Neumora™

Redefining Neuroscience Drug Development

July 2025

Important Disclosures

This presentation contains forward-looking statements about Neumora Therapeutics, Inc. (the “Company,” “we,” “us,” or “our”) within the meaning of the federal securities laws, including statements related to: Neumora’s intention to redefine neuroscience drug development by bringing forward the next generation of novel therapies that offer improved treatment outcomes and quality of life for patients suffering from brain diseases; the timing, progress and plans for its therapeutic development programs, including the timing of patient enrollment, initiation and data read outs for its programs and studies, including for the KOASTAL-2 and -3 studies, and the Phase 1b signal seeking study evaluating NMRA-511, as well as its clinical trial and development plans; timing and expectations related to regulatory filings; expectations and projections regarding future operating results and financial performance, including the sufficiency of its cash resources and expectation of the timing of its cash runway; changes to and optimization of the KOASTAL-2 and -3 studies; NMRA-861 best in class pharmacology and the potential of M4 PAMs; the potential for Neumora to advance other M4 PAMs in its portfolio; expectations regarding appropriate patients being enrolled in the KOASTAL program; intellectual property protection; and other statements identified by words such as “could,” “expects,” “intends,” “may,” “plans,” “potential,” “should,” “will,” “would,” or similar expressions and the negatives of those terms. Other than statements of historical facts, all statements contained in this presentation, are forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995. These statements are subject to risks and uncertainties that could cause the actual results or to be materially different from the information expressed or implied by these forward-looking statements, including, among others: the risks related to the inherent uncertainty of clinical drug development and unpredictability and lengthy process for obtaining regulatory approvals; risks related to the timely initiation and enrollment in our clinical trials; risks related to our reliance on third parties, including contract research organizations; risks related to serious or undesirable side effects of our therapeutic candidates; risks related to our ability to utilize and protect our intellectual property rights; and other matters that could affect sufficiency of capital resources to fund operations. For a detailed discussion of the risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to Neumora’s business in general, please refer to the risk factors identified in the Company’s filings with the Securities and Exchange Commission (SEC), including but not limited to its Quarterly Report on Form 10-Q for the quarter ended March 31, 2025 which was filed with the SEC on May 12, 2025. Forward-looking statements speak only as of the date hereof, and, except as required by law, Neumora undertakes no obligation to update or revise these forward-looking statements.

Neumora™

Our Mission

We are focused on redefining neuroscience drug development by bringing forward the next generation of novel therapies that offer improved treatment outcomes and quality of life for patients suffering from brain diseases

Redefining Neuroscience Drug Development

Industry leading CNS pipeline with long-dated IP into the 2040s

Multiple value-creating clinical catalysts on the horizon

Built at scale with strong balance sheet; $850M raised since 2021

Cash runway into 2027 supporting company growth

World-class team with differentiated approach

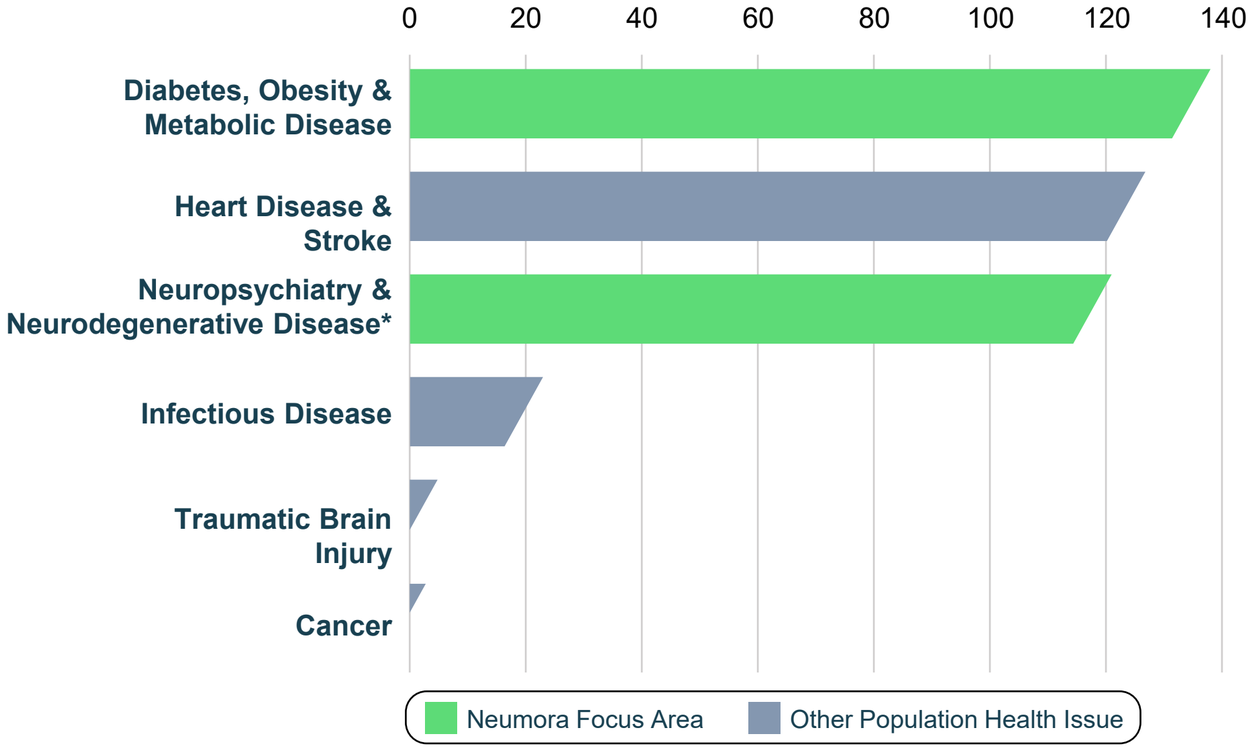
Maximizing probability of success with team and proprietary approach

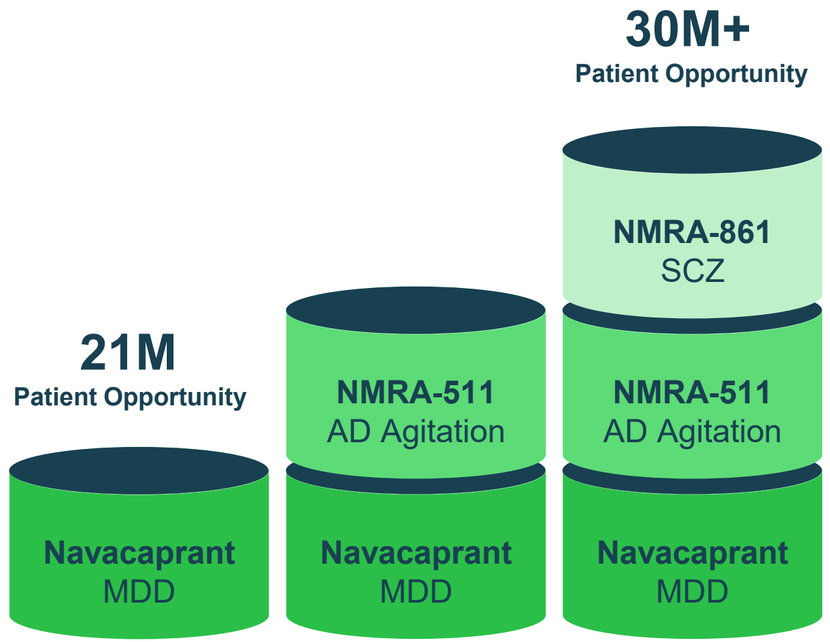
Neumora is Tackling Large Population Health Challenges

Neumora's clinical-stage pipeline has potential to reach up to ~30M+ patients with a robust IP runway into 2041+

Biggest Health Disorders Facing U.S.[1]

Patients Impacted (M)





[1]National Institutes of Health. Our Biggest Health Challenges. Accessed December 2023.
Note: Figure not intended as launch guidance or order. BPD = Bipolar Depression; MDD = major depressive disorder.
*Includes: MDD, BPD, Schizophrenia, Generalized Anxiety Disorder, Post Traumatic Stress Disorder, Substance Use Disorder, Alzheimer's Disease, Parkinson's Disease, Attention-Deficit Hyperactivity Disorder

Advancing a Leading Neuroscience Pipeline

- **Broad pipeline** addressing some of the most prevalent brain diseases
- Targeting novel mechanisms across a **broad range** of neuropsychiatric and neurodegenerative indications

PROGRAM *Target/Mechanism*	**INDICATION** *U.S. Prevalence*	Preclinical	Phase 1	Phase 2	Phase 3	**MILESTONE** *Guidance*
Clinical Programs						
Navacaprant *KOR Antagonist*	**Major Depressive Disorder** *21M*					**KOASTAL-3, -2 topline data** *1Q26, 2Q26*
NMRA-511 *V1aR Antagonist*	**Agitation in Alzheimer's Disease** *6M*					**Phase 1b data** *around year-end 2025*
NMRA-861 *M4 Modulator*	**Schizophrenia** *3M*					**Phase 1 SAD/MAD data** *1Q26*
Pre-Clinical Programs						
NMRA-215 *NLRP3 Inhibitor*	**Obesity/Parkinson's Disease** *103M/1M*					
NMRA-GCASE *GCase Activator*	**Parkinson's Disease** *1M*					
NMRA-NMDA *NMDA Modulator*	**Schizophrenia** *3M*					
NMRA-CK1δ *CK1δ Inhibitor*	**ALS/Alzheimer's Disease** *25K/6M*					

ALS = Amyotrophic lateral sclerosis; CK1 δ = Casein Kinase I Isoform delta; GCase = Glucocerebrosidase; IP = Intellectual Property; KOR = kappa opioid receptor; M4R = Muscarinic Acetylcholine Receptor M4; NLRP3 = Nucleotide-binding Domain, Leucine-rich–containing Family, Pyrin Domain–containing-3; NMDA = N-methyl-D-aspartate; V1aR = Vasopressin 1a Receptor.
**All dates are approximate / estimates / projections only

MDD Represents a Major Population Health Challenge

MDD is the leading cause of disability worldwide[1]

280M

people worldwide have MDD[1]

21M

adults in the U.S. have MDD[2]; the median onset is ~32.5 years of age

30 years

since a novel mechanism of action was approved for MDD

Many people have inadequate response to medication and experience tolerability issues

85%

of patients either don't receive pharmacological treatment or fail to achieve remission with first-line treatment[3–7]

70%

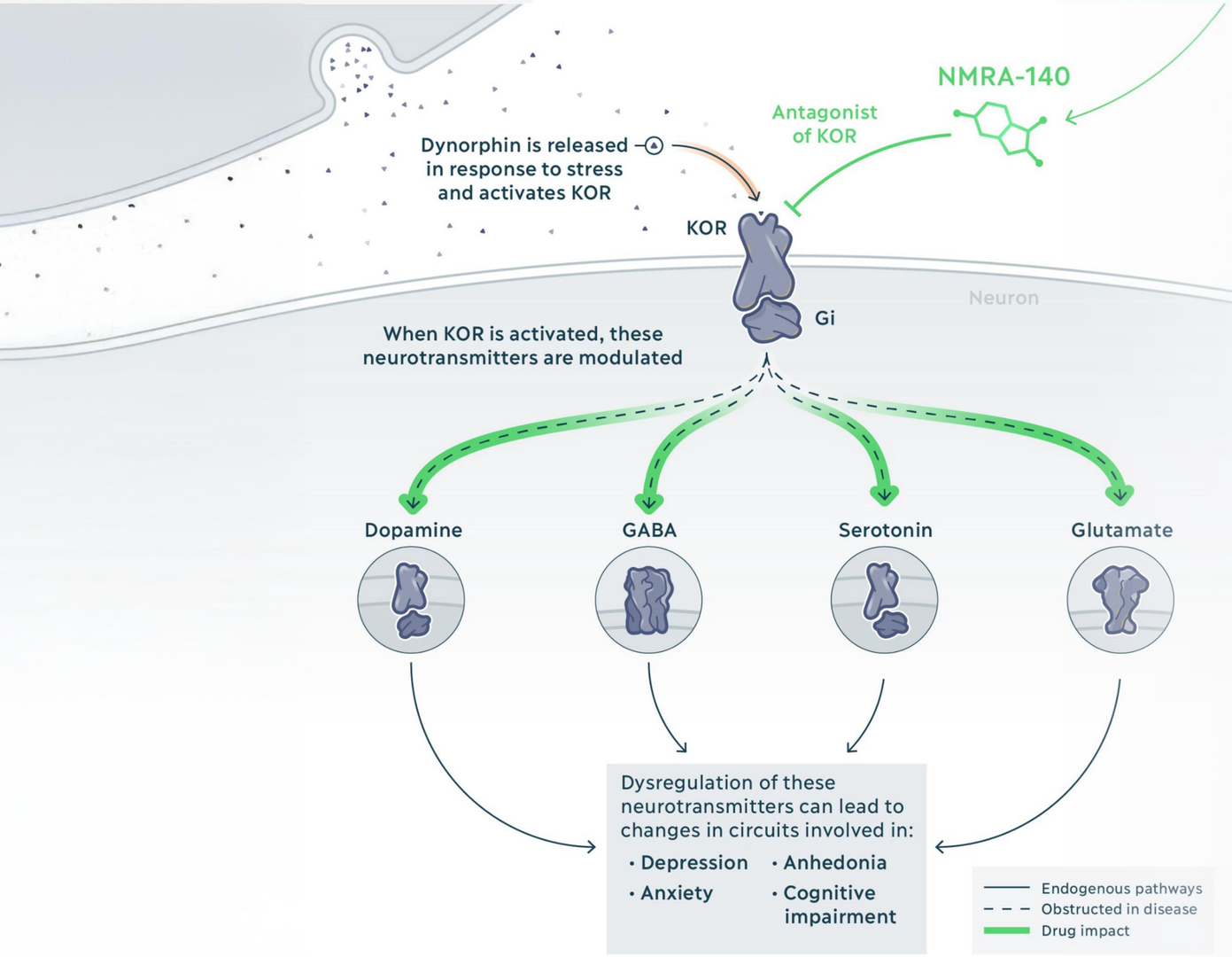
of people with MDD experience anhedonia[8]

60–85%

of patients treated with monotherapy[9]

1. World Health Organization. Depressive disorder (depression), Published March 31, 2023. Accessed June 5, 2024. https://www.who.int/news-room/fact-sheets/detail/depression#:~:text=An%20estimated%203.8%25%20of%20the,world%20have%20depression%20(1). 2. National Institute of Mental Health. Major depression. Published January 2022. Accessed May 9, 2022. https://www.nimh.nih.gov/health/statistics/major-depression. 3. Gaynes BN, et al. Cleve Clin J Med. 2008;75:57-66. 4. Corey-Lisle PK, et al. Arch Intern Med. 2004;164:1197-1204 5. Cartwright C, et al. Patient Prefer Adherence. 2016;10:1401-1407. 6. Ramanuj P, et al. BMJ. 2019;365:l835. 7. Moret C, et al. J Psychopharmacol. 2009;23:967-974. 8. Khazanov GK, et al. Behav Res Ther. 2020;125:103507. 9. Kern et al. Treatment patterns and sequences of pharmacotherapy for patients diagnosed with depression in the United States: 2014 through 2019. BMC Psychiatry. (2020) 20:4.
ADT = antidepressant therapy; AE = adverse events

The Role of Kappa Opioid Receptor Antagonism in MDD



- The **kappa opioid receptor (KOR)** / dynorphin system is a well-characterized pathway, and results from preclinical studies support its potential to modulate depression, anhedonia, and anxiety
- KOR system overactivation in response to stress and mediation of depressive-like symptoms including anhedonia
- KOR antagonism may allow DA and 5HT release to return to adaptive levels during reward processing

Near-term Clinical Development Plan Focused on MDD with Opportunity for Further Expansion

Major Depressive Disorder

KOASTAL

Three replicate, placebo-controlled, double-blind RCTs evaluating efficacy and safety of navacaprant in MDD

KOASTAL-1
Conducted in U.S.
Topline data announced Jan. 2025

KOASTAL-2
Conducted in U.S., Canada and Latin America

KOASTAL-3
Conducted in U.S. and Europe

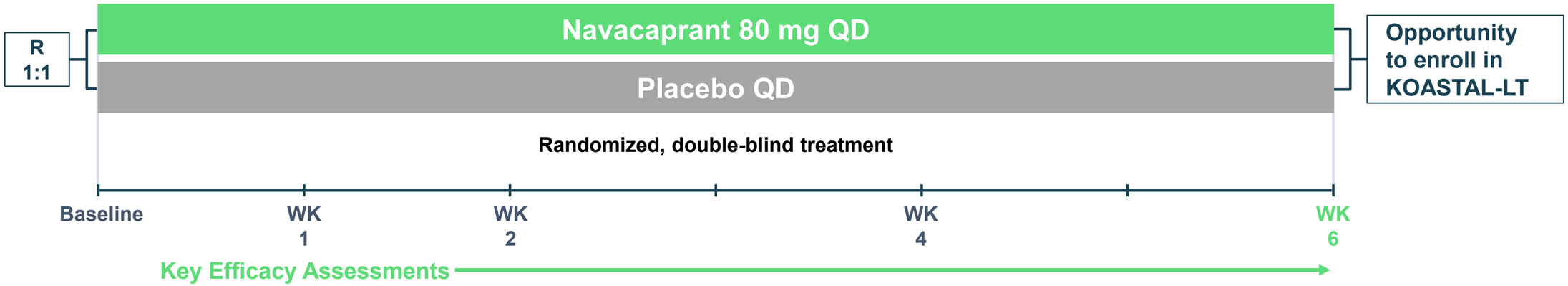
KOASTAL-LT
Open-label extension trial evaluating long-term safety of navacaprant in patients with MDD

Additional indication opportunities include bipolar depression, substance use disorder, ADHD, Generalize Anxiety Disorder and Post-Traumatic Stress Disorder

MDD = Major Depressive Disorder; RCT = Randomized Controlled Trial

KOASTAL Pivotal Study Design

KOASTAL Pivotal Efficacy Studies



KOASTAL-1, KOASTAL-2, KOASTAL-3 Summary			
Inclusion Criteria:	• Adults ages 18 – 65 diagnosed with MDD • MADRS ≥ 25 at baseline	Other Secondary Endpoints Include:	Δ from baseline to each timepoint in: • CGI-S and CGI-I • PHQ-9 • HAM-A • SDS
Primary Endpoint:	• Δ from baseline to Week 6 in MADRS total score		
Key Secondary Endpoint:	• Δ from baseline to Week 6 in SHAPS total score	Key Exploratory Endpoints*:	Δ from baseline to each timepoint in: • EQ-5D 5L • WPAI-GH

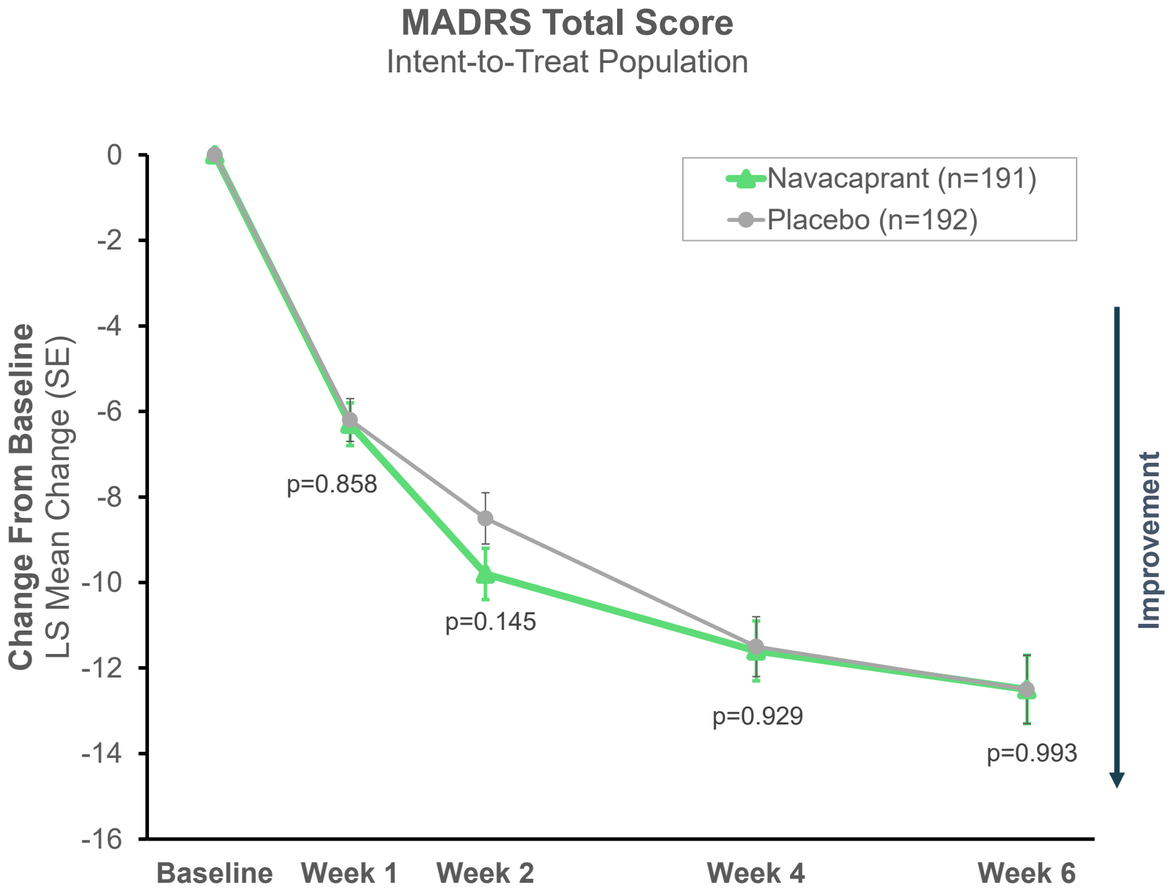
*Safety Assessments include Change in Sexual Functioning Questionnaire (CSFQ-14)
Δ = Change; CGI-I = Clinical Global Impression-Improvement scale; CGI-S = Clinical Global Impression-Severity scale; EQ-5D 5L = EuroQol-5D 5L; HAM-A = Hamilton Anxiety Rating Scale; MADRS = Montgomery-Åsberg Depression Rating Scale; MDD = Major Depressive Disorder; PHQ-9 = Patient Health Questionnaire-9; QD = once daily; SDS = Sheehan Disability Scale; SHAPS = Snaith-Hamilton Pleasure Scale; wk = week; WPAI-GH = Work Productivity and Activity Impairment Questionnaire – General Health.

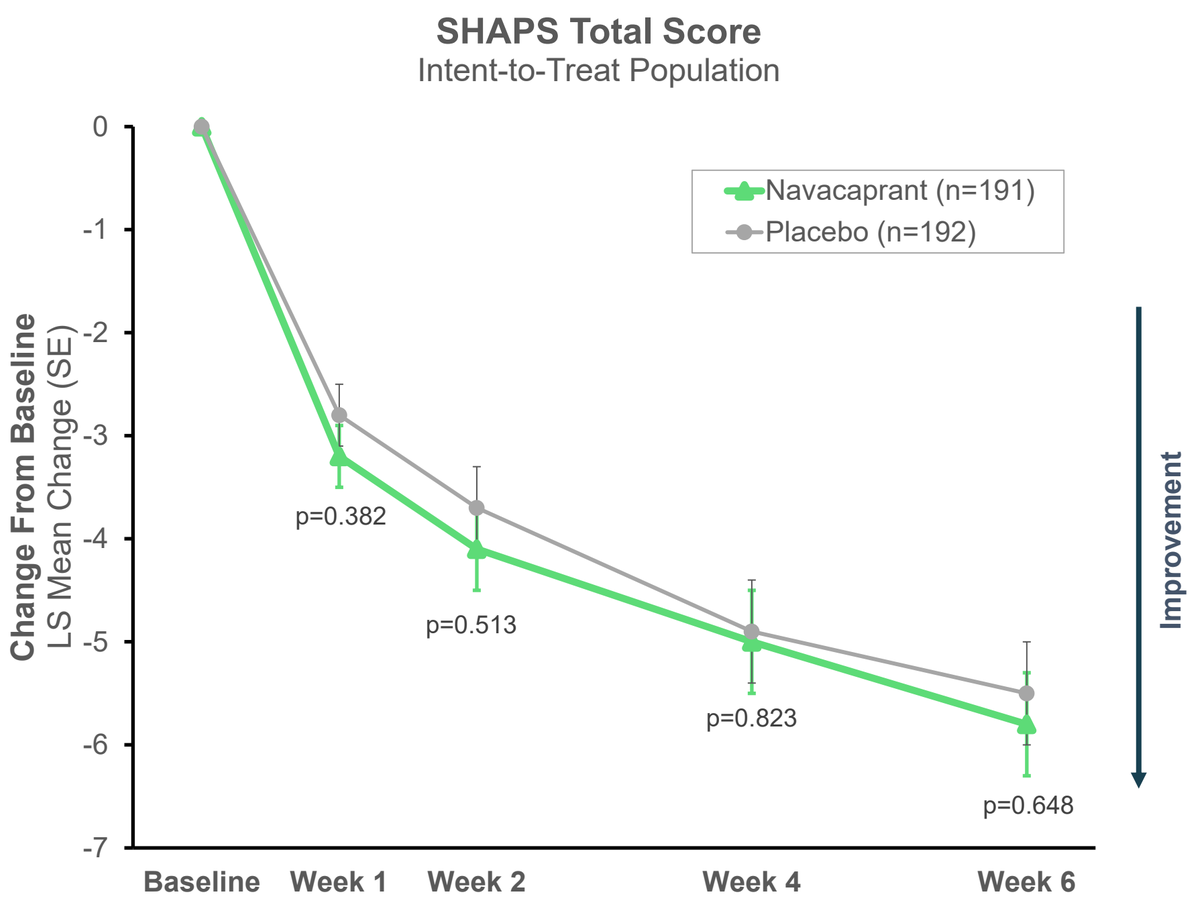
KOASTAL-1 Topline Data: Demographics and Baseline Characteristics

Intent-to-Treat Population	Navacaprant n = 191	Placebo N = 192
Age, mean (SD)	40.7 (14.0)	41.1 (13.2)
Sex, n (%)		
Male	86 (45.0%)	86 (44.8%)
Female	105 (55.0%)	106 (55.2%)
Race, n (%)		
White	112 (58.6%)	127 (66.1%)
Black or African American	38 (19.9%)	31 (16.1%)
Asian	25 (13.1%)	19 (9.9%)
Other	10 (5.2%)	10 (5.2%)
Missing/Unknown	6 (3.1%)	5 (2.6%)
Baseline MADRS total score, mean (SD)	32.2 (4.2)	32.8 (4.7)
Baseline SHAPS total score, mean (SD)	36.2 (6.2)	36.5 (6.7)

MADRS = Montgomery-Åsberg Depression Rating Scale
SHAPS = Snaith-Hamilton Pleasure Scale

KOASTAL-1 Topline Data: Primary & Key Secondary Endpoint





MADRS = Montgomery-Åsberg Depression Rating Scale
SHAPS = Snaith-Hamilton Pleasure Scale

KOASTAL-1 Topline Data: Favorable Safety Profile Demonstrated

Navacaprant was safe and generally well tolerated, with no serious adverse events reported

TEAEs Incidence (>2% in either treatment group)	Placebo n=192	Navacaprant n=191
Preferred Terms	**n (%)**	**n (%)**
Headache	14 (7.3%)	13 (6.8%)
Diarrhea	4 (2.1%)	10 (5.2%)
Nasopharyngitis	8 (4.2%)	7 (3.7%)
Pruritus	4 (2.1%)	7 (3.7%)
Nausea	6 (3.1%)	6 (3.1%)
Constipation	6 (3.1%)	5 (2.6%)
Insomnia	4 (2.1%)	3 (1.6%)
Fatigue	9 (4.7%)	2 (1.0%)
Upper respiratory tract infection	6 (3.1%)	2 (1.0%)
Dizziness	5 (2.6%)	2 (1.0%)
Dry mouth	4 (2.1%)	2 (1.0%)
Somnolence	4 (2.1%)	2 (1.0%)
Urinary tract infection	4 (2.1%)	2 (1.0%)
Back pain	5 (2.6%)	0

- No signal for increased suicidal ideation or suicidal behavior[1]
- Low discontinuation rate due to TEAEs (navacaprant 2.1%; placebo 3.1%)
- 83.3% of navacaprant-treated patients who completed 6 weeks' treatment elected to enroll in KOASTAL-LT

1. As measured by Columbia Suicide Severity Rating Scale (C-SSRS)

Optimizing KOASTAL-2 and -3 Phase 3 Trials

Topline data from KOASTAL-3 in the first quarter of 2026 and -2 in the second quarter of 2026

Site Selection

Adjusted clinical sites included in studies, with goal of including sites with demonstrated expertise in conducting MDD studies

Medical Monitoring

Using clinician-rated Massachusetts General Hospital Clinical Trials Network and Institute SAFER approach to verify the diagnosis and appropriateness of patient population

Screening Tools

Verified Clinical Trial (VCT) screening database complements the Clinical Trial Subject (CTS) database to screen for people who participate in multiple clinical trials

Target Enrollment

Option included in KOASTAL-2 and -3 protocols to overenroll the studies up to 25%

MDD = major depressive disorder

NMRA-511 is a Best-in-Class Vasopressin 1a Receptor Antagonist with Broad Potential Across Neuropsychiatric Disorders

Rationale

Vasopressin plays a role in the regulation of aggression, affiliation, stress and anxiety response

Indication

Agitation in Alzheimer's disease

Status

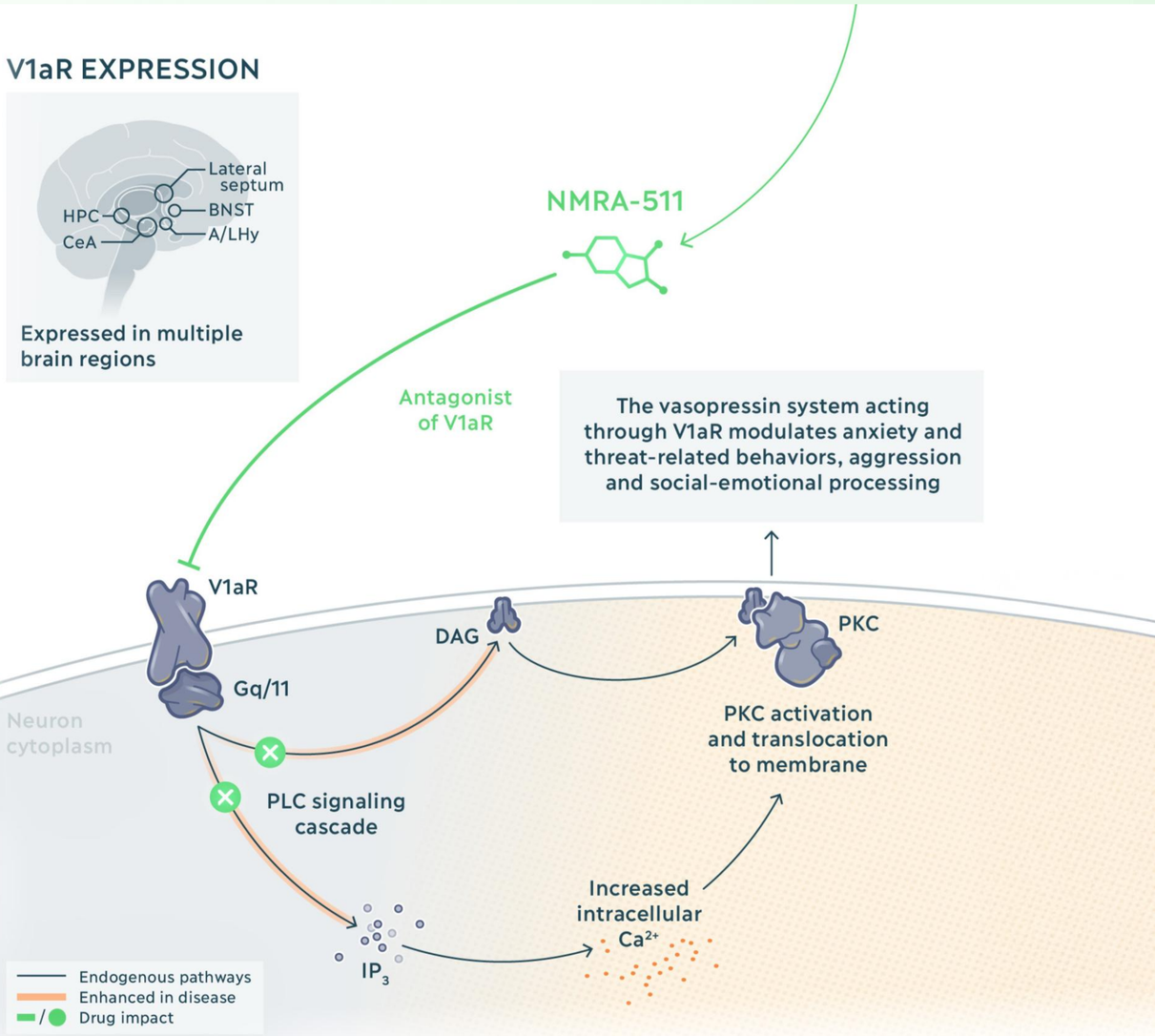
Phase 1b study underway with data anticipated around the end of 2025

Drug Profile

Oral, BID dosing

Strong IP Protection

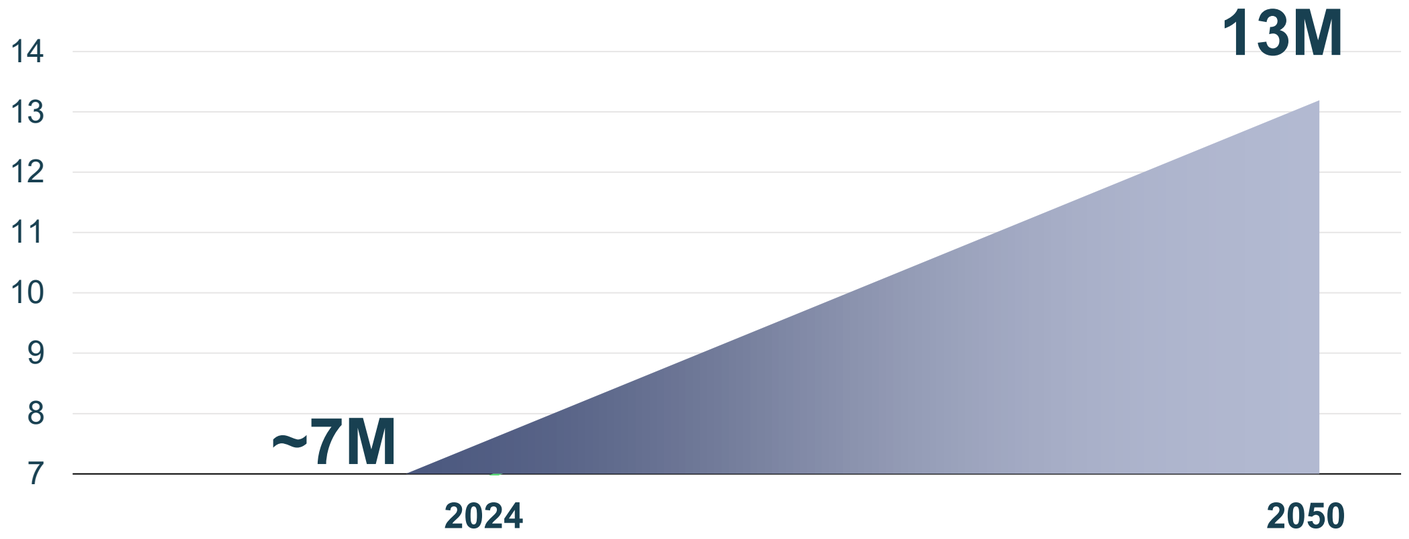
Expect exclusivity through 2042+, based on composition of matter protection and estimated patent term extension



Alzheimer's Disease Agitation Represents Large Market Opportunity with Significant Unmet Need

Agitation in Alzheimer's disease impacts a significant portion of the U.S. population; that number is expected to increase as the population ages[1]

U.S. Adults with Alzheimer's Disease (M)[1]



>70%

of people with AD experience agitation at some point in their disease[2]

Significant unmet medical need exists in this population[3]

Agitation is among the most disruptive symptoms of AD. It is associated with greater caregiver stress, increased morbidity and mortality and earlier placement in long-term care facilities. The only currently approved product carries a black-box warning for mortality in elderly people.

[1]Alzheimer's Association. Alzheimer's Disease Facts and Figures. May 2024. [2] Ijaopo et al., 2017., Translational Psychiatry.; [3]Koenig et al., 2016, Current Psychiatry.

Several Lines of Evidence Indicate that V1a Receptor Antagonists Have Therapeutic Potential for Reducing Symptoms of Agitation

The vasopressin system modulates social-emotional, anxiety and threat-related behaviors across species

- V1aR expression patterns critically affect social behavior[1-5]
- Rodent selection lines bred for aggression or anxiety show dysregulated vasopressin release and HPA axis functioning[6]
- Vasopressin-deficient rodents display impaired responses to threat stimuli, reduced anxiety and depressive-like behaviors, and impaired aggression toward intruders[7-9]

In healthy volunteers, vasopressin enhances reactivity to threatening stimuli and disrupts emotional control[1-2]

- Exogenously administered vasopressin increases autonomic responsiveness to threat stimuli and increases anxiety[2]
- V1a antagonist administration suppresses anxiety induced by unpredictable threats[10]

Positive association between vasopressin and aggression in people with personality disorders[11]

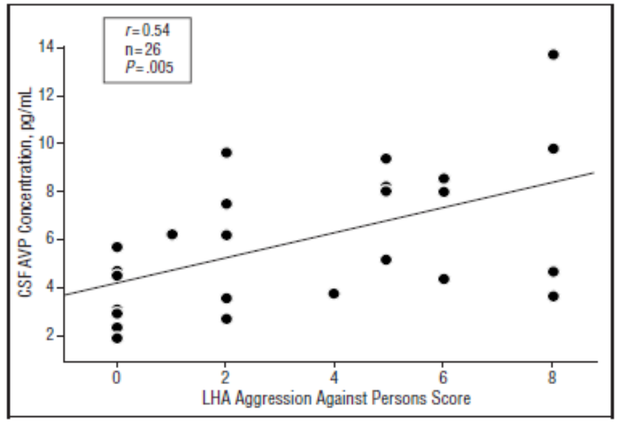


Figure 1. *Correlation between Aggression Against Persons (the fighting and assault items) scores on the Life History of Aggression (LHA) assessment and cerebrospinal fluid (CSF) arginine vasopression (AVP) concentrations in 26 individuals who met the* DSM-IV *criteria for personality disorder.*

In HD irritability, an investigational V1a receptor antagonist reduced an exploratory endpoint measuring aggression[12]

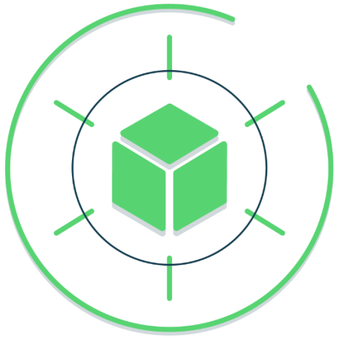
Together, these data support the development of a V1a receptor antagonist for the treatment of symptoms of agitation, aggression, and anxiety

[1]Ebstein et al., 2009, New York Academy of Sciences.; [2]Thompson et al., 2006, PNAS.; [3]Insel et al., 2010, *Neuron Review*, PNAS; [4]Carter et al., 1995, Neuroscience Biobehavioral Review.; [5]Wang et al., 1994, PNAS.; [6]Veenema and Neumann, 2007, Brain behavior, evolution.; [7]Zelena et al., 2009, Journal of Endocrinology.; [8]Mlynarik et al., 2007, Hormones and Behavior.; [9]Fodor et al., 2014, Psychoneuroendocrine.; [10]Lago et al., 2021, Psychopharmacology.; [11]Coccaro et al., 1998., JAMA Psychiatry.; [12]Maibach et al., 2022, Personalized Medicine.
HPA = hypothalamic-pituitary-adrenal

NMRA-511 Profile Supports Advancement into Alzheimer's Disease Agitation

Best-in-Class Pharmacology[1]

- Highly potent at V1a
- High selectivity over V1b, V2, and oxytocin receptors
- Excellent brain penetration



Strong Pre-Clinical Data[2]

- Robust pharmacodynamic (PD) effect in rodents
- Robust activity in a marmoset 'human threat test' model of stress/anxiety

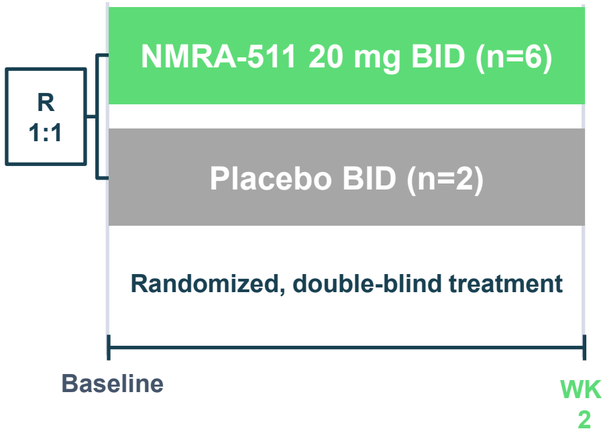
PK and Safety Data from Phase 1 Support Advancement[1]

- NMRA-511 was safe and very well-tolerated in Phase 1 SAD/MAD study
- NMRA-511 was safe and well-tolerated in healthy elderly volunteers

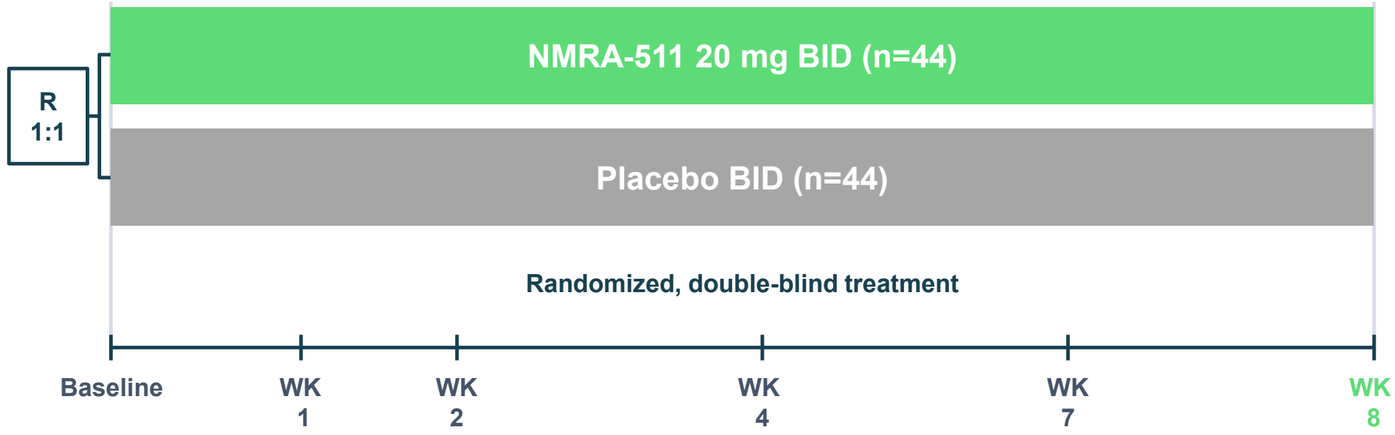
[1]NMRA Data on File. [2]Wallace et al., 2022., Presented at the American College of Neuropsychopharmacology 2022 Annual Congress.

NMRA-511 Signal Seeking Study in Alzheimer's Disease Agitation

Part A: 2-Week Evaluation Period Enrolling Healthy Elderly Participants



Part B: 8-Week Evaluation Period Enrolling People with Alzheimer's Disease Agitation (ADA)



NMRA-511 Phase 1b Study	
Part A Inclusion Criteria:	• Healthy elderly adult participants aged 65-80 years
Part B Inclusion Criteria:	• Adults aged 55-90 years with mild-severe dementia (MMSE score of 5-24) and clinically significant agitation (CMAI total score 45-100)
Part B Primary Endpoint:	• Δ from baseline to Week 8 in CMAI total score
Part B Other Endpoints Include*:	Δ from baseline to Week 8 in: • CGI-S Agitation total score • mADCS-CGIC total score • Caregiver Diary of participant agitation, aggression, and/or anxious behaviors • NPI total score
Statistics:	• Study not powered to demonstrate statistical significance • Designed as a signal-seeking study; effect size will inform the potential future development of NMRA-511 in ADA

*Safety Assessments include adverse events, clinical laboratory, vital signs, physical examination, 12-lead electocardiogram (ECG), Columbia-Suicide Severity Rating Scale (C-SSRS).
Δ = Change; BID = twice daily; CMAI = Cohen-Mansfield Agitation Inventory; MMSE =Mini-Mental State Examinations; CGI = Clinical Global Impression of Change for Agitation; mADCS-CGIC = mADCS-CGIC Agitation modified Alzheimer's Disease Cooperative Study – Clinical Global Impression of Change for Agitation; NPI = Neuropsychiatric Inventory.

M4 PAM Franchise: Differentiated M4R PAMs for Schizophrenia

NMRA-861 Target Profile

Pharmacology

Neumora has multiple series of chemically distinct, highly selective M4 muscarinic receptor PAMs, including NMRA-861, designed for antipsychotic-like efficacy with the potential for improved tolerability profile

Indication

Schizophrenia

Target Administration

Oral, once-daily

IP

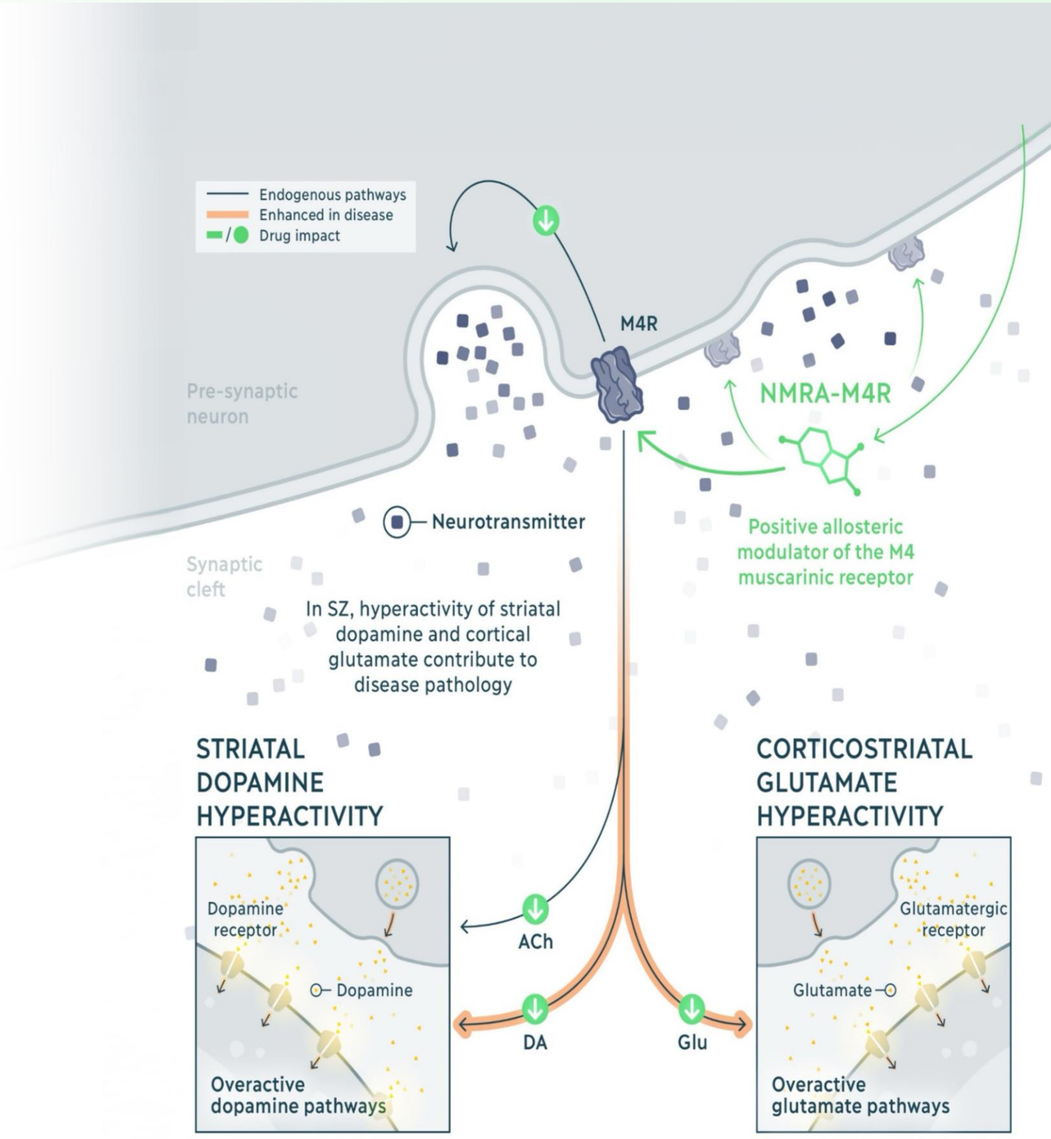
Composition of matter patent extending to 2044+*

Epidemiology

Estimated 3 million patients in the U.S. with schizophrenia[1]

Expected Milestones

Report Phase 1 SAD/MAD data for NMRA-861 in the first quarter of 2026

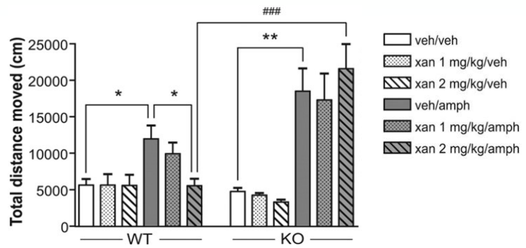


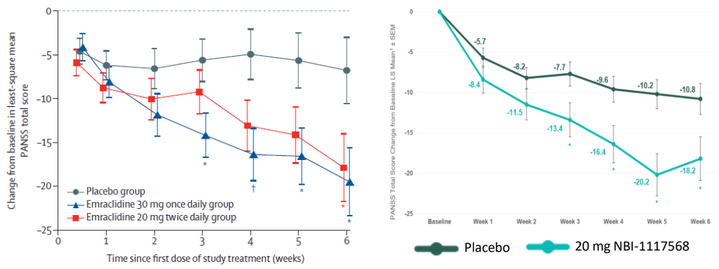
[1]Wander, C. *Am J Manag Care*. 2020;26:S62-S68.
*Excluding any patent term adjustment or extension
PAM = positive allosteric modulator

An Optimized Muscarinic Drug Profile Would Include Selectivity and Potency in the CNS

Preclinical data and clinical data in acute schizophrenia supports M4 as a driver of antipsychotic activity

Activity of xanomeline (active component of Cobenfy™) is dependent on M4R in mice





Non-selective muscarinic agents are associated with a range of peripheral AEs

M4

Cardiovascular

Transient increased BP & heart rate

M1, M2, M3

GI Tract

Increased gastric secretion & gastric motility

M1, M2, M3

Cardiovascular

Direct effect on cardiac function – increased BP & heart rate

M1, M3

Glands

Increased salivation
Increased lacrimation
Increased sweating

PAMs offer the benefits of greater selectivity

- Targeting the allosteric site specifically allows for greater selectivity for M4 over other muscarinic sub-types than if targeting the orthosteric site due to binding site conservation
- To date the pharmacology of agonists targeting the orthosteric site are often thought to display ‘partial’ agonism which could contribute to variable clinical responses
- PAMs allow for more precise potentiation of M4, maintaining the spatial and temporal signaling dynamics of ACh

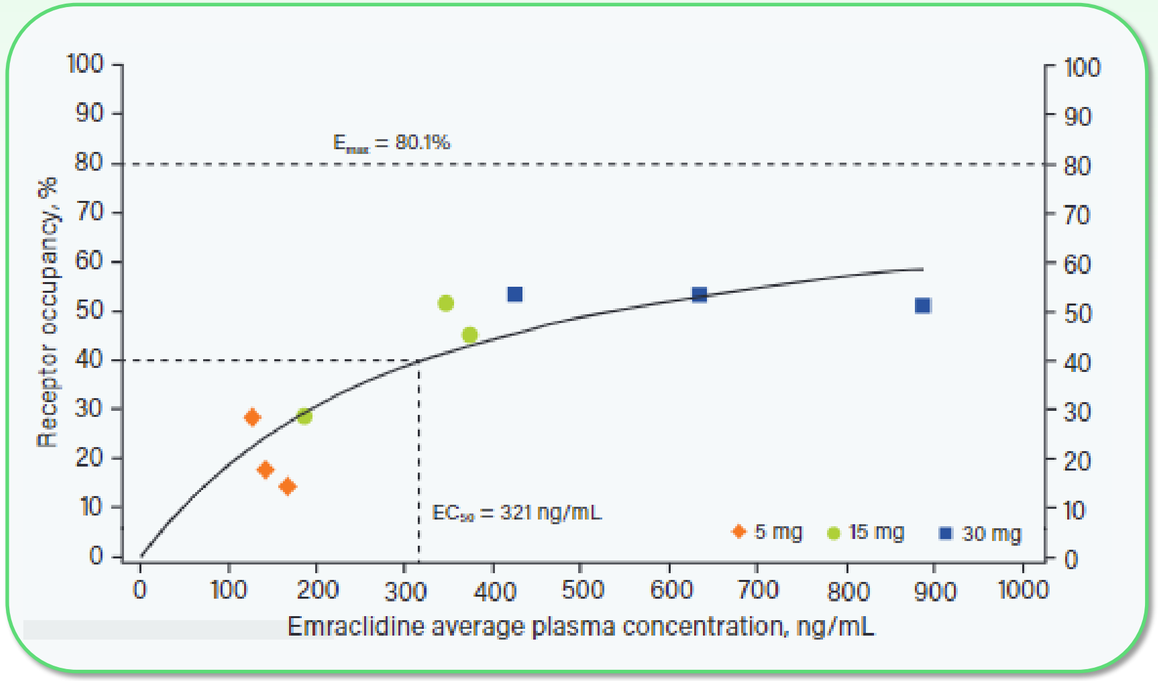
1. Digby GJ, et al. *J Neurosci*. 2012;32(25):8532-44. 2. Dencker D, et al. *J Neurosci*. 2011 April 20;31(16):5905-8. 3. Krystal JH, et al. *Lancet*. 2022 Dec 17;400(10369):2210-20. 4. Neurocrine Biosciences. Q1 Earnings Presentation. April 14, 2025. www.neurocrine.com/documents/86/NBIX_Q1_2025_Earnings_Presentation_Final_05.05.25.pdf. 5. Moran SP, et al. *Trends Pharmacol Sci*. 2019 Dec;40(12):1006-20. 6. Tobin AB. *Nat Rev Drug Discov*. 2024 Oct,23(10);743-58. 7. Paul SM, et al. *Biol Psychiatry*. 2024 Oct 15;96(8):627-37.

Emraclidine Receptor Occupancy Disconnected from Plasma Exposures

Receptor Occupancy for Emraclidine in Humans Suggests the Compound has Limited Brain Exposure

- In a human PET study, peripheral concentration of emraclidine shows dose linear response
- However, CNS receptor occupancy unchanged when dose doubled from 15 to 30 mg
- Data suggests emraclidine may have limitations in engaging the M4 receptor in the brain

Low CNS exposure could limit efficacy



CNS = central nervous system; PET = positron emission tomography

Duvvuri S, et al. *Evaluation of M4 Muscarinic Receptor Occupancy by Emraclidine Using [^{11}C]MK-6884 PET in Healthy Volunteers*. Poster M206. Presented at the 62nd Annual Meeting of the American College of Neuropsychopharmacology, Tampa, FL: December 3 – 6, 2023.

NMRA-861 Has Potential Best-in-Class Potency and Optimized Brain Penetration

NMRA-861 and –M4R potentially more potent than emraclidine across multiple assays

NMRA-861 and –M4R are selective for M4 over other muscarinic receptor subtypes

Neumora M4 PAMs are optimized for high CNS exposure

Neumora M4 PAMs are optimized for once daily dosing

Convulsions have not been observed with NMRA-861 or –M4R

	NMRA-861[1]	NMRA-M4R[1]	Emraclidine
M4 EC_{50} *(human; cAMP)*[1]	6 nM	13 nM	26 nM
M4 EC_{50} *(human; Ca^{2+})*[1]	2 nM	8 nM	180 nM
Selectivity at other muscarinic receptor subtypes *(EC_{50})*[1]	M1, M3, M5 > 10 µM, M2 0.7 µM	M1, M2, M3, M5 > 10 µM	M1, M3, M5 > 10 µM, M2 5.7 µM
Brain penetration potential *(P-gp A:B ratio)*[1,2]	High (1.26)	High (0.93)	Moderate (3, 6.02)[1,2]
Human half-life[3]	Pending Phase 1 Study	Pending Phase 1 Study	9 – 12 hr
Preclinical convulsions	Not observed in rat, dog or rabbit	Not observed in rat, dog or rabbit	Unknown

NMRA-861 has potential best in class pharmacology and clinical differentiation

Note: Data on this slide is presented for illustrative purposes only. These molecules have not been studied in head-to-head clinical trials.

cAMP = cyclic adenosine monophosphate; CNS = central nervous system; PAM = positive allosteric modulator

1. Data generated by The Warren Center for Neuroscience Drug Discovery at Vanderbilt University on behalf of Neumora across NMRA-861, NMRA-M4R and emraclidine. 2. Butler CR, et al. *J Med Chem.* 2024 Jul 11;67(13):10831-47. 3. Krystal JH, et al. *Lancet.* 2022 Dec 17;400(10369):2210-20.

SAD/MAD Evaluating NMRA-861 in Healthy Adults and People with Stable Schizophrenia

Study Objectives

- Confirm once-daily dosing – based on PK profile in humans
- Evaluate tolerable doses in people with stable schizophrenia
- Establish CNS penetration – based on CSF exposure

SAD – Part 1 CSP

	Dose Cohorts[a]	Participants	Randomization
Part 1A	5 mg, 15 mg, 45mg, 80 mg, final dose TBD[b]	Healthy adults	6:2 active:placebo
Part 1B (Fed-Fasted cohort)	Dose to be determined	Healthy adults	12 active

MAD – Part 2 CSP

	Dose[c]	Participants	Randomization
Cohort 1	15 mg QD	Healthy adults	6:2 active:placebo
Cohort 2	45 mg QD	Healthy adults	
Cohort 3	Dose to be determined	Healthy adults OR with stable schizophrenia[d]	
Cohort 4	Dose to be determined	Healthy adults OR with stable schizophrenia[d]	
Cohort 5	Dose to be determined	Adults with stable schizophrenia	

Healthy adults Adults with stable schizophrenia

[a]Sentinel dosing done in each cohort. [b]Additional optional cohort dose to be decided. [c]Sentinal dosing only if recommended by SRC. [d]Participants enrolled into flexible cohorts will be determined by SRC recommendation.

Pre-Clinical Pipeline of Four Novel Programs, Each with A Strong Biological Rationale

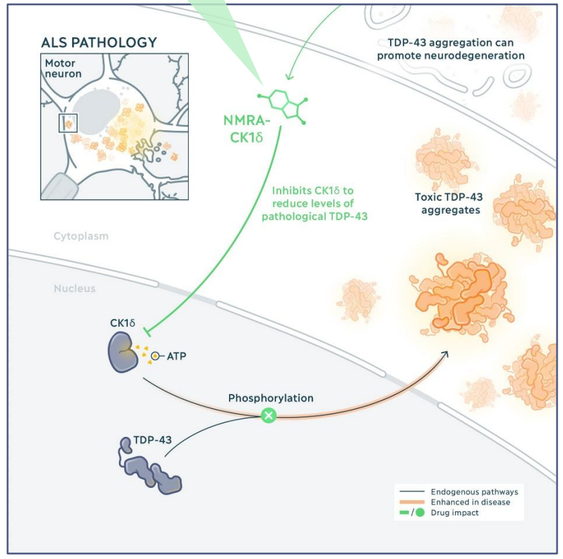
NMRA-CK1δ

Focused on inhibiting the protein casein kinase-1δ (CK1δ) to reduce levels of the pathological form of TDP-43 and slow disease progression in ALS

Potential Indications

ALS, Alzheimer's disease

CK1δ phosphorylates TDP-43, a key driver of TDP-43-driven pathology in ALS



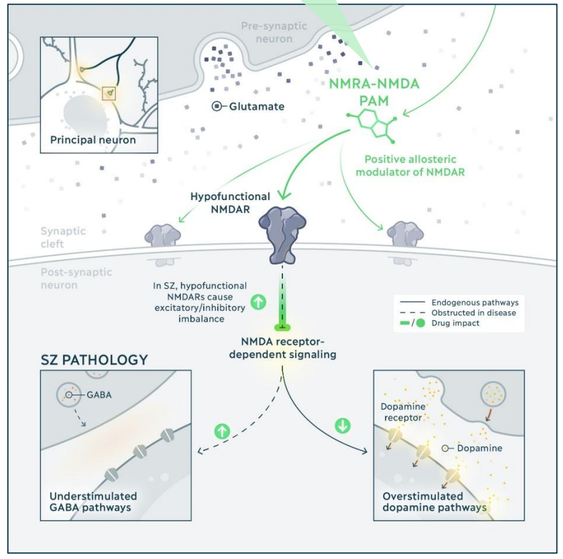
NMRA-NMDA

NMDA receptor hypofunction is a leading hypothesis for the cause of schizophrenia.

Potential Indications

SCZ

NMDA PAMs can selectively enhance physiological NMDAR function and decrease network hypersynchrony observed in SCZ



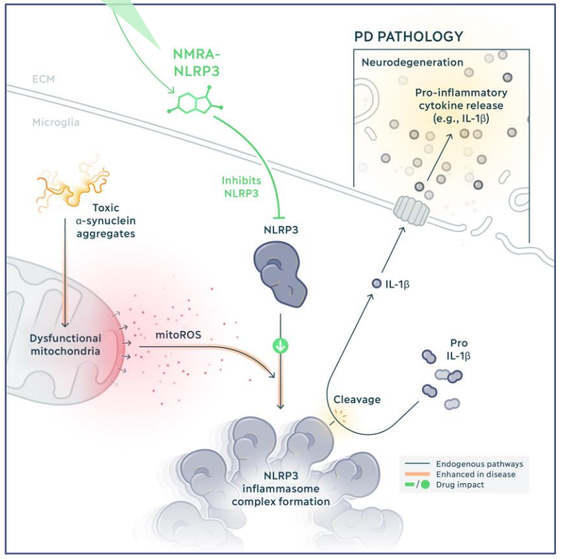
NMRA-NLRP3

Focused on inhibiting the NLRP3 inflammasome to modulate the immune response in neurodegeneration

Potential Indications

Obesity, Parkinson's disease

NLRP3 inflammasome is activated in microglia in response to disease linked proteins such as α-synuclein, leading to proinflammatory signaling



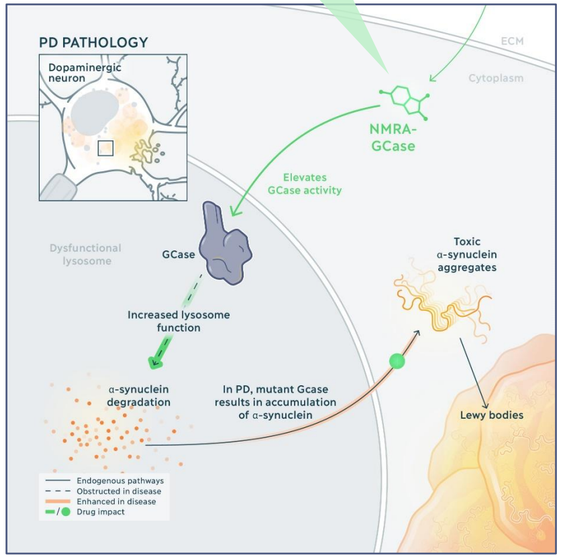
NMRA-GCASE

Focused on elevating activity of the GCase enzyme, which is encoded by the GBA1 gene, and may help to degrade toxic a-synuclein aggregates

Potential Indications

Parkinson's disease

GCase deficiencies lead to lysosomal dysfunction and the accumulation of alpha-synuclein, a hallmark of Parkinson's



PAM = positive allosteric modulator; SCZ = schizophrenia; ALS = Amyotrophic lateral sclerosis; CK1 δ = Casein Kinase

Redefining Neuroscience Drug Development

Industry leading CNS pipeline with long-dated IP into the 2040s

Multiple value-creating clinical catalysts on the horizon

Built at scale with strong balance sheet; $850M raised since 2021

Cash runway into 2027 supporting company growth

World-class team with differentiated approach

Maximizing probability of success with team and proprietary approach

Appendix

Led by Experienced Company Builders and Leading Neuroscience Drug Developers

Leadership

Paul L. Berns
Co-Founder, Chief Executive Officer & Chairman of Board of Directors

Carol Suh
Chief Strategy Officer & Co-Founder

Michael Milligan
Chief Financial Officer

Amy Sullivan
Chief Human Resources Officer

Joshua Pinto, Ph.D.
President

Jason Duncan
Chief Legal & Administrative Officer

Lori Houle
Chief Technical Operations & Quality Officer

Pablo Gersberg
Chief Information Officer

Bill Aurora, Pharm.D.
Chief Operating & Development Officer

Nick Brandon, Ph.D.
Chief Scientific Officer

Mary Chamberlain-Tharp, Ph.D.
Chief Business Officer

Board of Directors

Paul L. Berns
Co-Founder, Chief Executive Officer, Chairman

Kristina Burow
Managing Director, ARCH Venture Partners

Matthew K. Fust
Biotechnology Advisor

Alaa Halawa
Executive Director, Mubadala Capital

Maykin Ho, Ph.D.
Retired Partner, Goldman Sachs

David Piacquad
Biotechnology Advisor

KOASTAL-1 Topline Study Summary Results

The KOASTAL-1 study enrolled 383 adult patients with Major Depressive Disorder (MDD)

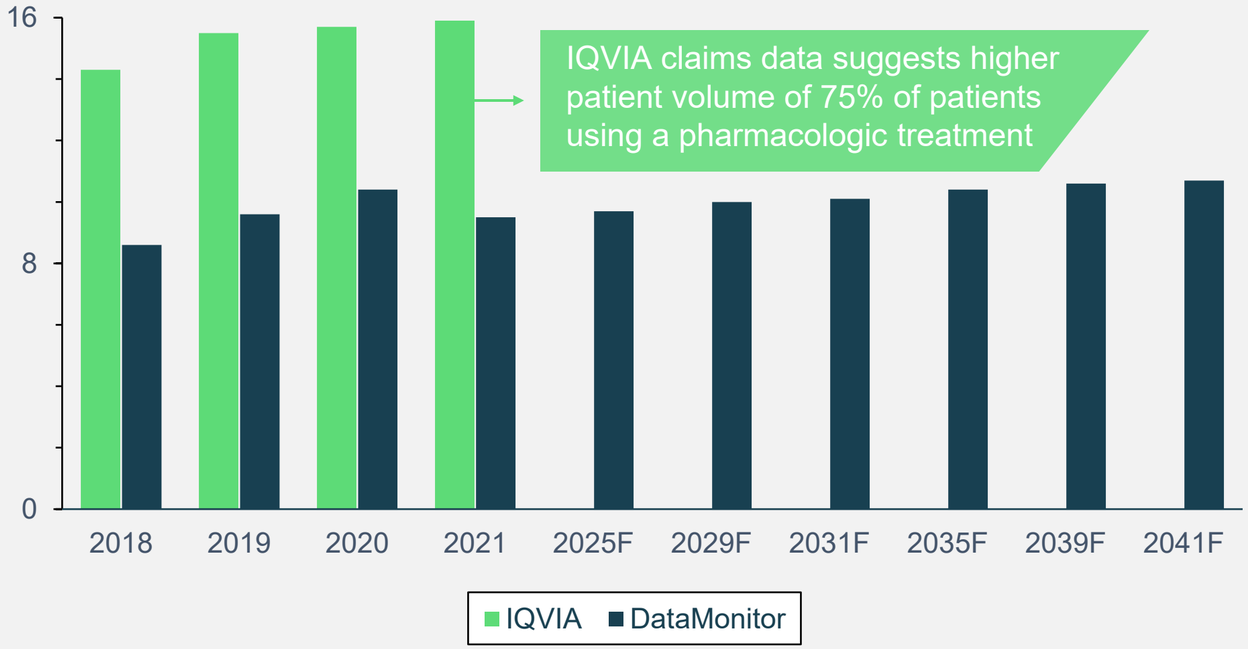
	MADRS Total Score			SHAPS Total Score		
Outcome	**Navacaprant 80 mg**	**Placebo**	**LSMD**	**Navacaprant 80 mg**	**Placebo**	**LSMD**
ITT population CFB at Week 6 (Primary Endpoint)	-12.5 (n = 191)	-12.5 (n = 192)	0.0 (p = 0.993)	-5.8 (n = 191)	-5.5 (n = 192)	-0.3 (p = 0.648)
Female population CFB at Week 6	-14.0 (n = 105)	-11.4 (n = 106)	-2.7 (p = 0.072)	-7.2 (n = 105)	-4.9 (n = 106)	-2.3 (p = 0.015)
Male population CFB at Week 6	-10.6 (n = 86)	-13.8 (n = 86)	3.2 --	-4.3 (n = 86)	-6.3 (n = 86)	2.0 --

CFB = change from baseline; LSMD = difference in LS mean change from baseline between navacaprant and placebo groups generated from mixed-effects model for repeated measures. Subgroup analysis for male or female are pre-specified.

Navacaprant Would Enter Large MDD Market with a Highly Differentiated Profile

GROWTH IN ADDRESSABLE MDD MARKET EXPECTED IN-LINE WITH POPULATION GROWTH

U.S. MDD diagnosed, pharmacologically treated prevalent population (2018-41F) Millions of people



60-80% of MDD patients across lines of therapy are treated with a monotherapy agent[1]

Monotherapy treatment rates across lines of therapy

Treatment Line	CCAE	MDCD	MDCR	Optum
1st	79.6%	82.1%	84.6%	81.7%
2nd	67.3%	67.8%	69.3%	66.1%
3rd	63.9%	64.9%	67.2%	62.1%
4th	61.4%	61.4%	68.1%	60.0%

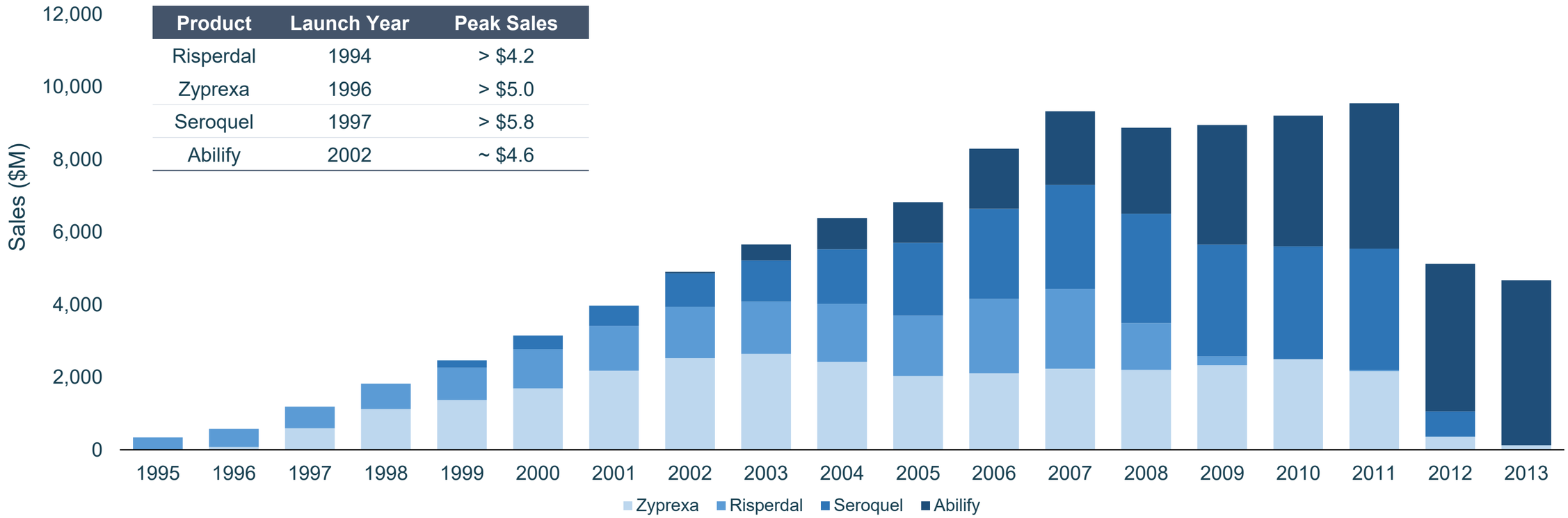
[1]Kern et al. Treatment patterns and sequences of pharmacotherapy for patients diagnosed with depression in the United States: 2014 through 2019. BMC Psychiatry. (2020) 20:4.
U.S. Census Population Projections; DRG; Datamonitor; National Survey of Drug Use and Health 2018, 2019, 2020, 2021; Torre et al. (2021); L.E.K. research and analysis
CCAE = IBM MarketScan Commercial Database; MDCD = IBM Market Scan Multi-State Database; MDCR = IBM MarketScan Medicare Supplemental Database

Schizophrenia Market Supports Multiple Treatment Options

Historically the schizophrenia market has supported multiple branded products with similar MOAs, with new entrants driving higher overall market sales volume

Sales of Branded 5-HT2 to D2 Receptor Antagonists (1995 – 2013)

Product	Launch Year	Peak Sales
Risperdal	1994	> $4.2
Zyprexa	1996	> $5.0
Seroquel	1997	> $5.8
Abilify	2002	~ $4.6



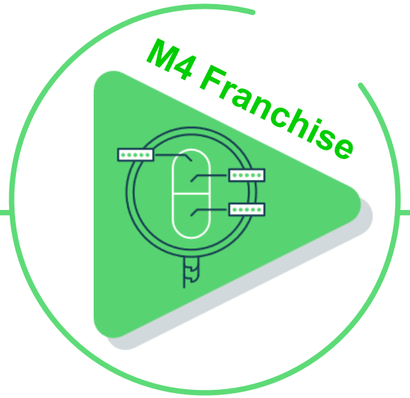
Sources: EvaluatePharma, L.E.K. interviews, research, and analysis; GK associates “The order of entry effect in prescription (Rx) and over the counter (OTC) pharmaceutical drugs”, International Journal of Pharmaceutical and Healthcare, Marketing Vol. 2 No. 1, 2008 pp. 35 46. MOA = Mechanism of Action.

Opportunity to Build a Leading Neuropsych Product Franchise

Potential for **broad indication expansion** in poorly served disorders with a **novel mechanism**

Schizophrenia Opportunities

- Acute Schizophrenia Monotherapy
- Acute Schizophrenia Adjunctive
- Residual Negative Symptoms Adjunctive
- Cognition in Schizophrenia



Additional Opportunities

- Bipolar Depression
- Behavioral Disturbances in Parkinson's Disease and Alzheimer's Disease
- Huntington's Disease
- Cognition

Neumora™