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Cognitive Impairment Associated with Schizophrenia – a KOL webinar

January 2022

Disclaimer

This document contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. The words "believe," "may," "will," "estimate," "continue," "anticipate," "intend," "expect," "initiate," "could," "would," "project," "plan," "potentially," "preliminary," "likely," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these words. Forward-looking statements include express or implied statements relating to, among other things: continued investor confidence in atai's platform and its potential to serve the growing mental health crisis; the success, cost and timing of development of our product candidates; and our business strategy and plans. The forward-looking statements in this press release are neither promises nor guarantees, and you should not place undue reliance on these forward-looking statements because they involve known and unknown risks, uncertainties, and other factors, many of which are beyond our control and which could cause actual results, levels of activity, performance or achievements to differ materially from those expressed or implied by these forward-looking statements.

We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy, short-term and long-term business operations and objectives, and financial needs. These forward-looking statements are subject to a number of risks, uncertainties, and assumptions, including without limitation the important factors described in the section titled "Risk Factors" in our final prospectus, dated June 17, 2021, filed with the Securities and Exchange Commission ("SEC") pursuant to Rule 424(b) under the Securities Act, and in our other filings with the SEC, that may cause our actual results, performance or achievements to differ materially and adversely from those expressed or implied by the forward-looking statements. Any forward-looking statements made herein speak only as of the date of this press release, and you should not rely on forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee that the future results, performance, or achievements reflected in the forward-looking statements will be achieved or will occur. We disclaim any obligation or undertaking to update or revise any forward-looking statements contained herein, which speak only as of the date on which they are made. Unless the context otherwise requires, all references herein to "we," "us," "our," "ATAI" or the "Company" refer to ATAI Life Sciences N.V. and its consolidated subsidiaries. This document is not an offer to buy or sell any security.

Agenda

1	Introductions	Dr. Srini Rao, CSO, atai Life Sciences	12:00-12:05
2	CIAS background and landscape	Dr. Richard Keefe, Professor Duke University	12:05-12:20
3	RL – 007 background and phase 2a data	Dr. Matt Pando, CEO, Recognify	12:20-12:40
4	Q&A		12:40-1:00

atai executive summary & key highlights

1. Mental health disorders have become one of largest global health burdens. Despite the unmet patient need, innovations remain limited.
2. As a response to the technical risks associated with CNS drug development, atai focuses on compounds with prior evidence in humans.
3. Our platform consists of 11 drug development programs and 6 enabling technologies, focusing on differentiated, potentially disease-modifying therapies.
4. We employ a decentralized drug development process, leveraging the atai team and our enabling technologies to aim for improved safety, efficacy and probability of clinical success across our pipeline.
5. Validation of atai's operating model: IPO of COMPASS Pathways and Otsuka partnership with Perception.
6. We have a team of more than 200 highly experienced FTEs / consultants across our platform, and a strong cash position of approx. $430M.

Development program overview: lead compounds, lead indications and stage of development

Lead Compound	Lead Indication	Preclinical	Phase 1	Phase 2	Phase 3	Affiliate Company[1]
PCN-101/R-ketamine	Treatment-Resistant Depression					Perception Neuroscience
RL – 007/Compound[2]	Cognitive Impairment Associated with Schizophrenia					Recognify Life Sciences
DMX-1002/Ibogaine	Opioid Use Disorder					DemeRx IB
GRX-917/Deuterated etifoxine	Generalized Anxiety Disorder					GABA Therapeutics
NN-101/N-acetylcysteine	Mild Traumatic Brain Injury					Neuronasal
KUR-101/Deuterated mitragynine	Opioid Use Disorder					Kures
EMP-01/MDMA derivative	Post-Traumatic Stress Disorder					EmpathBio
RLS-01/Salvinorin A	Treatment-Resistant Depression					Revixia Life Sciences
VLS-01/DMT	Treatment-Resistant Depression					Viridia Life Sciences
LIMITED TO EQUITY INTEREST						
Developing COMP360 therapy, with psychological support from special trained therapists, for treatment-resistant depression. Phase 2b topline data read out in Nov'21.						COMPASS Pathways
Developing DMX-1001, a formulation of noribogaine, as a potential at-home maintenance therapy for OUD. Preclinical stage						DemeRx NB

Note: DMT = N,N-dimethyltryptamine; MDMA = 3,4-Methylenedioxymethamphetamine.
(1) Perception, Recognify, DemeRx IB and Neuronasal are all variable interest entities; GABA is a non-consolidated VIE with operational involvement through MSA model, including Srinivas Rao serving as CMO; EmpathBio, Revixia and Viridia are wholly-owned subsidiaries; COMPASS Pathways and DemeRx NB are non-controlling equity interests.
(2) RL-007 compound is (2R, 3S)-2-amino-3-hydroxy-3-pyridin-4-yl-1-pyrrolidin-1-yl-propan-1-one(L)-(+) tartrate salt.

Speaker bios

Richard S.E. Keefe, PhD
Professor at Duke University School of Medicine & CEO of VeraSci

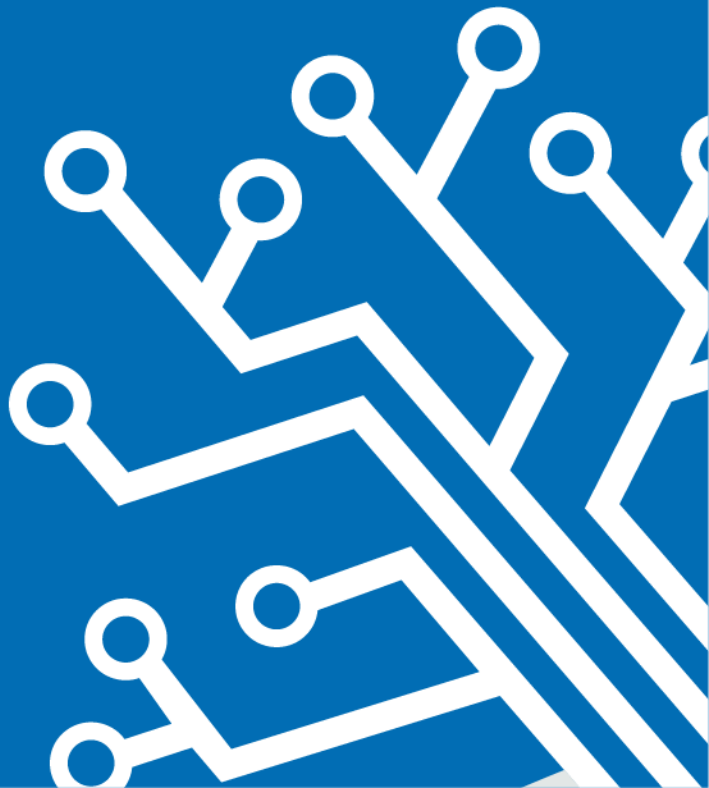
Dr. Keefe has over 25 years of experience in clinical psychology, neuroscience, and drug development. In addition to founding & service as CEO of VeraSci, Dr. Keefe is also a Professor of Psychiatry, Psychology, & Neurosciences at Duke University Medical Center. Furthermore, Dr. Keefe is also a Fellow of the American College of Neuropsychopharmacology and on the Scientific Board of the Brain & Behavior Research Foundation. Dr. Keefe received his PhD in Clinical Psychology from NYU.

Matt Pando, PhD
CEO of Recognify Life Sciences

Dr. Pando has over 20 years of experience in private & public biotech companies. Prior to founding & serving as CEO of Recognify Life Sciences, Dr. Pando served as CSO of Diaxonhit and was most recently a consultant for ForSight Labs. In his previous roles, Dr. Pando contributed to the discovery and development of various products for CNS disease, oncology, & infectious disease. Dr. Pando preformed his thesis work at The Salk Institute and received his PhD in Biology from University of California, San Diego.

The Opportunity of Cognitive Impairment in Schizophrenia (CIAS): Major Cause of Disability With No Approved Treatment

Richard Keefe, PhD
CEO, WCG VeraSci
Professor, Duke University Medical Center



Disclosure

Dr. Keefe is CEO of WCG VeraSci, a for-profit company that provides support for over 100 commercial and academic entities, most of which are drug companies doing clinical trials, and includes atai/Recognify, Boehringer-Ingelheim, Sunovion, and Neurocrine, which are mentioned in this presentation

Worldwide, Schizophrenia is A Leading Cause of Disability

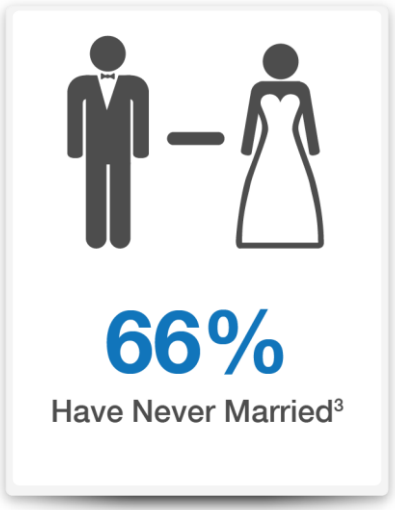
Leading global causes of years of life lived with disability in 15- to 44-year-olds

1. Unipolar depressive disorders
2. Alcohol-use disorders
3. **Schizophrenia**
4. Iron-deficiency anemia
5. Bipolar disorder
6. Hearing loss, adult onset
7. HIV/AIDS
8. Chronic obstructive pulmonary disease
9. Osteoarthritis
10. Road traffic accidents

World Health Organization. The World Health Report 2001. Mental Health: New Understanding, New Hope. Geneva, Switzerland: World Health Organization; 2001.

People With Schizophrenia in the United States Rarely Marry or Find Jobs and May End Up Homeless at Some Point

- Schizophrenia affects about 1% of the general population[1]
- There are over a dozen medications approved to treat Schizophrenia[2] *(primarily targeting positive symptoms)*
- And yet, functional outcomes remain exceedingly poor in this population



[1]*http://www.nimh.nih.gov/health/topics/schizophrenia/index.shtml*
[4]*Rosenheck et al. Am J Psychiatry. 2006;163(3):411-417.*

[2]*http://www.nami.org*
[5]*Folsom et al. Am J Psychiatry. 2005;162(2):370-376.*

[3]*Tandon et al. Schizophr Res. 2009;110:1-23.*
[6]*Harvey et al. J Psychiatry Res. 2012;46:1546-1552.*

In a 2012 study of milestone achievements, only 19% had ever achieved all 3 milestones (marriage, employment and financial responsibility)[6]

The Economic Burden of Schizophrenia

In the United States, the economic burden of schizophrenia for 2013 was estimated at $155.7 billion:

Excess direct health care costs of **$37.7 billion (24%)**

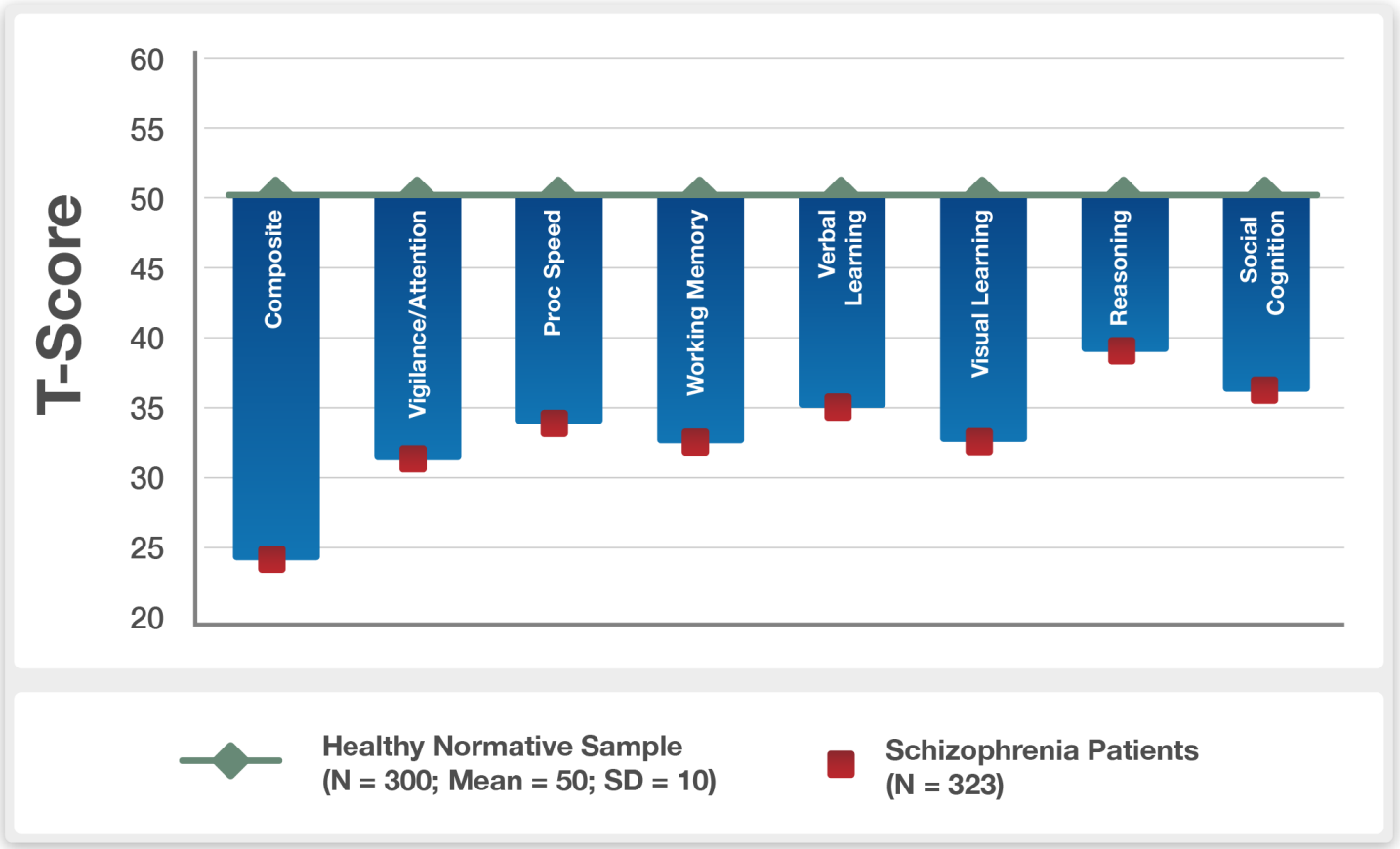
Direct non-health care costs of **$9.3 billion (6%)**

Indirect costs of **$117.3 billion (76%)**

The largest components were excess costs associated with unemployment (38%), productivity loss due to caregiving (34%), and direct health care costs (24%).

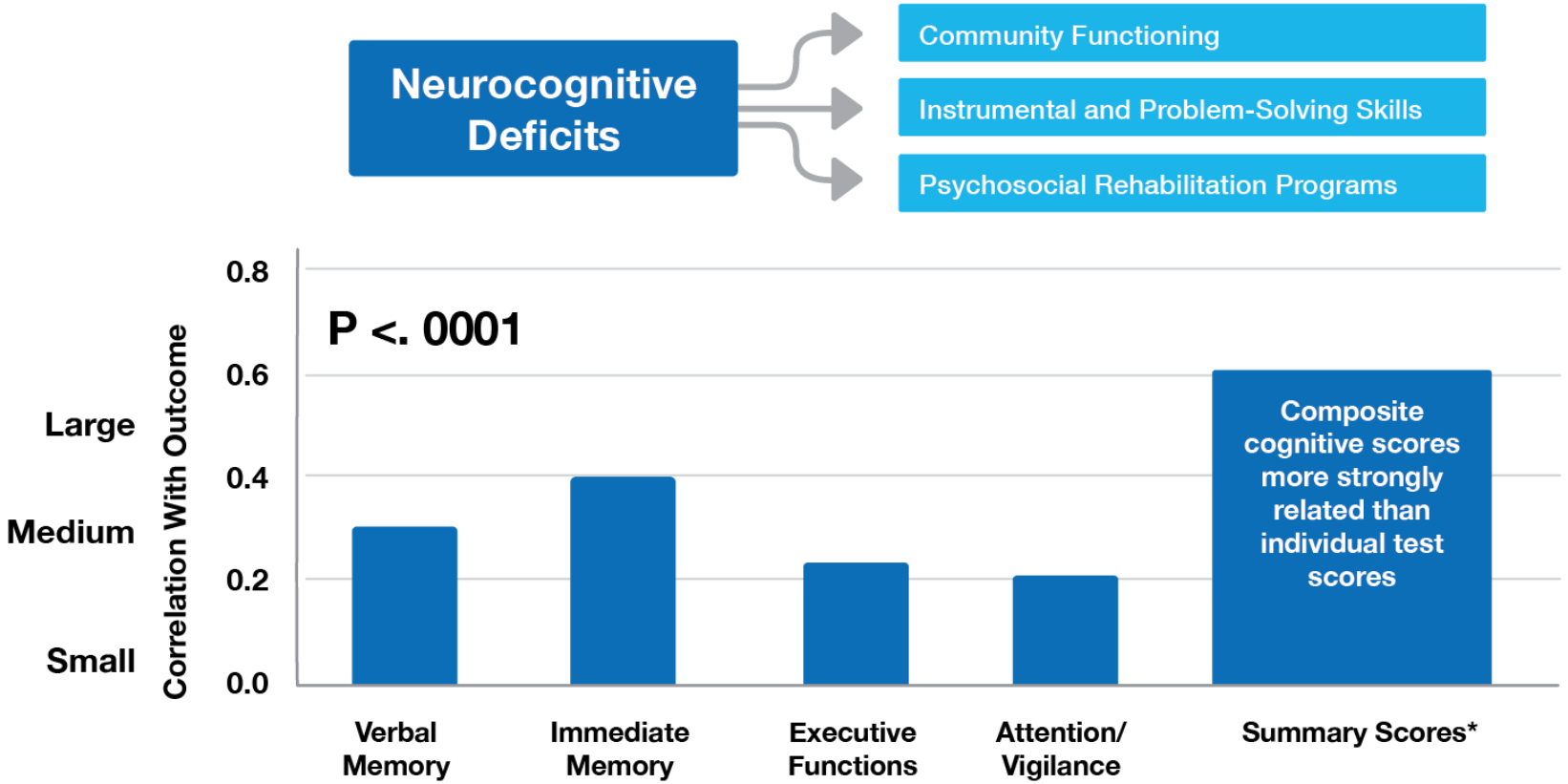
Cloutier et al, The economic burden of schizophrenia in the United States in 2013. J Clin Psychiatry 2016;77(6):764-771

Severity and Profile of Cognitive Impairment in Schizophrenia



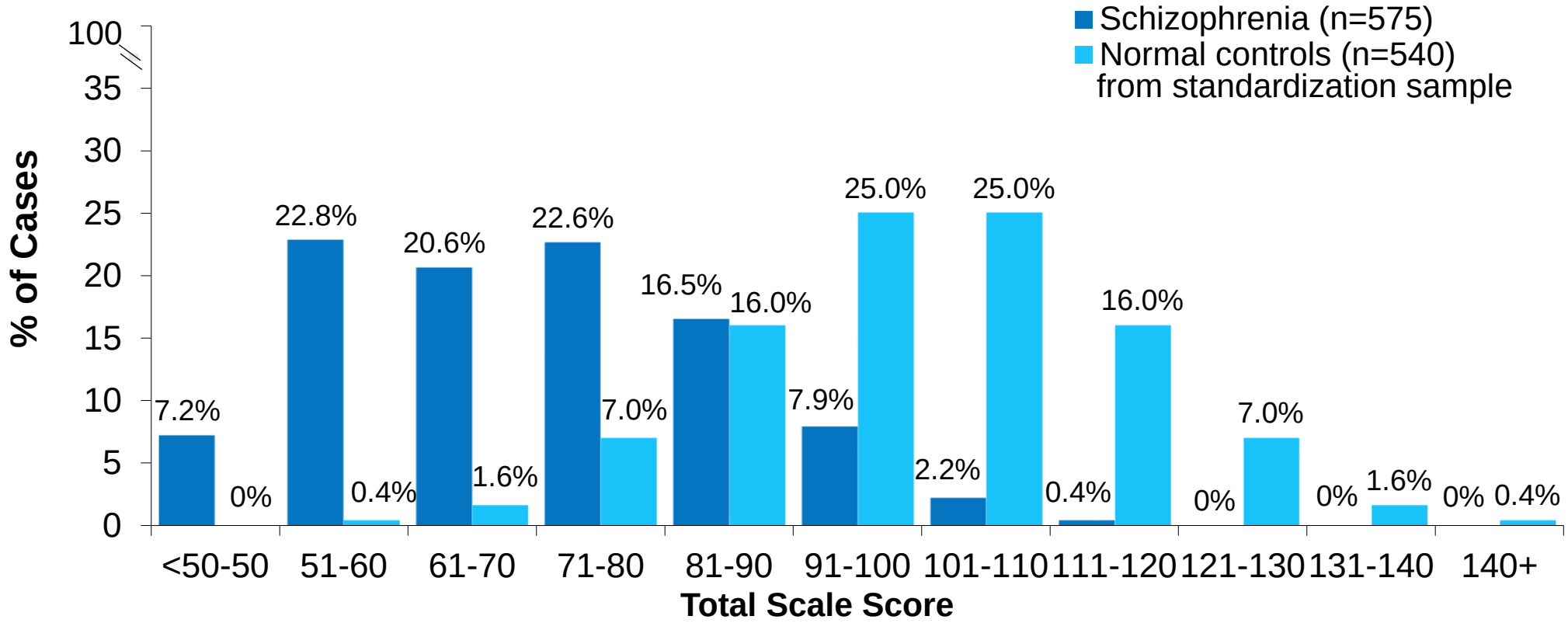
Keefe et al, *Schiz Res*, 2011

Disability in Schizophrenia is Primarily Caused by Cognitive Impairment



*Relationship between outcome and summary scores based upon data on file, Dr Michael F Green, and previously presented in Medscape CME Unraveling the complexities of Schizophrenia: New targets, new opportunities. http://www.medscape.orgiviewarticle/763725transcript. Accessed April 25, 2014. Green MF, et al. Schizophr Bull. 2000;26(1):119-136.

The Large Majority of Patients With Schizophrenia Have Cognitive Impairment



Wilk CM, et al. *Schizophr Res.* 2004;70(2-3):175-186. RBANS: Repeatable Battery for Assessment of Neuropsychological Status

Why Are There No Approved Treatments for CIAS?

200+ CIAS treatment trials listed on Clinicaltrials.gov

Amount of investment that is being made in the development of treatments and opportunities for success

- NIH spend 25 times more on cancer than on schizophrenia
- Industry investment in clinical trials in 2017 was $71.5 billion, dwarfs government efforts.
- There are over 1,000 ongoing clinical trials in cancer for every one in CIAS
- Progress from phase 1 to the US Food and Drug Administration (FDA) approval is 5.1% for cancer indications, very similar to psychiatry at 6.2%.

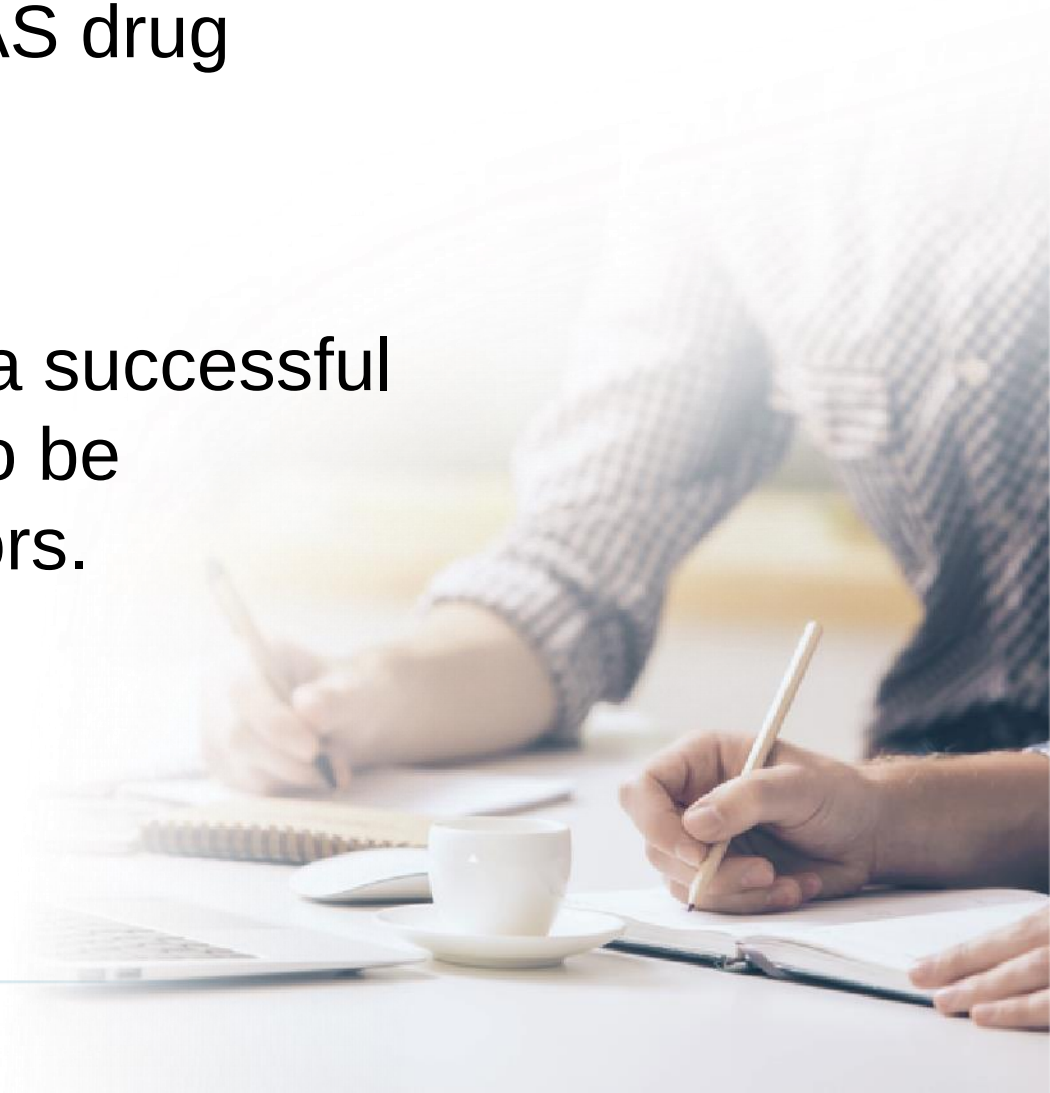
Curing cancer is more likely to be more personally tangible and may appear to be more morally compelling to investors

Keefe RSE. Why are there no approved treatments for Cognitive Impairment in Schizophrenia? World Psychiatry, 2019; Jun; 18(2): 167-168. (*PMID: 31059617*).

Positive Developments

FDA clearly recognizes the severity of the unmet need and continues to be open to expediting CIAS drug development programs

The potential economic gain to society for a successful treatment of CIAS is enormous and likely to be welcomed by private and government payors.



Key Phase 2 and 3 CIAS Programs in Progress

Cognition as a Primary Endpoint

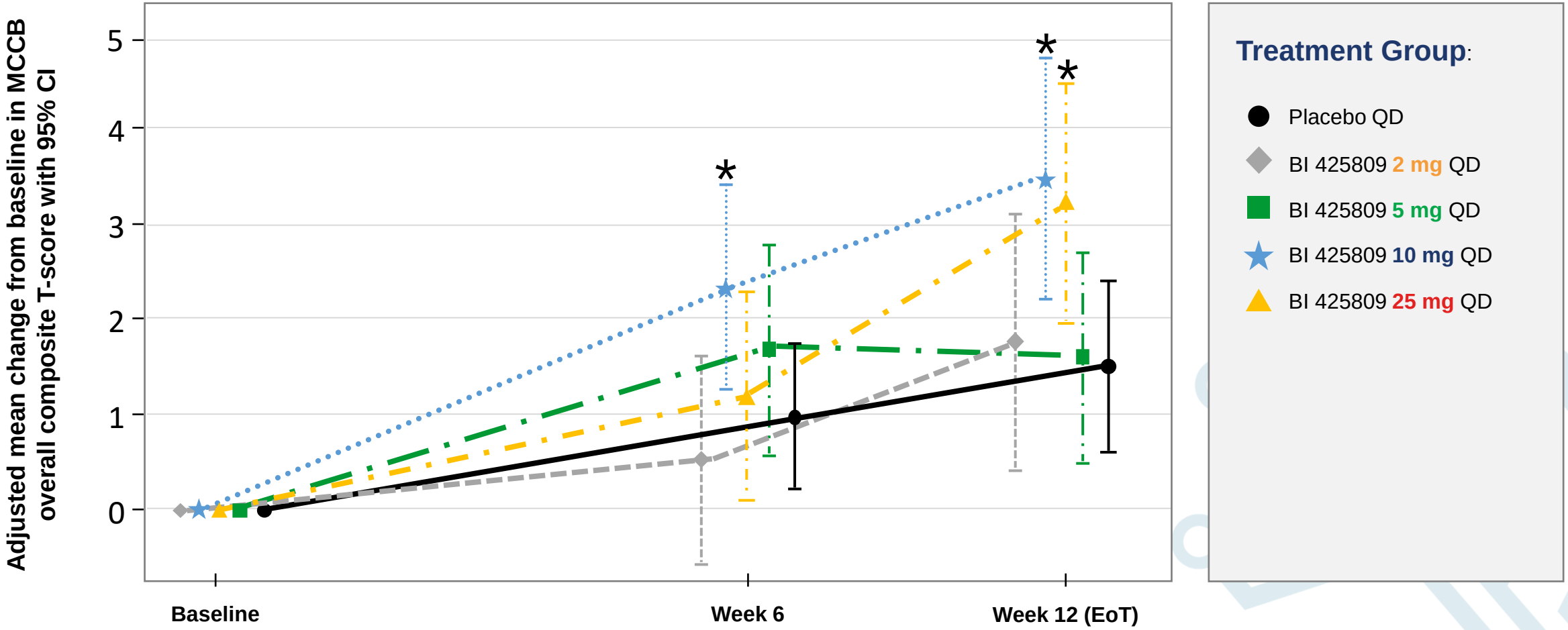
- Boehringer Ingelheim GlyT1 inhibitor
- Biogen positive allosteric modulator (potentiator) of the α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor
- Neurocrine/Takeda Luvadaxistat (Cognition secondary but changing to primary)
    - Negative Outcome on Primary Endpoint Measured by the Change from Baseline on the Positive and Negative Syndrome Scale/Negative Symptom Factor Score (PANSS NSFS)
    - Positive Outcome on Cognitive Assessment Secondary Endpoints Support Potential Further Clinical Development of Luvadaxistat

Cognition as Secondary Endpoint

- Several studies but cognition not listed on clinicaltrials.gov

Note that many of these programs have specific mechanisms of action while the Recognify compound has a more complex MoA that modulates multiple receptor systems. Other compounds that target multiple receptor systems, such as clozapine, have demonstrated powerful efficacy in schizophrenia.

Change From Baseline in the MCCB Overall Composite T-Score



*p<0.05 vs placebo

CI, confidence interval; EoT, end of treatment; MCCB, MATRICS Cognitive Consensus Battery; MMRM, mixed model repeated measures; QD, once daily

Fleischhacker WW,et al. Efficacy and safety of the novel glycine transporter inhibitor BI 425809 once daily in patients with schizophrenia: a double-blind, randomised, placebo-controlled phase 2 study. Lancet Psychiatry. 2021 Mar;8(3):191-201. doi: 10.1016/S2215-0366(20)30513-7. PMID: 33610228.

RL – 007 development & phase 2a data

Recognify team members

- Matt Pando, PhD – CEO, Co-founder + RL – 007 Developer
- Gary Walker, PhD – VP Clinical Development, Co-founder
- John Donello, PhD – CSO + RL – 007 Inventor/Developer
- Rolando Gutierrez-Esteinou, MD – CMO, atai
- Georgina Kilfoil, BS, MBA – Clinical Operations, atai
- Sarah McEwen, PhD – Clinical Scientist, atai
- Kelly Palmer , BS – Study Monitor

RL – 007 is a potentially game changing compound

RL – 007:

a well tolerated neuromodulator that

potently enhances synaptic plasticity &

elicits statistically significant & clinically meaningful effects

on learning & memory

RL – 007: a de-risked pro-cognitive treatment for CIAS

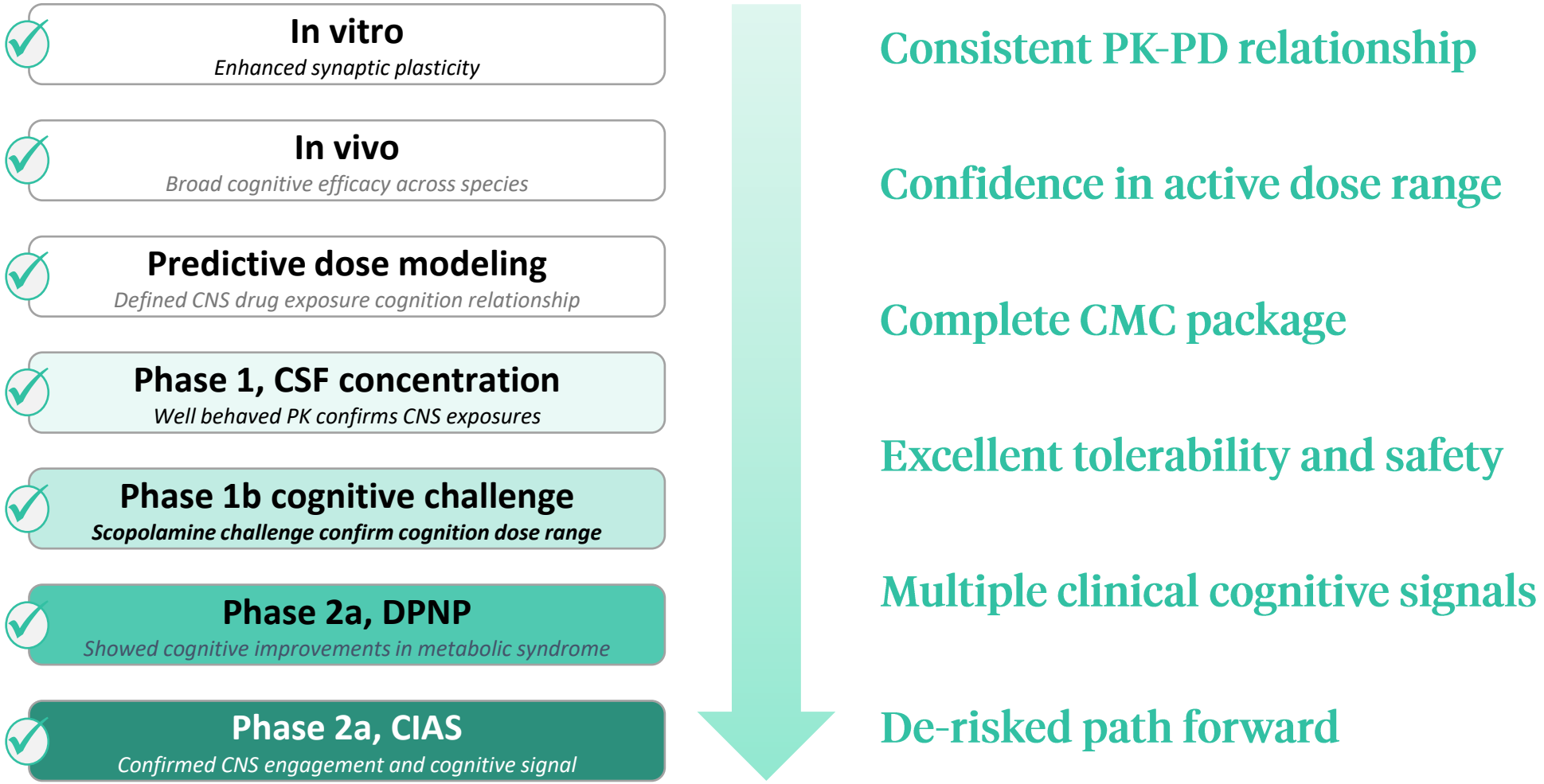
De-risked pharma developed product in-licensed with impressive ($100M) pre-clinical & clinical data package

Human Phase 1 + 2 data show replicated, clinically significant learning and memory effects, consistent with broad pre-clinical pro-cognitive data

Well tolerated (>500 subjects dosed), centrally acting oral drug

Initial indication: Cognitive Impairment Associated with Schizophrenia (CIAS) is characterized by episodic learning and memory deficits – no approved treatment

RL – 007 de-risked through extensive & consistent translational data



Positive pre-clinical results (over 40 *in vitro* and *in vivo* models)

Positive in broad range pre-clinical models & multiple species

Neuroplasticity enhancement

Cognition

Anxiety

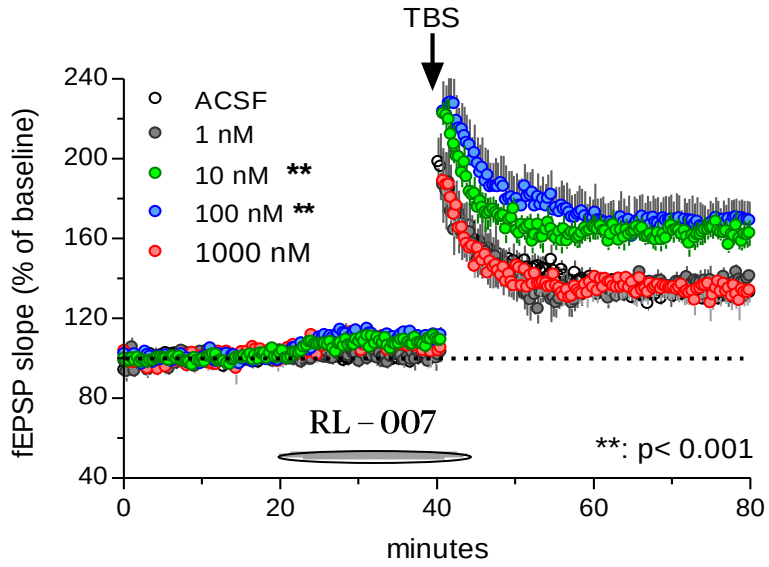
Functional recovery

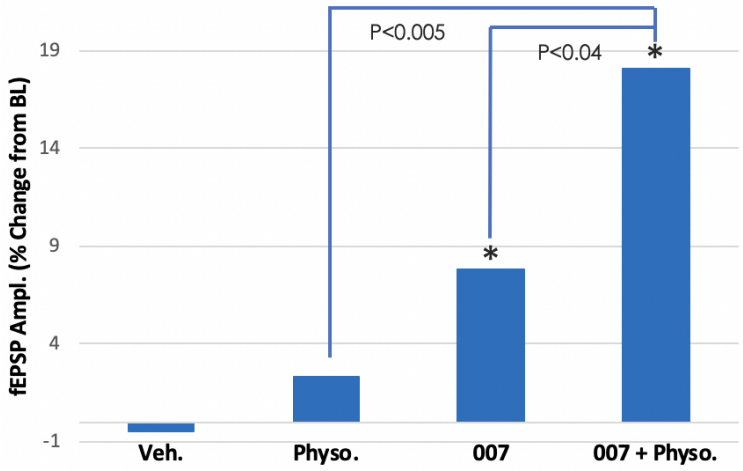
Non-sedating & no dose-limiting CNS tolerability effects

Neuromodulator discovered in vivo, pharmacologically defined using electrophysiology

RL – 007 enhances basal neuronal signaling, glutamatergic synaptic transmission, and long-term potentiation (LTP)

- ✓ Discovered through an in vivo phenotypic screen
- ✓ Enhances basal glutamatergic tone, enhances LTP, and synergizes with AChEIs
- ✓ In Vivo behavioral effects are blocked by GABAB and nACHR antagonists





RL – 007 is active in a broad range of pre-clinical cognition models

Enhances cognition in mouse, rat and dog species:

- Normal young animals
- Aged animals
- Aged mouse model of Alzheimer's Disease (3XTG-AD)

Reverses scopolamine-induced memory deficit in both tox species

Consistent inverted u-shaped PK/PD relationship across species

Pro-cognitive effects translate to humans

RL – 007 development: in CIAS, cognitive deficits have highest impact on everyday functioning & are a critical target for improving QOL

RL – 007 development: excellent PK, well tolerated with replicated cognitive signals

Study		Phase 1	Phase 2	Safety /PK	Target Engaged	Biphasic
HV PK	N=48	7 Day Multiple Dose	Cognition	✓	✓	✓
Scopolamine Cognitive Challenge	N= 23	DB Placebo, 4x XO	Cognition	✓	✓	✓
Peripheral Diabetic Neuropathy	N=180		Double Blind Placebo XO, 1 month, 5 month ext; Cognition	✓	✓	✓

- Excellent PK and CSF exposure
- >500 subjects exposed (9 prior Phase 1 and 2 studies)
- Safety to 6 month (300 mg TID)
- Multiple independent cognitive signals (30-80 mg dose range)

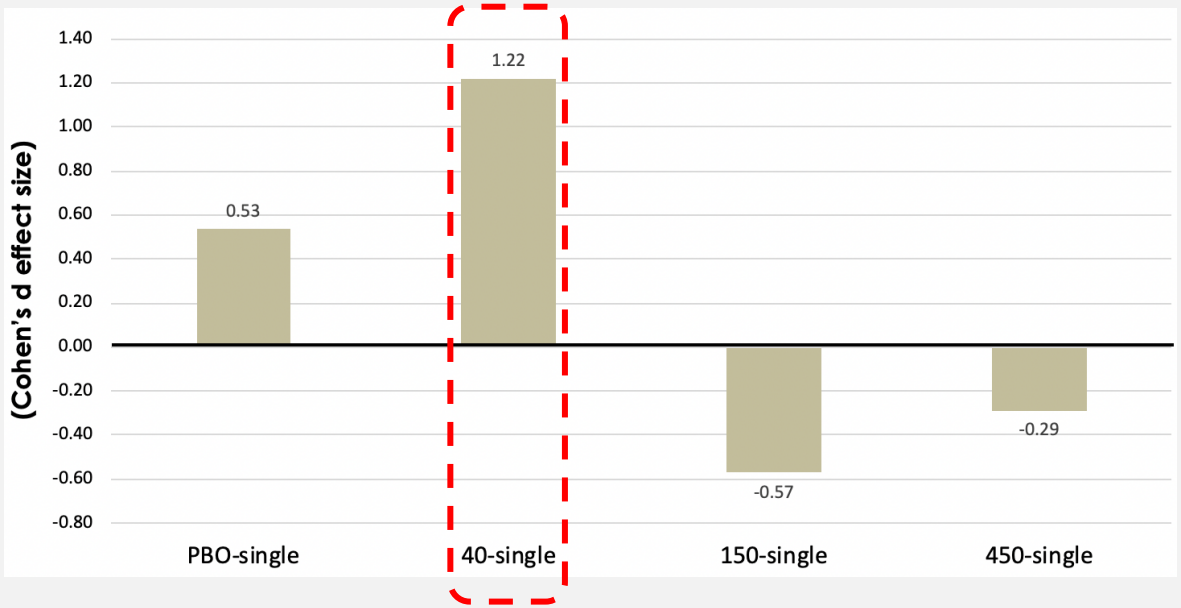
Cognitive improvements seen at the lowest doses in prior studies

Exploratory endpoint analysis shows clinically meaningful learning effects – ISLT (PK study – young caucasians)

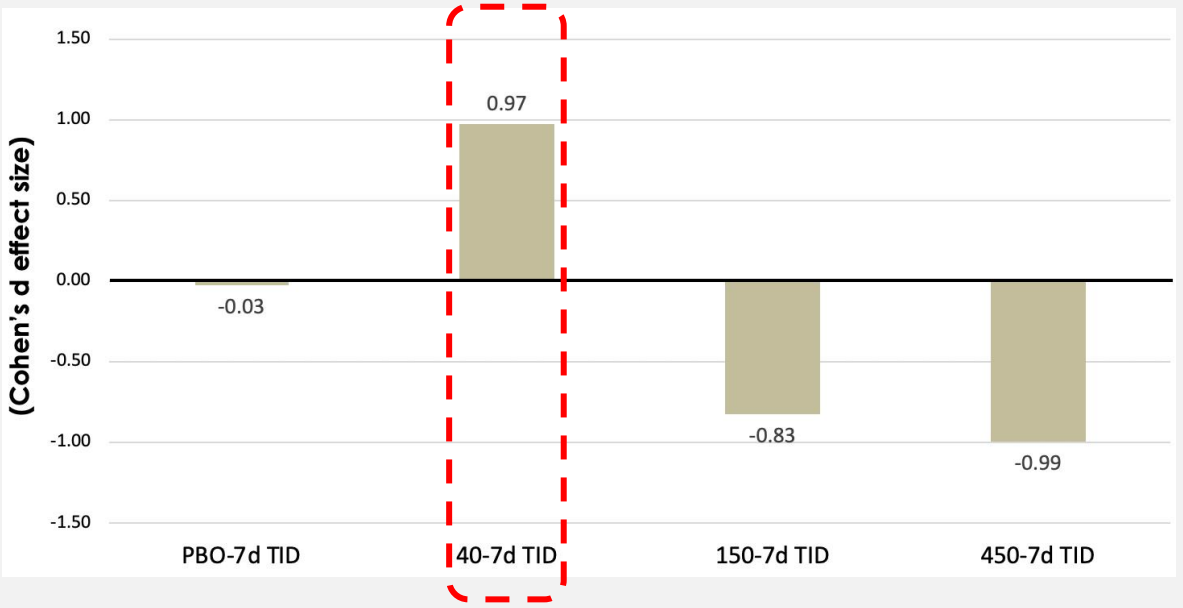
RL – 007 40 mg low dose enhanced learning with single & 7 day TID dosing

(Phase 1 exploratory endpoints – 6 patients/dose)

Single dose



7 day TID dosing

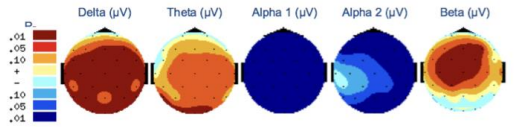


Cohen's d = 0.2 considered a 'small' effect size, 0.5 a 'medium' effect size & 0.8 a 'large' effect size

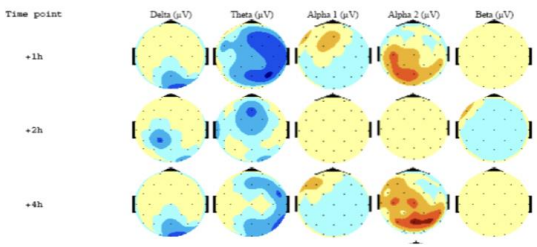
RL – 007 significant clinical learning & memory effects

Phase 1 – reverses cholinergic memory challenge better than market leader Aricept (donepezil)

2 hr Placebo/Scopolamine vs Baseline

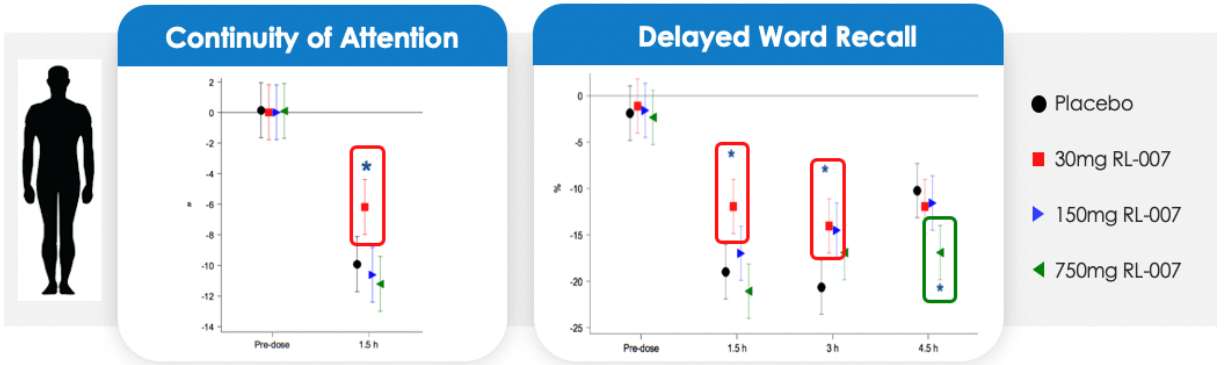


30 mg/Scop vs. Placebo/Scop



Inhibited scopolamine induced EEG changes in Alpha and Theta

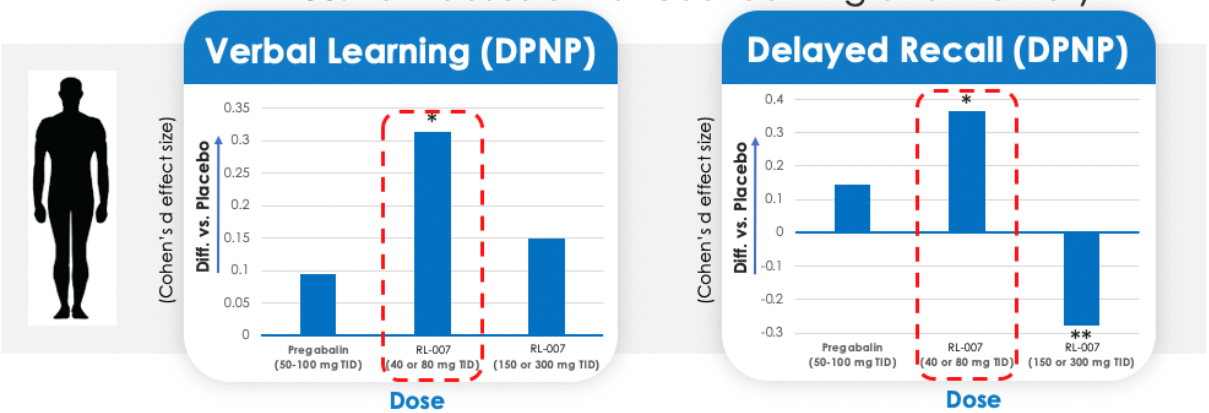
RL-007 significantly **reverses memory deficits** at lowest dose and delays recovery at extremely high dose



*"The effects on delayed word recall were **more marked than seen with the target clinical dose of Aricept (donepezil)**, the most widely prescribed anti-Alzheimer's drug"* [1]

* CSR 209323-502; P<0.05, n = 18; CNS effects also monitored by EEG 1: Keith Wesnes in CDR study report

RL-007 low doses enhanced learning and memory



↑ Indicates direction of improvement

(Phase 2 exploratory endpoints – 180 patients)

* = P< 0.05 vs Placebo; **missed significance (P=0.075); Diabetic Peripheral Neuropathic Pain (DPNP) n=60 patients/treatment group; dosed TID; randomized, cross-over design

Phase 2 - exploratory endpoint analysis shows statistically significant & clinically meaningful positive learning & memory effects

Clinical insights in cognition

Clinically and statistically significant signals in learning and memory across multiple studies

- ✓ **Phase 1 scopolamine challenge:** reverses learning and memory deficits better than donepezil (30 mg TID)
- ✓ **Phase 1 PK study:** single and multi-dose (1 week) effects on learning (40 mg TID)
- ✓ **Phase 2 DPNP pain study:** statistically significant effects on learning and memory (40-80 mg TID)
- ✓ **Phase 2 DPNP pain study:** statistically significant effects on Cognitive and Physical Function Questionnaire (data not shown; 40-80 mg TID)

Objective of RL – 007 Phase 2a study

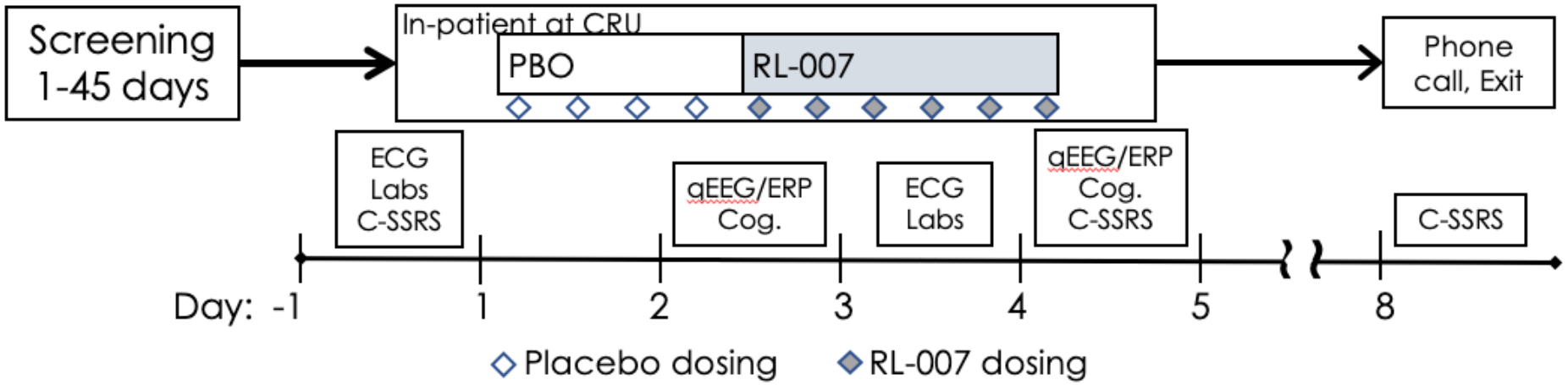
Extend prior clinical data to CIAS patients

- Safety and tolerability
- Confirm CNS activity via EEG in schizophrenia population
- Define dose-response in pro-cognitive dose range (go lower than previous studies)
- Explore acute cognitive signals in CIAS patients

CIAS phase 2a: study schematic

Evaluate independent cohorts dosed with 10, 20, 40, or 80 mg TID

(5 doses TID reaches approximate CSF steady state)



- A single-center, single-arm, single blind, multiple dose study
- Schizophrenia patients stable on a protocol-allowed antipsychotic
- Patients blinded to the dose strength & sequence of active/placebo dosing
- Single site study – Collaborative Neuroscience Research, Dr. David Walling (PI)

Wide-spread beneficial qEEG changes at middle and high doses

EO Condition

Endpoint	Cohort 2 (10 mg)					Cohort 3 (20 mg)					Cohort 4 (40 mg)					Cohort 1 (80 mg)				
	F	C	T	P	O	F	C	T	P	O	F	C	T	P	O	F	C	T	P	O
Delta (2.0 - 4.0 Hz)				↑								↓		↓	↓	↓				
Theta (4.0 - 8.0 Hz)					↑			↓	↓						↑		↓		↓	
Alpha (8.0 - 12.0 Hz)			↓	↓	↓	↑	↑	↑	↑	↑	↑		↑	↑	↑		↑	↑	↑	
Alpha 1 (8.0 - 10.0 Hz)					↓	↑	↑	↑	↑	↑	↑		↑	↑	↑	↑	↑	↑	↑	↑
Alpha 2 (10.0 - 12.0 Hz)	↓	↓	↓	↓	↓	↑	↑	↑	↑	↑	↑	↓	↑	↑	↑	↑	↘		↑	↑
Beta (12.0 - 25.0 Hz),	↓	↓	↓	↓	↓													↑		
Beta 1 (12.0 - 15.0 Hz)	↓	↓	↓	↓	↓	↘	↑	↘	↓	↓	↓	↓	↘	↓		↑	↓			↓
Beta 2 (15.0 - 18.0 Hz)	↓	↓	↓	↓	↓			↓	↑			↑		↑	↑	↓	↓		↓	↘
Beta 3 (18.0 - 25.0 Hz)	↘	↓	↓	↓	↓					↑		↓	↑		↑	↑	↑	↑		↓
Alpha Slow wave index (ASI)			↓	↓	↓	↑	↑	↑	↑	↑	↑		↑	↑	↑	↑	↑	↑	↑	↑
Theta-Beta Ratio (TBR)	↑	↑	↑	↑	↑	↑			↓					↑		↓	↓	↓	↓	↑
Beta-Alpha Ratio (BAR)	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↓	↑	↓		↓	↓
Dominant frequency, frontal (IAF1)																				
Dominant frequency, occipital (IAF2)																				

Eyes open qEEG change: day 2 to day 4

Small	Large	
↑	↑	Increase only
↓	↓	Decrease only
↗	↗	Decrease then increase
↘	↘	Increase then decrease
↓↑	↓↑	Variable over time or regions

Small <-0.10 or >0.10
Large <-0.15 or >0.15
Nothing >=-0.10 & <=0.10

±0.05 and ±0.10 for 1/F slope

Orange-shaded and blue-shaded cells respectively indicate increases and decreases that were significant in the mixed model (MMRM) analysis.

Increased alpha/ASI (middle & high doses), decreased beta (lowest dose)

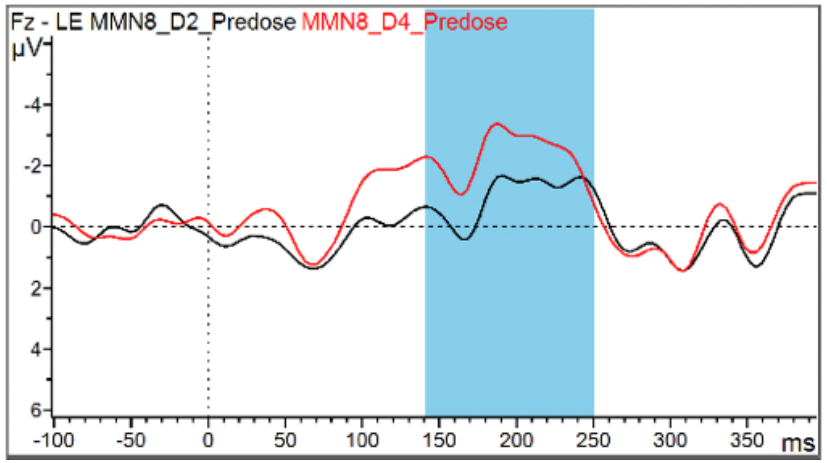
Preliminary evidence for dose-dependent changes in ERP

ERPs support physiological differences between acute administration of low and high dose groups

RL – 007 may remediate SZ-related deficits in sensory memory at low to moderate doses

- With this acute treatment there was no consistent response across timepoints
- Significantly increased MMN amplitudes at the lower dose levels (10 & 20 mg) vs salient decrease in MMN amplitudes at 40 mg
- No effect on P300b at low doses vs sporadic significantly decreased in P300b amplitudes at the highest dose levels (40 & 80 mg)

MMN8 trough exposure time-point D2 vs D4 – PKAMP (20mg)



- Mismatch negativity – passive auditory oddball event-related potential (ERP)
- Salient beneficial changes in MMN amplitude and latency observed in 20 mg cohort

Cognitive testing: subset of tasks from MCCB

MATRICS Consensus Cognitive Battery (MCCB): 10 tests measuring seven cognitive performance domains

- speed of processing, attention/vigilance, working memory, verbal learning, visual learning, reasoning and problem solving, and social cognition

Hopkins Verbal Learning Test-Revised (HVLT-R) – Positive in multiple previous studies

- Cognitive domain: **verbal learning and memory**
- Procedure: learn and recall a list of 12 words – given 3 trials, then 20-25 minutes later
- Outcome measurement: number of words recalled (immediate)

Brief Assessment of Cognition in Schizophrenia (BACS): symbol coding test

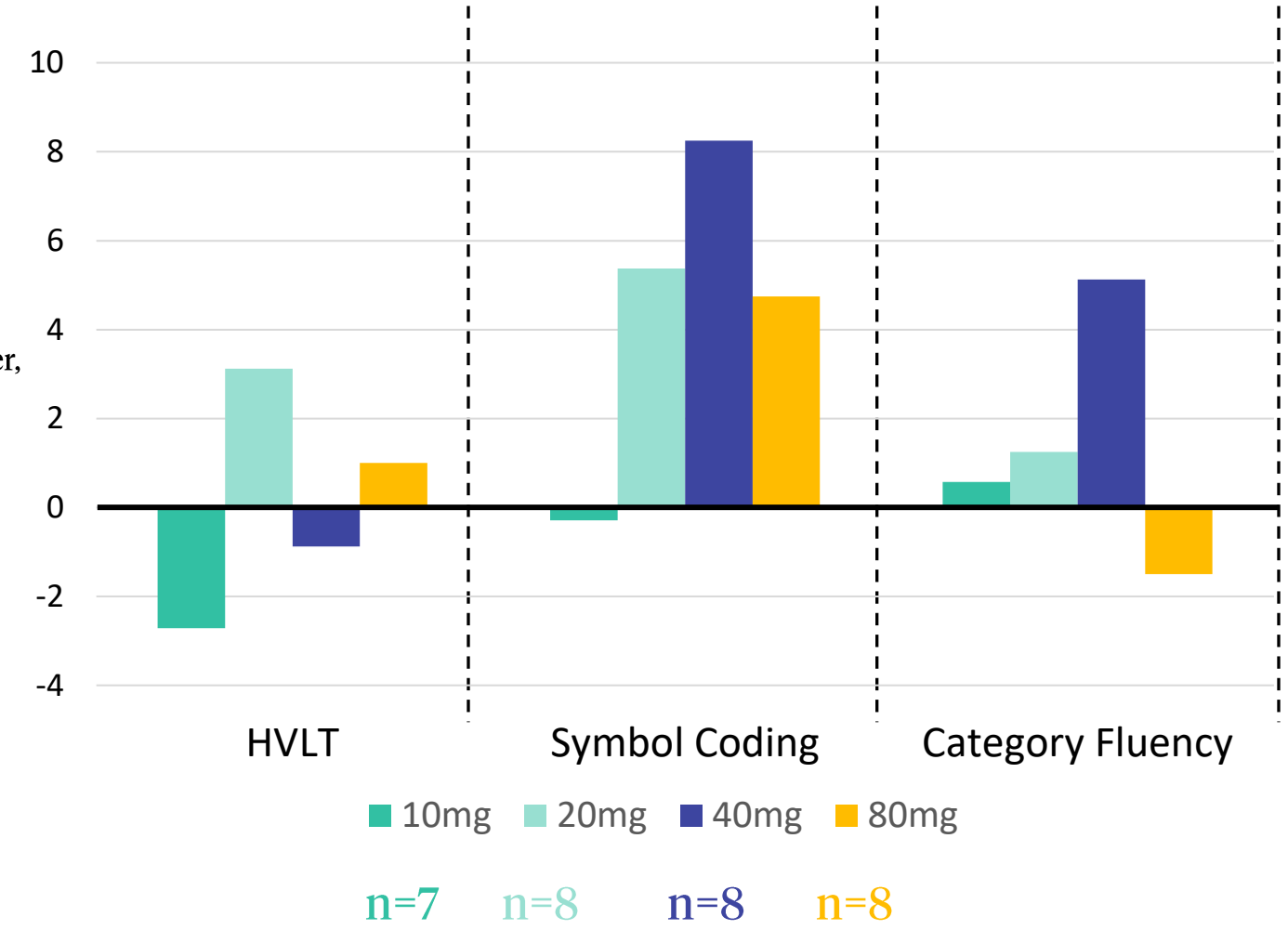
- Cognitive domain: **processing speed, attention & working memory**
- Procedure: matching symbols and numbers (1-9) as quickly as possible
- Outcome measurement: number of correct responses

Category fluency task

- Cognitive Domain: **processing speed (verbal ability and executive control)**
- Procedure: say as many words in 1 minute in a specific category (e.g. "animals")
- Outcome measurement: number of appropriate items provided in the category

Acute bell-shaped cognitive response

T-scores
(Normalized for age, gender, and education level)



"Symbol coding is the most sensitive measure and it correlates well with the other tasks in MATRICS" – Rich Keefe

Improved

Cohen's d* (Symbol Coding):
20 mg: 0.79
40 mg: 0.56
80 mg: 0.38

*Day 4 vs Day 2

Clinical insights in cognition

Encouraging bi-phasic trends in cognitive data

Symbol coding a well-established and sensitive measure in CIAS

- Correlates well with overall movement of MATRICS battery
- Observed changes larger than expected from practice
- Effect size in range that correlates with improvement in work/school performance

Immediate word recall effects similar to previous studies (20 mg)

Longer duration of treatment may extend/strengthen initial findings in CIAS

* Keefe, Harrison, Green, & Nuechterlein

CIAS phase 2a result summary

Extension of prior clinical data to CIAS

- Safe and well-tolerated in SZ population
- Dose dependent, widespread qEEG changes across brain regions
  - EO - Increased alpha/ASI (middle & high doses), decreased beta (lowest dose)
  - EC - Observed elevations in resting state alpha, increased ASI, and decreased TBR
  - **Suggesting a relaxed wakeful state without drowsiness at the mid to high dose levels**
- ERP data suggests some dose-dependent cognition relevant changes
- Evidence of bell-shaped dose-response
- HVLT-R immediate recall effect size = 0.44 (20 mg)
- Symbol coding effect size ~0.4-0.8

"Symbol coding response is at a level that would correlate with better work/school performance" – Keith Nuechterlein

Key considerations for future studies

Study		Phase 1	Phase 2	Safety /PK	Target Engaged	Biphasic
HV PK	N=48	7 Day Multiple Dose	Cognition	✓	✓	✓
Scopolamine Cognitive Challenge	N= 23	DB Placebo, 4x XO	Cognition	✓	✓	✓
Cognitive Impairment Associated with Schizo	N=32		Open Label, 2 Day; Cognition	✓	✓	✓
Peripheral Diabetic Neuropathy	N=180		Double Blind Placebo XO, 1 month, 5 month ext; Cognition	✓	✓	✓
Cognitive Impairment Associated with Schizo	N= X		Double Blind Placebo			

Initial CIAS study: confirmed safety, target engagement & cognitive response

Next study: demonstrate statistically significant improvement in cognition

Key considerations: dose, effect size & treatment duration

- Optimum dose range: 20 mg – 80 mg TID
- Pro-cognitive: Cohen's d effect sizes of ~0.2 – 1.0+ (verbal learning, symbol coding)
- Durations tested: single dose to 4 weeks TID

RL – 007: future directions

RL – 007 is well tolerated with the potential to address multiple areas of high patient need, starting with CIAS

RL – 007 is a potential central pillar for atai in neuropsych

With additional high-value opportunities in CNS