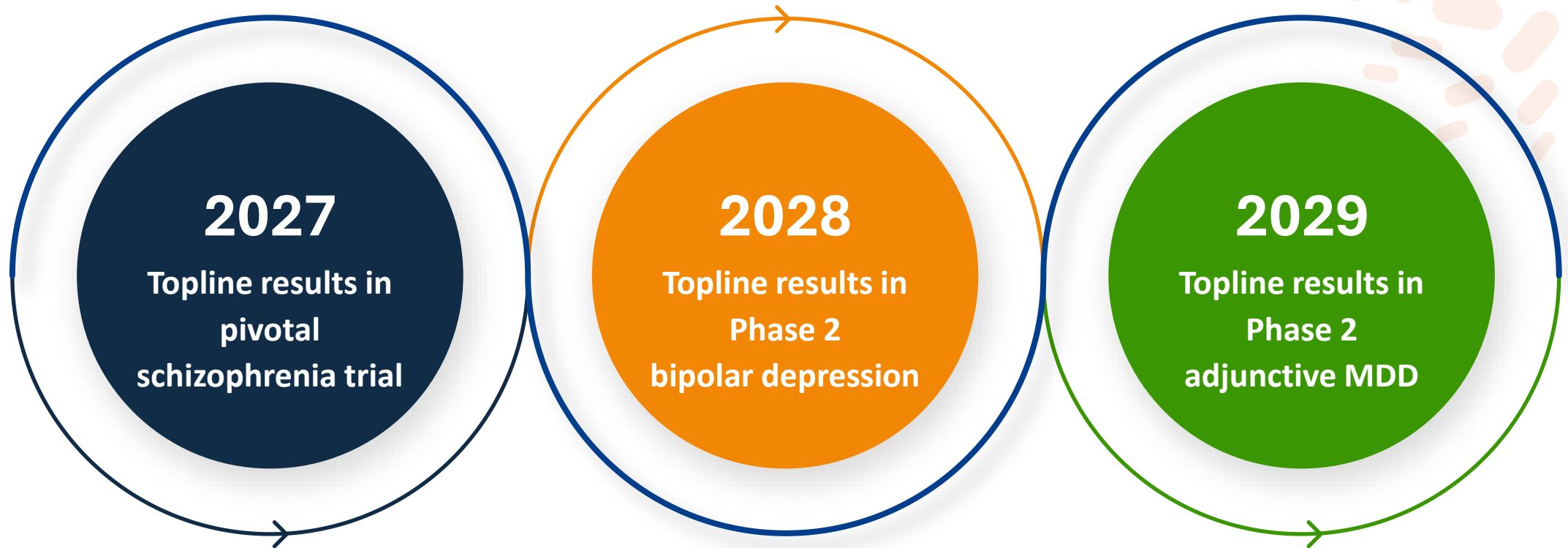
- ~900 patients – rollover from Phase 3 trial + new patients
- Supports accrual of safety database for approval
- Subset analyses: cognition and negative symptoms

| Phase 3 NOVA-2 Trial: Topline Results and Next Steps

Topline disclosure expected to provide fulsome view of clinical profile, pre-NDA meeting guides timing of NDA submission



Looking Ahead: A Series of Potentially Value-Creating Clinical Milestones



All fully funded with cash runway expected beyond Q2 2029

Building a Fully-integrated Company Developing High-Impact Neuromedicines for Real-World Care



Topline data from pivotal NOVA-2 trial expected 1H 2027

Potential for meaningful differentiation on what matters to patients

Expansion potential across psychosis, mood disorders, and LAI

Team of seasoned drug developers and experienced operators in place to scale



| Q&A