UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): February 27, 2017

Minerva Neurosciences, Inc.
(Exact name of registrant as specified in its charter)

Delaware	001-36517	26-0784194
(State or other jurisdiction of incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

1601 Trapelo Road Suite 284 Waltham, MA	02451
(Address of principal executive offices)	(Zip Code)

(Registrant's telephone number, including area code): (617) 600-7373

(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 8.01. Other Events.

Minerva Neurosciences, Inc. (the "Company") is filing the investor presentation slides attached as Exhibit 99.1 to this Current Report on Form 8-K, which the Company may use from time to time in conversations with investors and analysts. The presentation will also be available in the investor relations section of the Company's website.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Investor Presentation dated February/March 2017.

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

MINERVA NEUROSCIENCES, INC.

By: /s/ Mark S. Levine
Name: Mark S. Levine
Title: Senior Vice President, General Counsel and Secretary

Date: February 27, 2017

INDEX OF EXHIBITS

Exhibit No.	Description
99.1	Investor Presentation dated February/March 2017.

Exhibit 99.1

MINERVA NEUROSCIENCES, INC.

Innovation to change the way we treat CNS disease

February / March 2017

Nasdaq: NERV

Forward-Looking Statement Safe-Harbor

MINERVA NEUROSCIENCES, INC.

This presentation contains certain forward-looking statements about Minerva Neurosciences that are intended to be covered by the safe harbor for "forward-looking statements" provided by the Private Securities Litigation Reform Act of 1995, as amended. Forward-looking statements are statements that are not historical facts. Words such as "expect(s)," "feel(s)," "believe(s)," "will," "may," "anticipate(s)" and similar expressions are intended to identify forward-looking statements. These statements include, but are not limited to: the benefits, efficacy and safety of our new formulations; whether studies performed on analogs or backups of our compounds are a good predictor of the clinical efficacy of our compounds; the timing and results of future clinical milestones; the timing of future clinical trials and results of such clinical trials; statements regarding our ability to successfully develop and commercialize our therapeutic products; our ability to expand our long-term business opportunities; our expectations regarding approval for our products by the U.S. Food and Drug Administration or equivalent foreign regulatory agencies; estimates regarding the market potential for our products; financial projections and estimates and their underlying assumptions; and future performance. All of such statements are subject to certain risks and uncertainties, many of which are difficult to predict and generally beyond the control of the Company, that could cause actual results to differ materially from those expressed in, or implied or projected by, the forward-looking statements. These risks and uncertainties include, but are not limited to: the benefits, efficacy and safety of our new formulations; whether analogs or backups of our compounds are a good predictor of the clinical efficacy of our compounds; the timing and results of future clinical milestones; the timing of future clinical trials and results of such clinical trials; whether any of our therapeutic candidates will advance further in the clinical trials process and whether and when, if at all, they will receive final approval from the U.S. Food and Drug Administration or equivalent foreign regulatory agencies and for which indications; whether any of our therapeutic candidates will be successfully marketed if approved; whether our therapeutic product discovery and development efforts will be successful; our ability to achieve the results contemplated by our collaboration agreements; the strength and enforceability of our intellectual property rights; competition from pharmaceutical and biotechnology companies; the development of and our ability to take advantage of the market for our therapeutic products; our ability to raise additional capital to fund our operations on terms acceptable to us; general economic conditions; and the other risk factors contained in our periodic and interim reports filed with the Securities and Exchange Commission which are available on the SEC website at www.sec.gov. Our audience is cautioned not to place undue reliance on these forward-looking statements that speak only as of the date hereof, and we do not undertake any obligation to revise and disseminate forward-looking statements to reflect events or circumstances after the date hereof, or to reflect the occurrence of or non-occurrence of any events.

Investment highlights

MINERVA
NEUROSCIENCES, INC.

- Late-stage pipeline targets schizophrenia, major depressive disorder (MDD), insomnia and Parkinson's
- Our product candidates represent true innovation in disease treatment and management by addressing significant unmet needs of large patient populations
- MIN-101 may change schizophrenia treatment paradigm
  - FDA end-of-Phase 2 meeting early Q2
  - Initiation of Phase 3 in Q3 2017
- >$91.9m million in cash 30 Sept 2016
- Experienced clinical development team who have participated in more than 800 clinical studies

Our team

MINERVA NEUROSCIENCES, INC.

- ***Remy Luthringer, Ph.D., President and Chief Executive Officer***
  - Deep clinical development experience with > 150 CNS molecules
  - Previous head of FORENAP Institute for Research in Neurosciences and Neuropsychiatry
  - Extensive practice in clinical psychiatry, with Ph.D. in neurosciences and clinical pharmacology

- ***Geoff Race, Executive Vice President, Chief Financial Officer and Chief Business Officer***
  - Senior executive with multiple clinical and development-stage biopharmaceutical companies
  - Expertise in mergers, acquisitions and licensing
  - Track record in business development

- ***Michael Davidson, M.D., Chief Medical Officer***
  - Consultant to the biopharmaceutical industry, with insights into development strategy and regulatory review of CNS compounds
  - Internationally recognized author, researcher, award recipient and thought leader
  - Professor of Psychiatry at Sackler School of Medicine, Tel Aviv University

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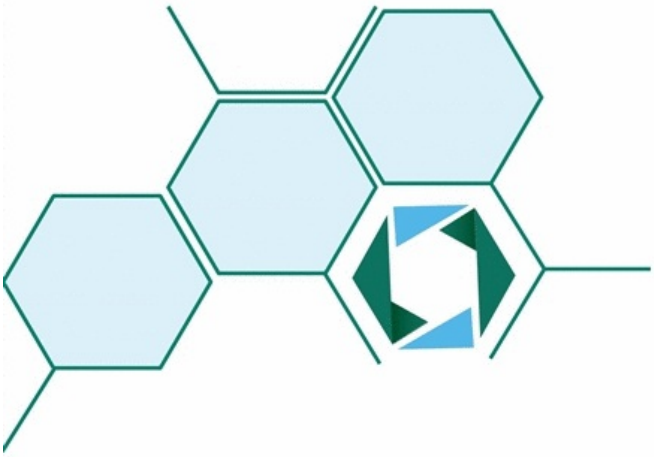
True differentiation in our approach

- Extensive knowledge of the pathology, course and impact of CNS diseases
- Focus on outcomes that address unmet needs (no “me-too” products) and on patient populations who will benefit from treatment with our compounds
- Understanding of the impact of novel MOAs and the limitations of current treatments
- Expertise in CNS clinical trial design and conduct

Changing the way we treat CNS diseases

MINERVA NEUROSCIENCES, INC.

Program	Origin	Primary Indications	Mechanisms of Action	Pre-clinical	Phase I	Phase II	Status	Next Steps
MIN-101	Mitsubishi Tanabe	Schizophrenia	• 5-HT2A antagonist • Sigma2 antagonist	**Phase IIb completed**			Results announced May & October 2016	• End of Phase II meeting with FDA scheduled early in Q2 2017 • Initiation of pivotal Phase III trial planned in Q3 2017
MIN-202	Janssen (under co-development)	Primary Insomnia Major Depressive Disorder	• Selective Orexin2 antagonist	**Phase IIa completed** **Phase Ib completed**			Results Announced January 2016 Results announced March 2016	• Phase II trial preparation underway • Next trials in insomnia disorder and MDD planned for second half of 2017
MIN-117	Mitsubishi Tanabe	Major Depressive Disorder	• 5-HT1A • 5HT transporter • Alpha-1a, b • Dopamine transporter • 5-HT2A antagonist	**Phase IIa completed**			Results announced May 2016	• Planning underway for next phase of clinical trials expected to begin in late 2017
MIN-301	Mind-NRG	Parkinson's Disease	• Neuregulin 1β1 activating ErbB4	**Pre-clinical**			Pre-clinical activities ongoing	• Filing of IND or IMPD, with Phase I expected to initiate thereafter



MIN-101

A new paradigm for the treatment of schizophrenia

Schizophrenia: a devastating chronic disease with a high burden for patients, families and society

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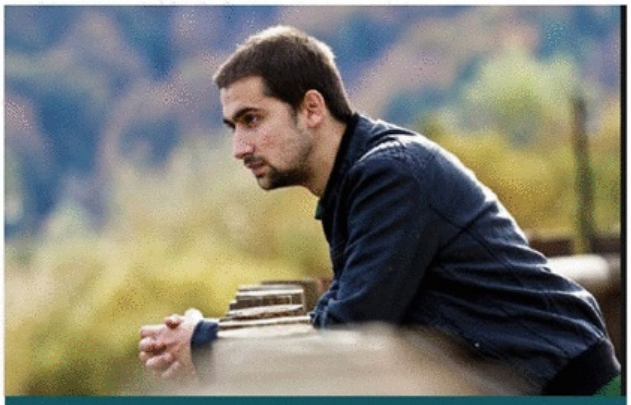
- Affects ~30 million people worldwide[1]
- Often starts in late teens or early adulthood[2]
- 75% patients are non-adherent to existing therapies within 2 years of being discharged from hospital[3]
- Medication non-adherence is the single largest factor in relapse[4]
- Schizophrenia: not a classic neurodegenerative disease yet associated with progressive atrophic changes



What do we need?

Treatments that:

- Improve negative symptoms and cognition
- Free patients from debilitating side-effects
- Improve sleep

1. *Global Prevalence of Schizophrenia PLOS Medicine, 2005*
2. *NIMH*
3. *Weiden PJ et al. Psychiatr Serv,1995; 46:1049-1054*
4. *Weiden PJ (2004), Kozma C, Grogg A et al. Psychiatr Serv, 2004, 55:886-891*

Schizophrenia is a dynamic chronic disease - prevalence of symptoms changes over the lifetime of the patient

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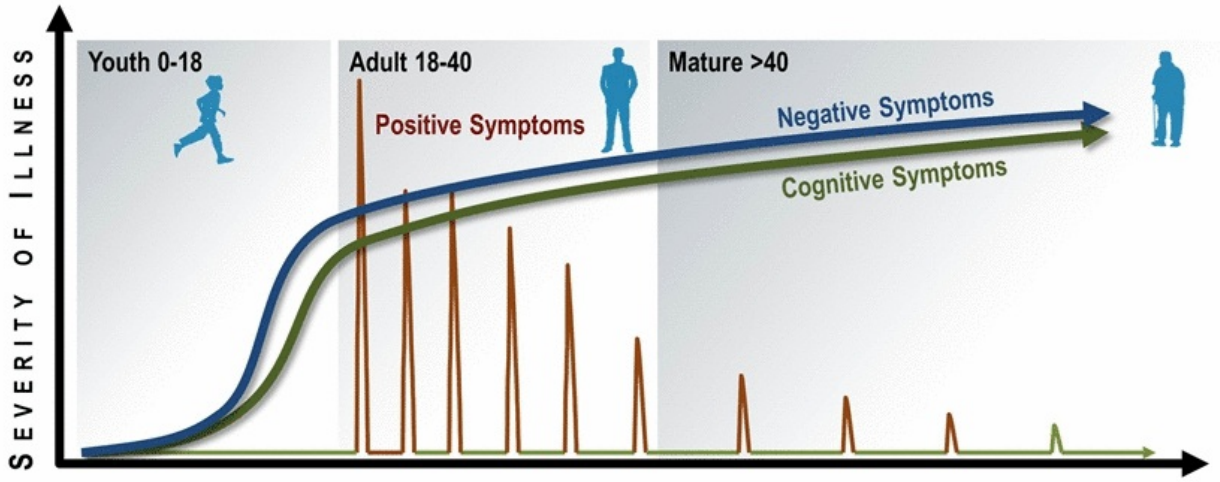

The unmet needs in schizophrenia

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Tranquilizers and antipsychotics treat

1. Positive Symptoms

- *delusions and hallucinations*

but not

- ***Cognition***
- ***Negative Symptoms***

and have

- *debilitating side effects caused by blocking D2*

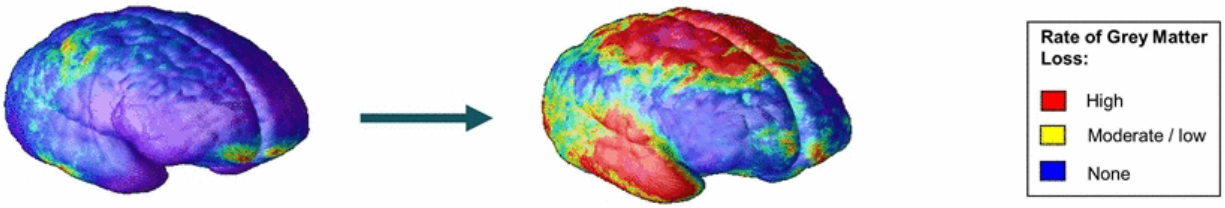
2. Impaired Cognition

3. Negative Symptoms

- *apathy*
- *restricted social interaction*
- *poor emotional feelings*
- *physical and mental slowness*
- *depressed mood*

Normal brain

Brain of a patient with long-standing schizophrenia



Source: Business Insights, NIMH, DSM-5, Journal of Clinical Psychiatry, PNAS.

Responding to unmet needs in schizophrenia

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Unmet need	MIN-101 clinical benefits (Ph IIb)
Lack of efficacy of current Rx on negative symptoms	✓ A direct (not pseudo-effect) on negative symptoms and an improvement in depression in schizophrenia
Substantial side effect burden	✓ Absence of typical side effects associated with D2 blockers
Lack of efficacy of current Rx on cognitive decline	✓ MIN-101 showed cognitive improvement

In addition, positive symptom scores remained stable over ~9 months when patients were taking MIN-101

MIN-101: a new approach and MOA

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- A unique dual MOA; 5-HT_{2A} antagonist + $Sigma_2$ antagonist
- No direct dopamine blocking, unlike most (or all) available antipsychotics
- Specific affinity for σ_2, 5-HT_{2A} and α_1-adrenergic receptors
- No affinity (>1000 nM) for other receptors including dopaminergic, muscarinic, cholinergic and histaminergic receptors
- Behavioral pharmacology is consistent with an antagonistic effect for σ_2 and 5-HT_{2A} receptors

MIN-101 Phase IIb study: monotherapy, double-blind, placebo-controlled in schizophrenic patients with negative symptoms

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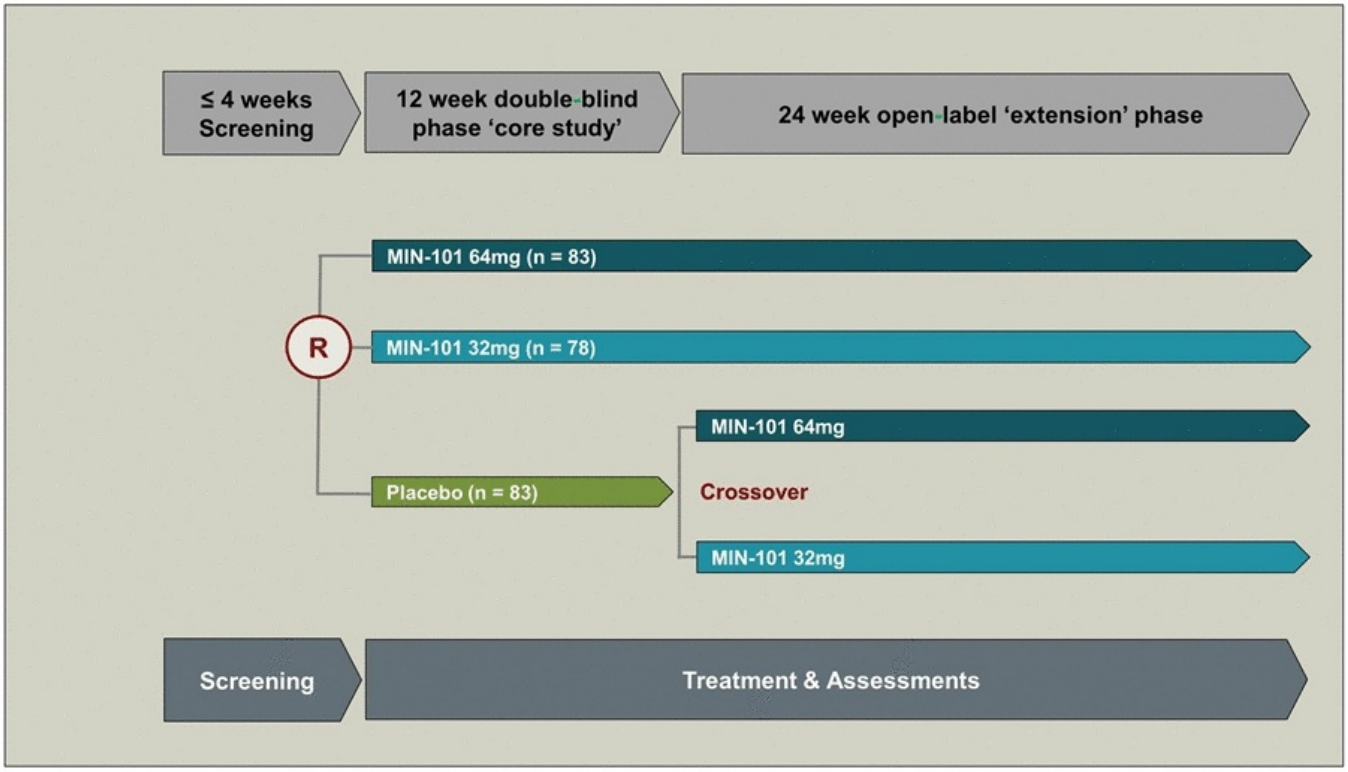

R = Randomization

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MIN-101 Phase IIb data – *setting a new standard*

- Treatment resulted in **statistically significant improvement in PANSS negative symptoms** and total PANSS scores
- Statistically **superior to placebo on multiple key secondary endpoints**
- Positive **effects were specific for negative symptoms** and not secondary to improvement in other symptoms or side effects
- MIN-101 **well tolerated**, with incidence and types of side effects not differing significantly from placebo; no “atypical side-effects” observed

  (two patients (2/162) receiving highest dose of MIN-101 discontinued based on QT prolongation)

Final results of MIN-101 Phase IIb efficacy analyses (12 weeks)

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		p-value (MIN-101 vs placebo)		Effect size (MIN-101 vs placebo)	
	Endpoint at 12 weeks	32mg	64mg	32mg	64mg
Primary objective	5-Factor Negative Score (i.e., Negative Symptoms, Pentagonal Structure):	**0.0240**	**0.0036**	**0.45**	**0.57**
Secondary objectives	PANSS total score	0.0819	**0.0031**	**0.34**	**0.57**
	3-Factor Negative Score	**0.0064**	**0.0004**	**0.54**	**0.70**
	3-Factor Positive Score	0.4018	0.3067	0.16	0.20
	3-Factor General Psychopathology Score	0.2359	**0.0034**	0.23	**0.56**
	5-Factor Positive Score	0.5045	0.2146	-0.13	0.24
	5-Factor Dysphoric Mood Score	0.5644	**0.0266**	0.11	**0.43**
	5-Factor Activation Score	**0.0240**	**0.0118**	**0.44**	**0.49**
	5-Factor Autistic Preoccupation Score	0.6700	0.2408	0.08	**0.22**
	CGI-S* (severity)	0.0982	**0.0234**	**0.35**	**0.43**
	CGI-I** (improvement)	0.2378	**0.0032**	**0.33**	**0.57**
	BNSS (Brief Negative Symptom Scale)	0.0869	**0.0040**	**0.33**	**0.56**
	BACS cognition assessment (Composite T-Score)	0.0595	0.6967	**0.30**	0.06
	- Executive Function: Tower of London	0.3937	0.5995	**0.16**	**-0.10**
	- Motor Function: Token Motor Test	**0.0306**	**0.0493**	**0.42**	**0.38**
	- Motor Function: Symbol Coding Task	0.6310	0.0781	**-0.09**	**-0.33**
	- Total Verbal Fluency	**0.0076**	0.0554	**0.51**	**0.36**
	- Verbal Memory & Learning: Verbal Memory	0.1544	0.3158	0.27	0.19
	- Working Memory: Digit Sequence Task	0.0664	0.8826	**0.36**	0.03
Exploratory objectives	CDSS depression scale	0.1756	**0.0091**	**0.25**	**0.46**
	PSP personal and social performance				
	- Socially Useful Activities	0.4775	0.0601	0.14	**0.38**
	- Personal & Social Relationships	0.9174	**0.0129**	0.02	**0.53**
	- Self-care	0.1736	**0.0210**	0.27	**0.46**
	- Disturbing & Aggressive Behavior	0.0532	**0.0057**	**0.36**	**0.51**

Bold red text indicates p-value ≤ 0.05

Green text indicates moderate or large ES

* Analyzed using ranked data: change from Baseline and ES are based on observed change from baseline data

** Analyzed using ranked data; ES is based on observed data

Efficacy: Primary endpoint – clinically meaningful effect on negative symptoms with rapid onset of effect and continuous improvement throughout the study at both doses

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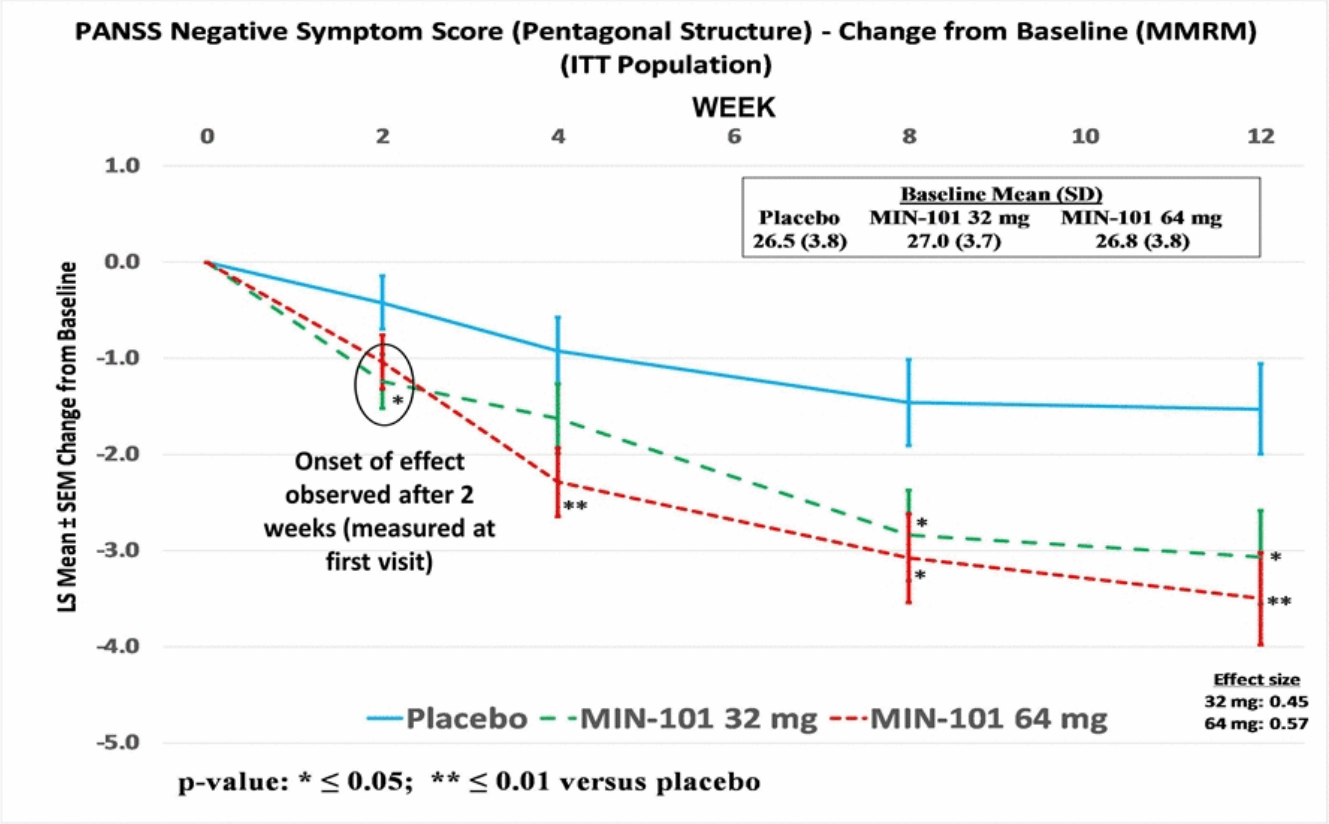

Efficacy: Secondary endpoint (1)
Improvement in negative symptoms also observed with additional measurement scale

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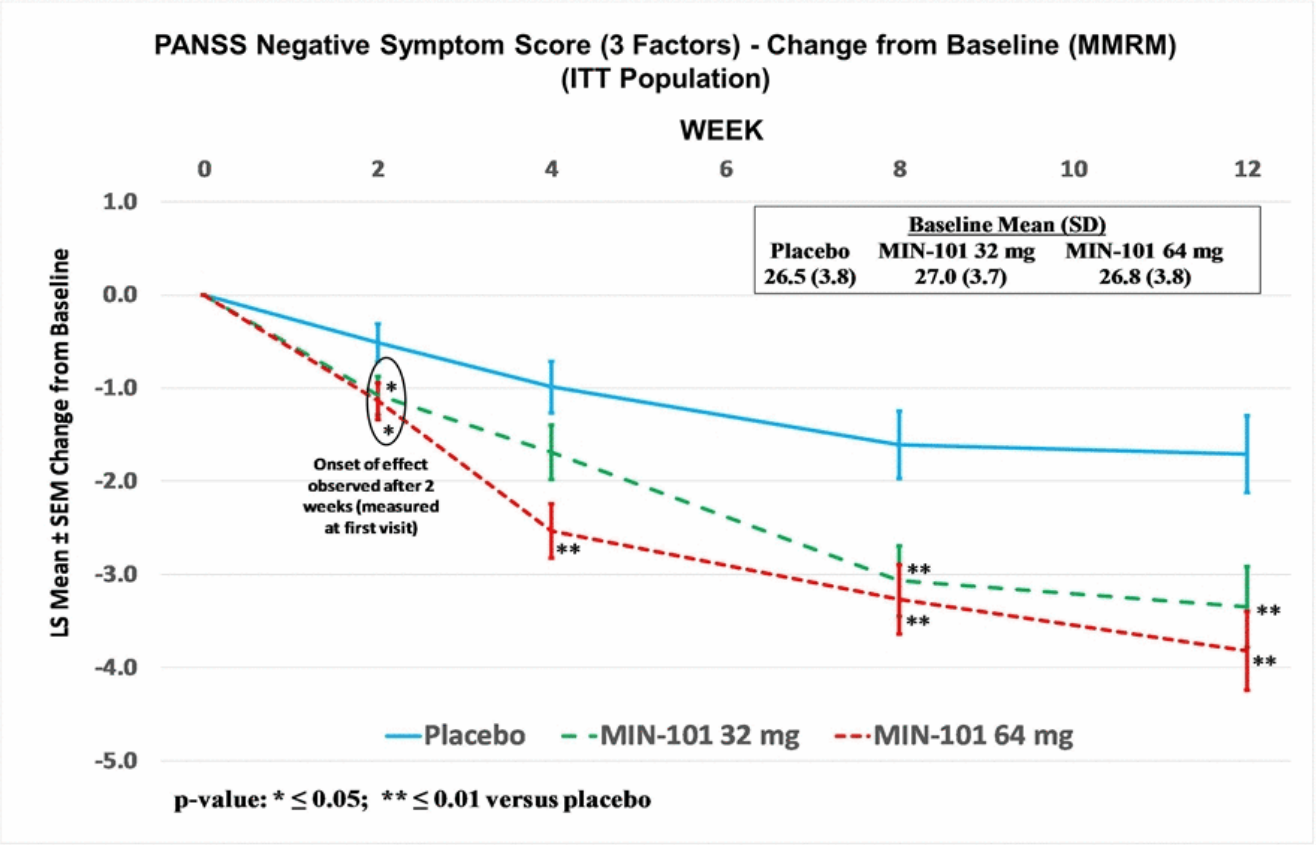

Efficacy: Secondary endpoint (2)
Demonstrated improvement in Total PANSS driven by improvement in negative symptoms

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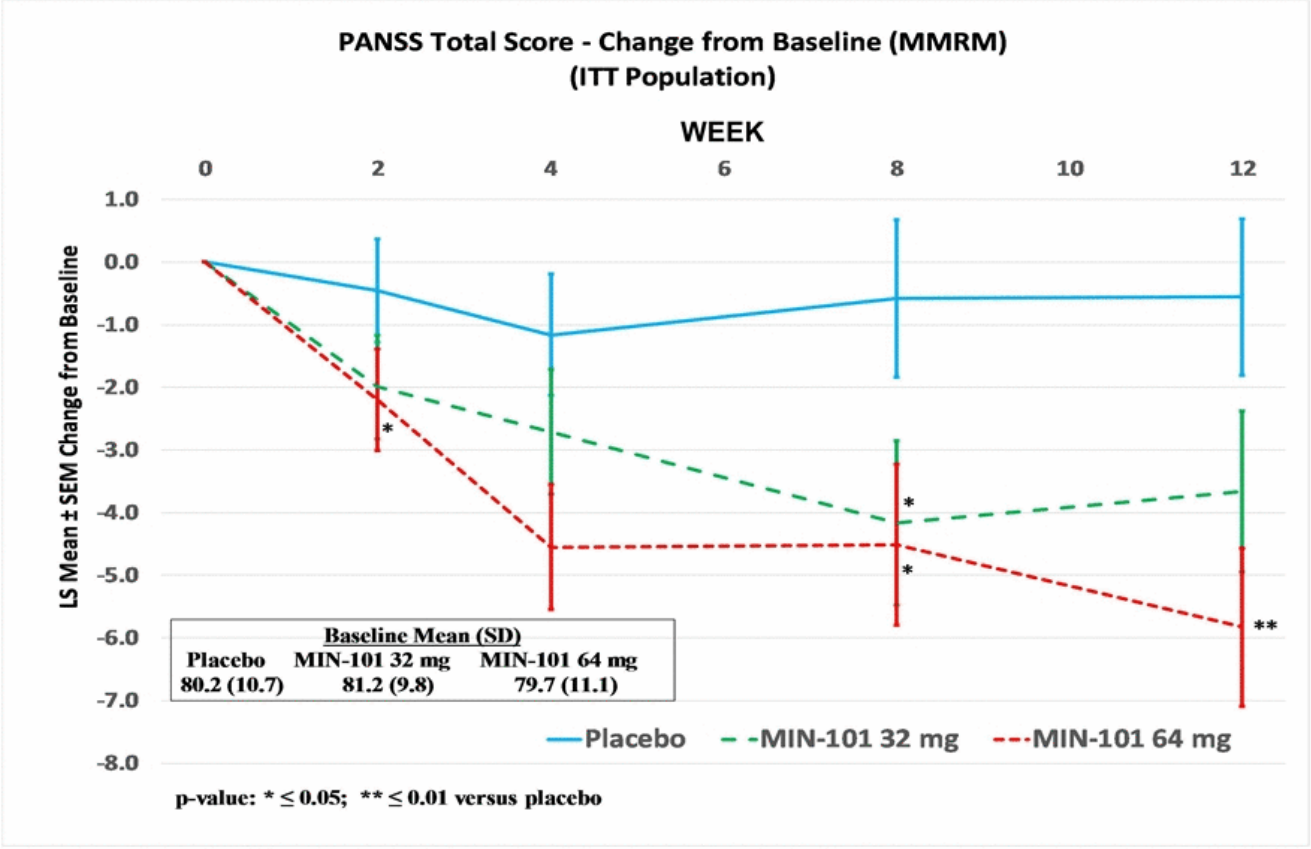

Efficacy: Secondary endpoint (4)
PANSS positive symptoms score (3 Factors) indicates MIN-101 maintains stability in positive symptoms

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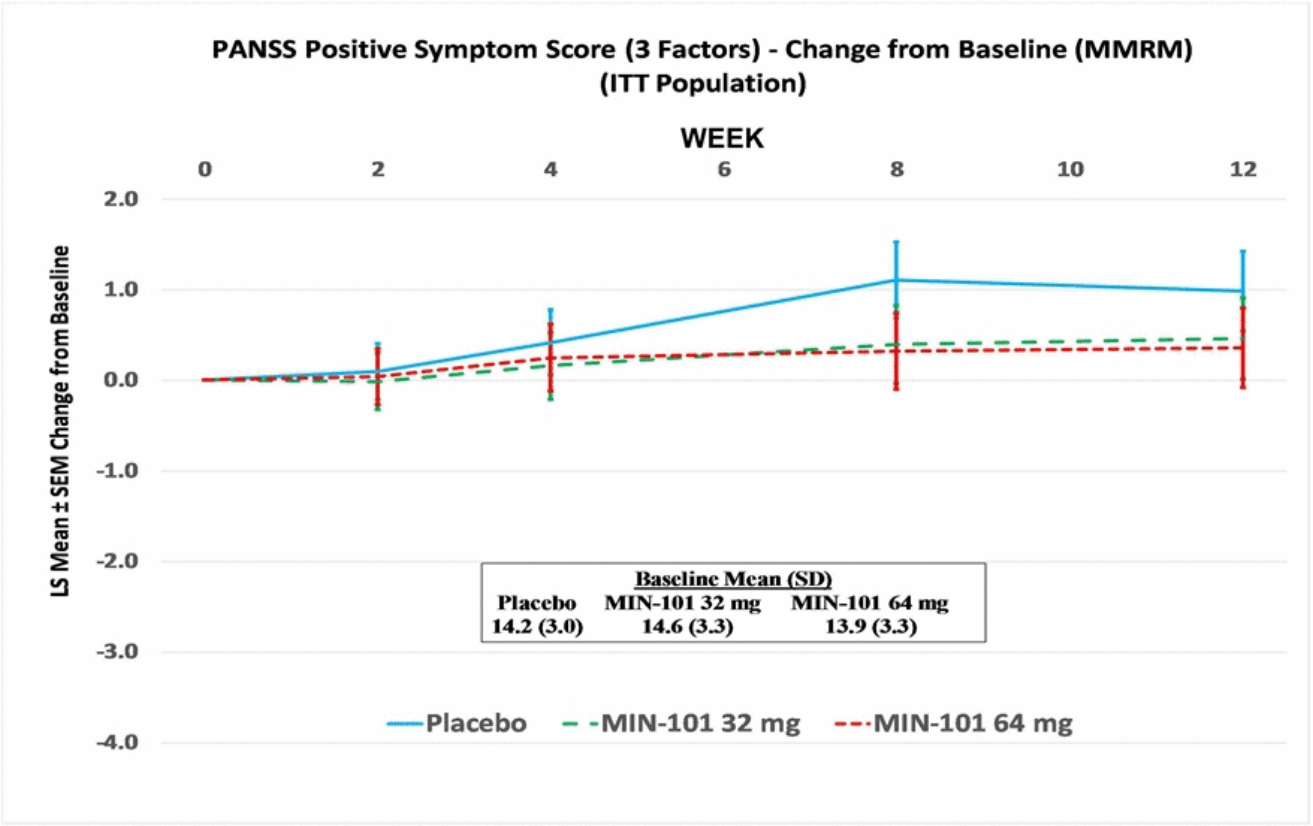

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Extension Phase

Baseline for patients who crossed from placebo to MIN-101 is start of open label (Week 12)

MIN-101C03: Negative Symptoms (Pentagonal Structure)
Continued improvement over 36 weeks in both doses

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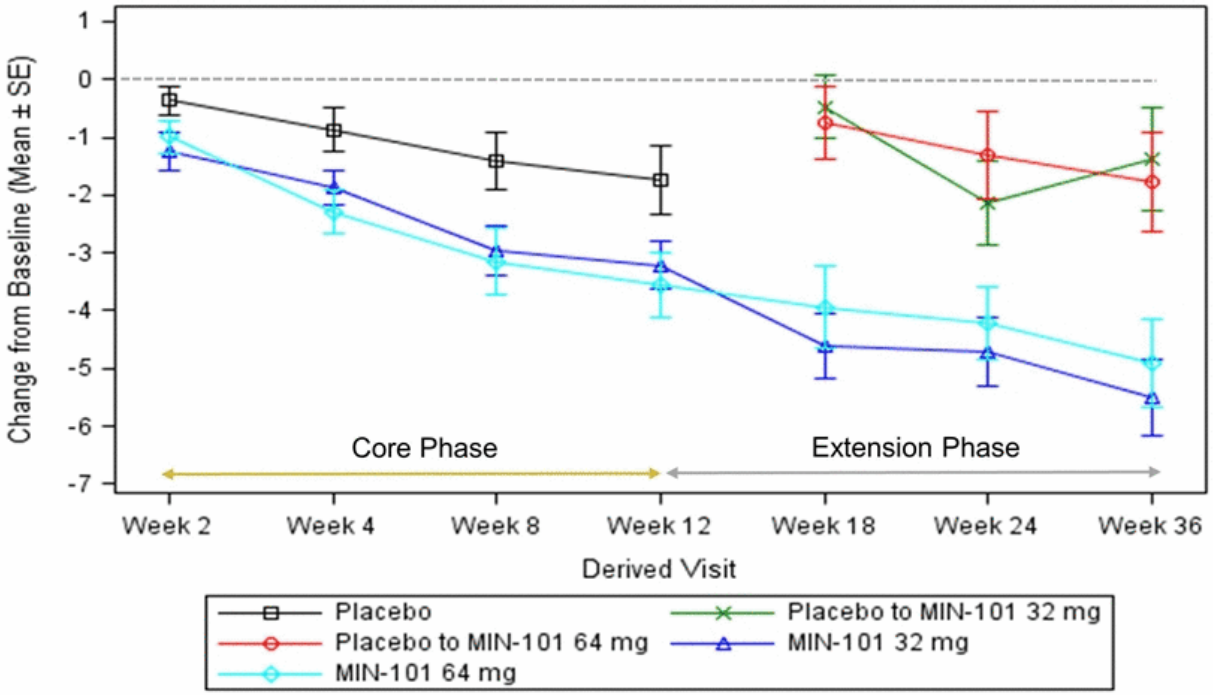

Baseline for Placebo-to-MIN-101 is From Start of Open Label (Week 12)

MIN-101C03: Positive Symptoms (3-Factors) Stable over 36 weeks in both doses

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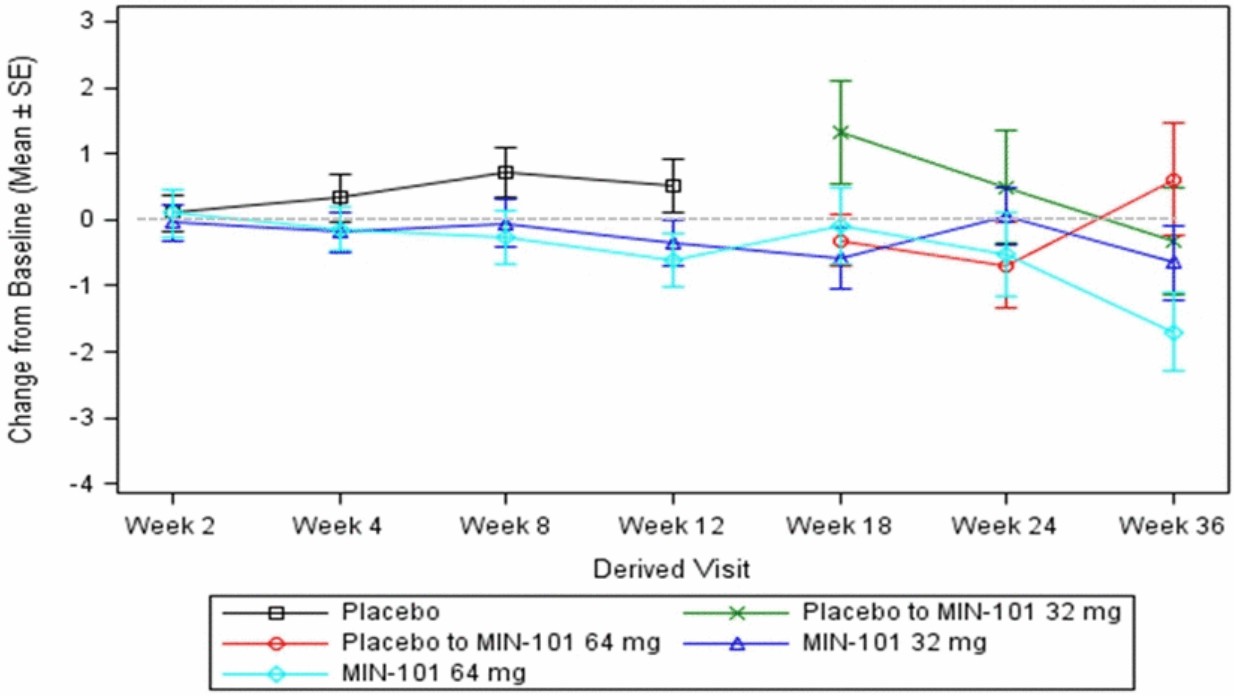

Baseline for Placebo-to-MIN-101 is From Start of Open Label

MIN-202

(JNJ42847922)

A drug to treat insomnia & depressive disorders by restoring physiological sleep

A co-development/co-commercialisation program with;

janssen | PHARMACEUTICAL COMPANIES OF Johnson&Johnson

Insomnia affects about 10% of adults and the majority of people with depression

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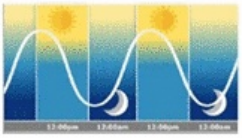
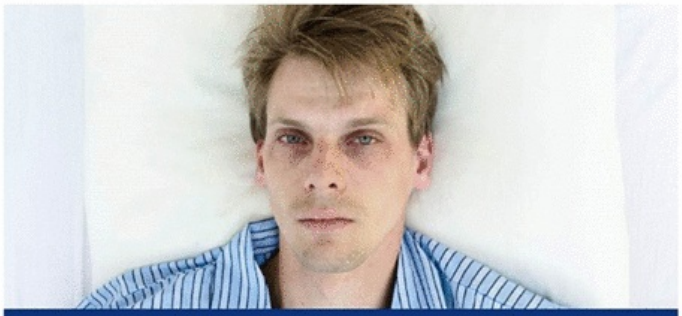
- ~85% of patients with major depressive disorder have symptoms of insomnia, which often persists despite treatment with currently available sleep medications
  - ~13.6 million Americans have major depression and insomnia
- Most existing treatments "force" sleep, rather than physiologically attenuating the "wake drive"
- The Orexin system regulates the wake drive



Circadian Rhythm

CNS *Spectr*. 2010 Jun;15(6):394-404.
Insomnia in patients with depression: a STAR*D report.
NIMH



What Do We Need?

Therapies that provide:

- A more physiological approach to treat insomnia
- Rapid onset of action
- Preservation of deep, restful sleep
- Minimal residual daytime sleepiness or cognitive impairment

MIN-202: Exploratory study in patients with MDD and comorbid insomnia (n=20) indicate significant improvements in key sleep metrics

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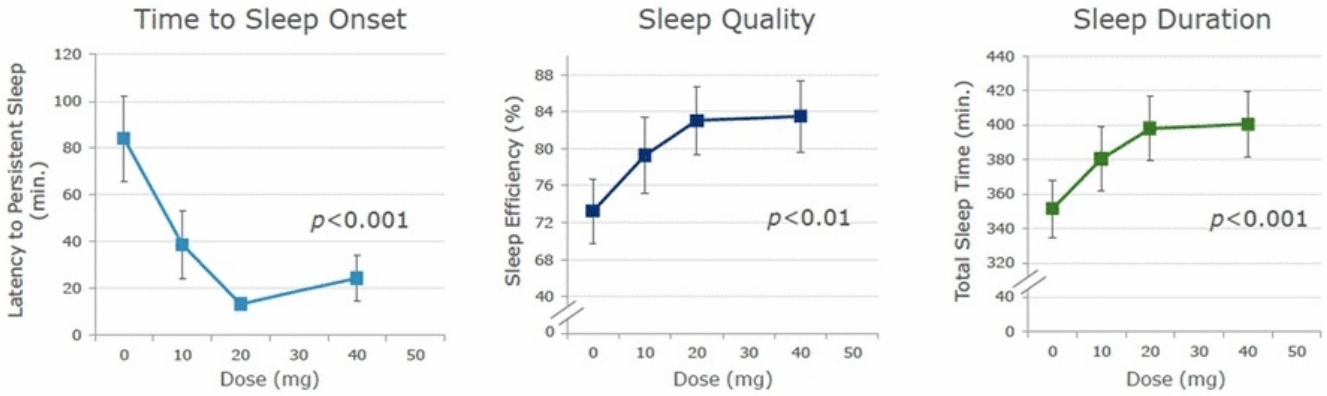

Reference: Internal data, study 42847922ED1002, disclosed by Minerva Neurosciences, Q1 2015.

Phase IIa in primary insomnia: achievement of primary endpoint of improvement in sleep efficiency complemented by increase in total sleep time

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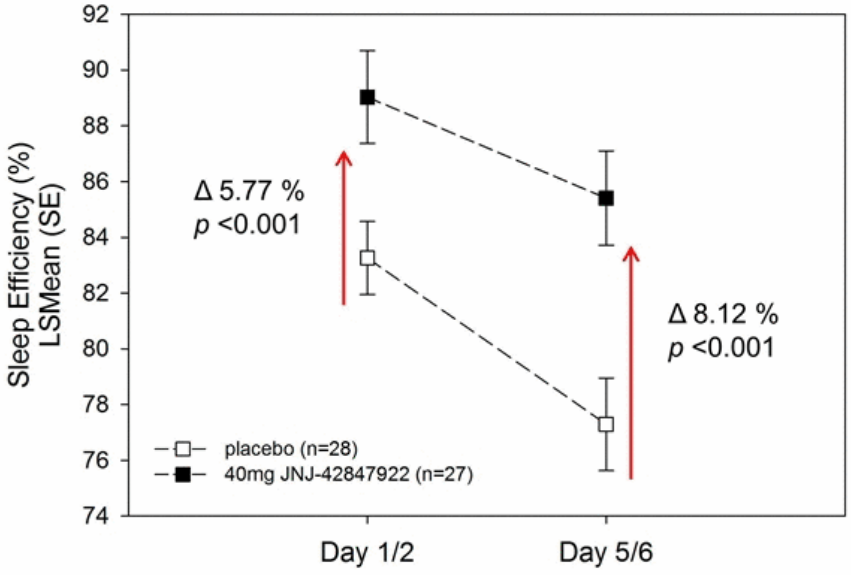
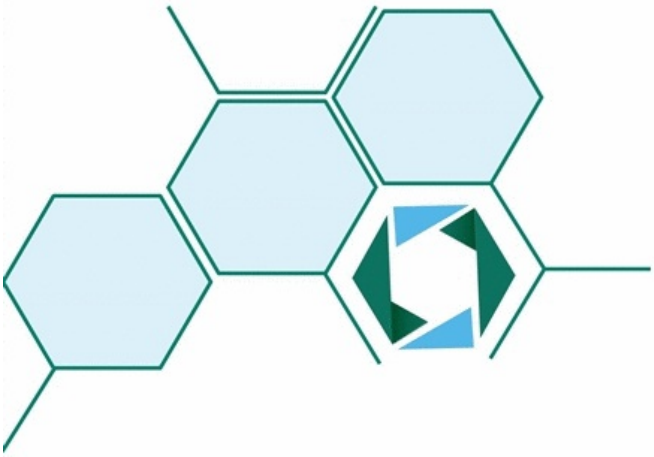
42847922ISM2002



Sleep Efficiency = (Total Sleep Time/480) * 100%

PSG recording = 480 min



MIN-117

Potential for a more effective and safer treatment to address the unmet medical needs of Major Depressive Disorder patients

Unmet need in Major Depressive Disorder: treatments with faster onset and better response, without side effects

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- Major depression: primary cause of disability worldwide by 2030[1]
- ~6 million patients in US with treatment- resistant depression[2]
- Only ~30% of patients achieve remission using current treatments[3]
- Current therapies have slow onset of effect; typically 4 – 8 weeks

What do we need?

Treatments that:

- Act rapidly
- Are effective in patients who do not respond to or receive only partial benefit from existing medicines
- Do not impair cognition or sexual function
- Free patients from debilitating side-effects
- Improve sleep

1. *World Health Organization, "Global Burden of Mental Disorders," 2011*
2. *IMS and Truven Health*
3. *Cleveland Clinic Journal of Medicine Volume 75. Number 1 January 2008*

MIN-117C01: Phase IIa study design

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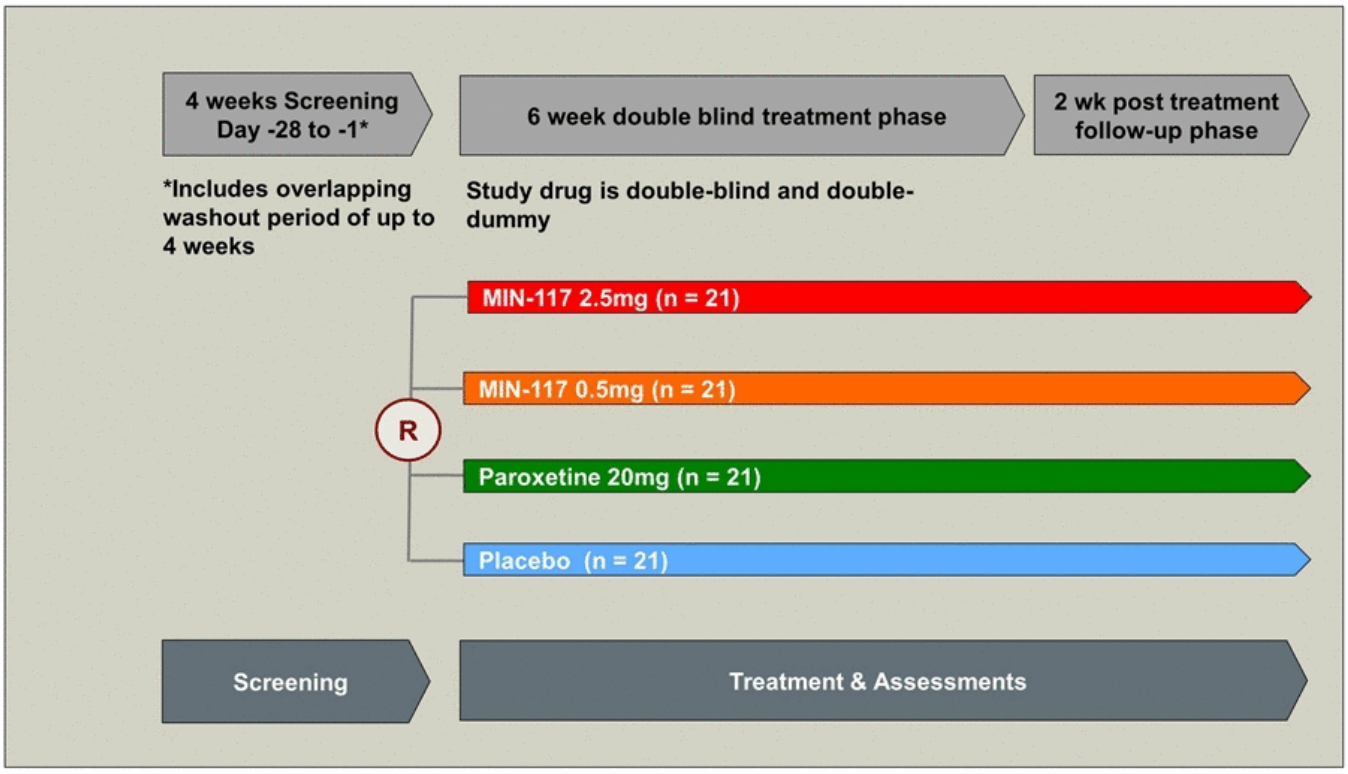

Phase IIa efficacy: MADRS primary endpoint
Clinically meaningful effect in a depressed patient population at both doses

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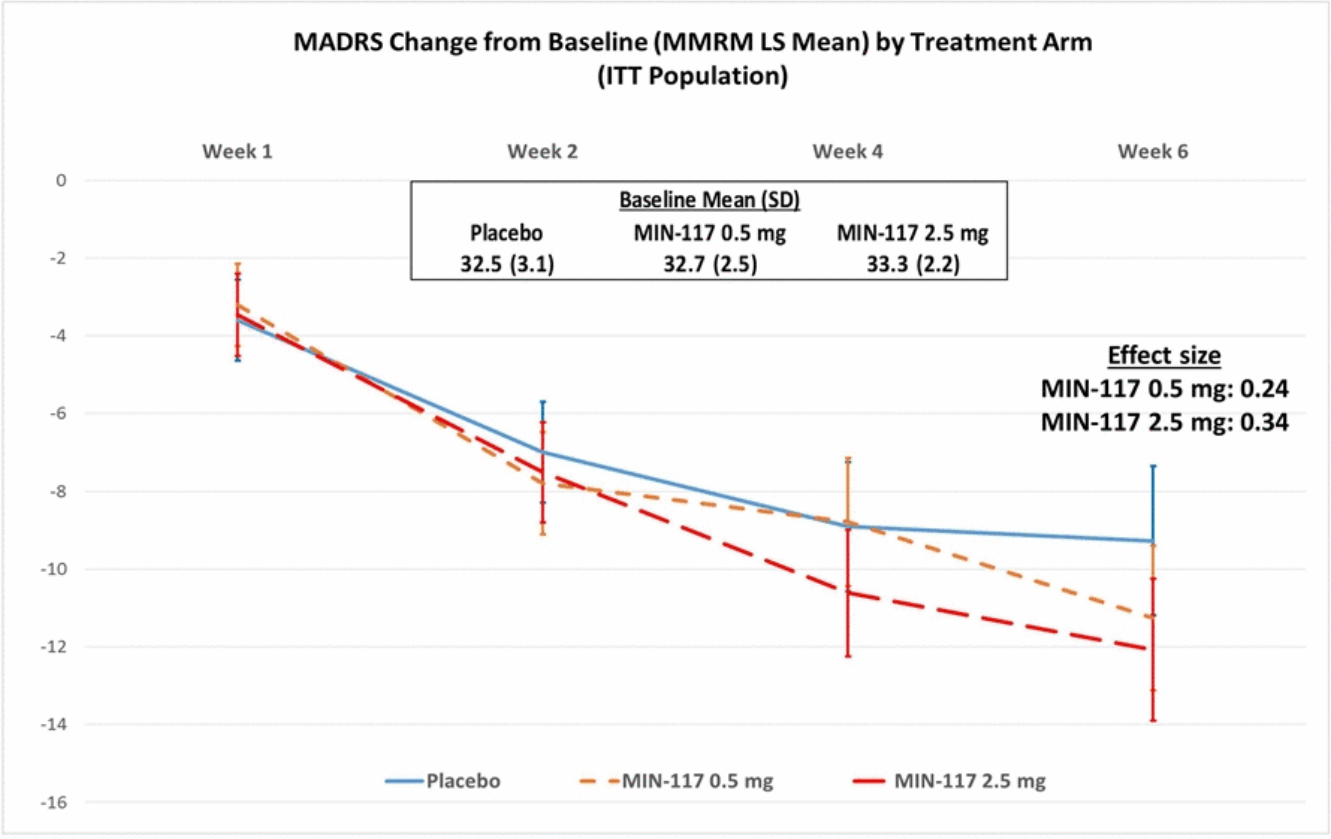

Phase IIa efficacy: HAM-A secondary endpoint
Unexpected effect on anxiety

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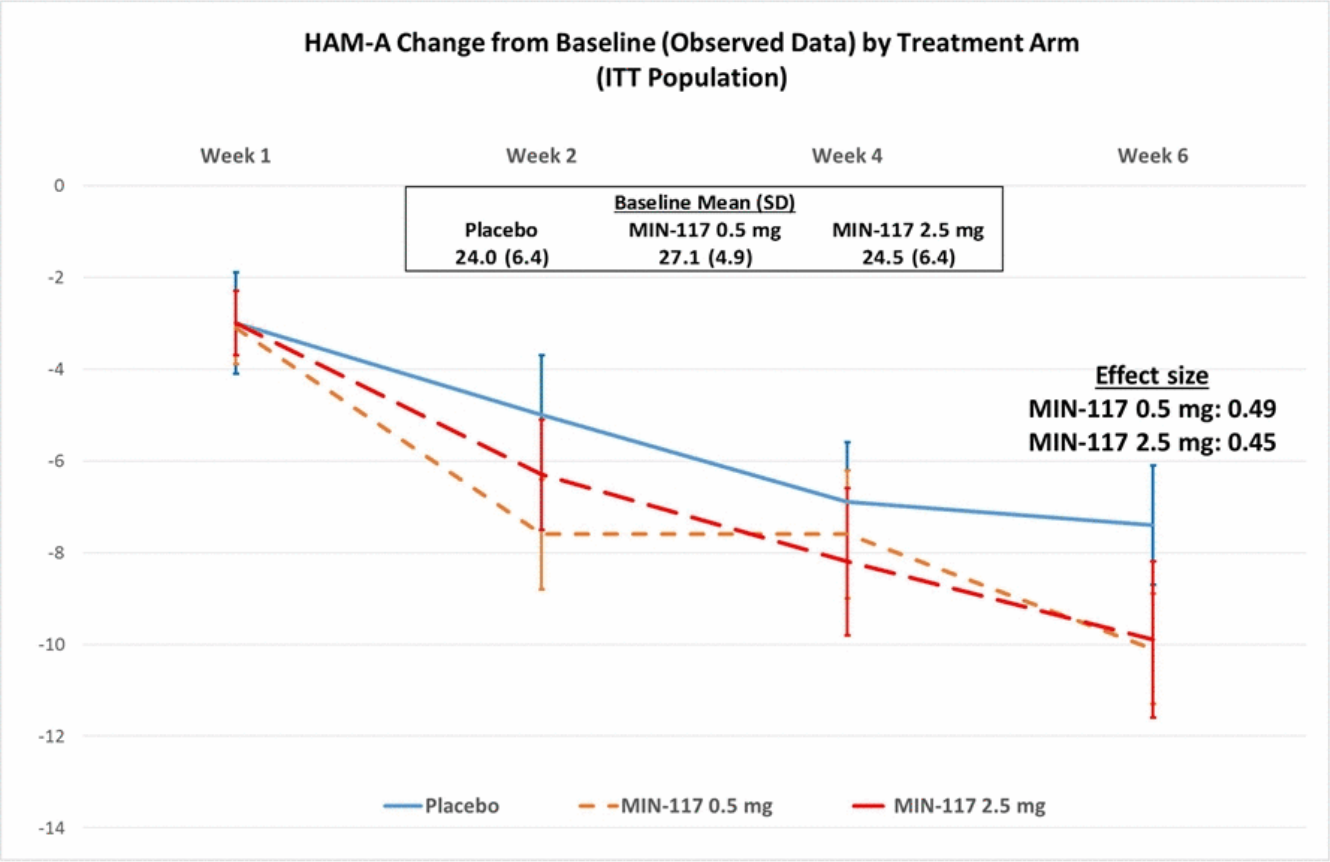

Sleep polysomnography: REM latency

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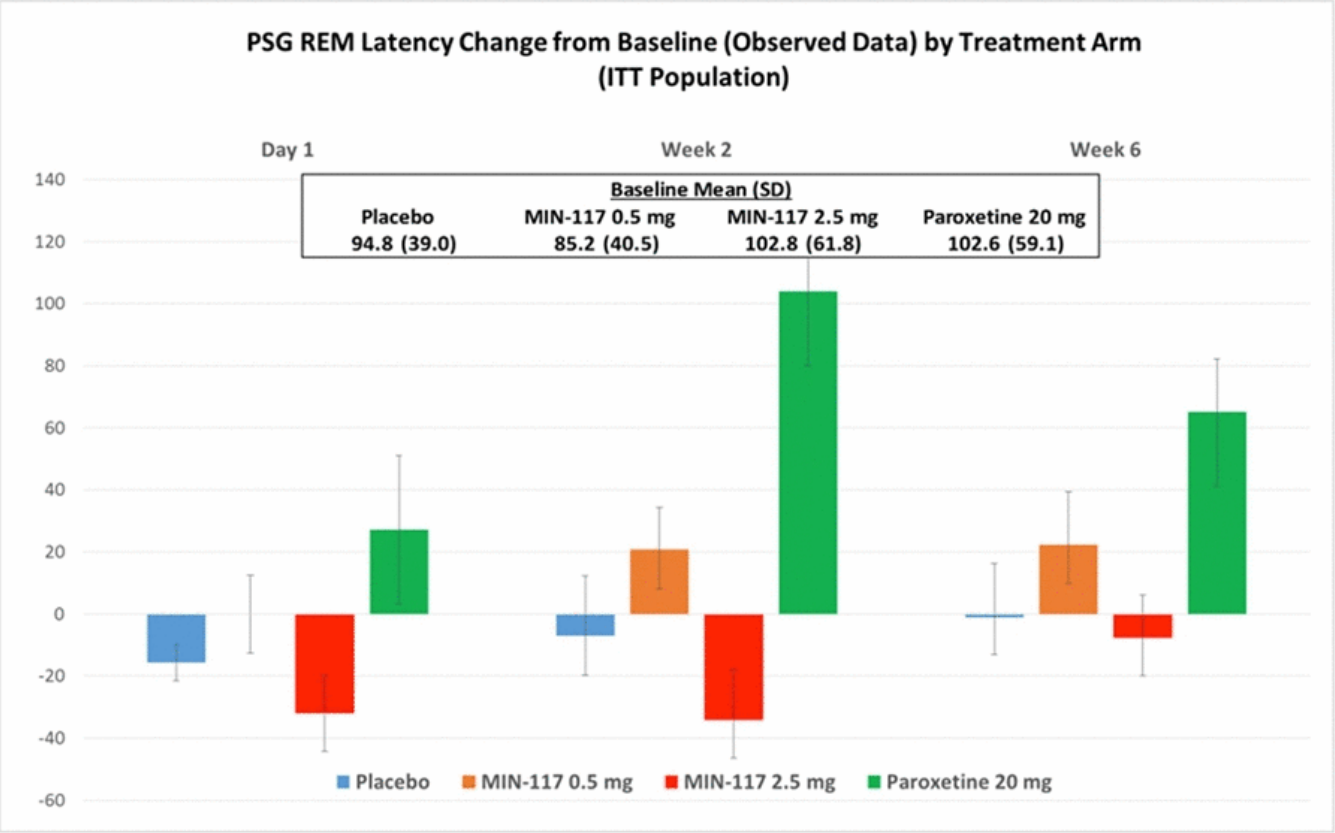

MIN-301

A protein drug with disease modifying potential for the treatment of unmet medical needs in major CNS indications

Parkinson's Disease
Large and growing prevalence with huge burden to patients, families and society

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Caused by a cascade of events leading to the death of dopamine-generating cells

- Progressive and incurable
- Leads to lower quality of life, disability
- Loss of speech, mobility, cognitive abilities
- Lower life expectancy

- Parkinson's disease is a chronic, degenerative neurological disorder that affects one in 100 people over age 60.
- The average age at onset is 60
- There is no objective test, or biomarker
- Estimates of the number of people living with the disease vary but recent research indicates that at least one million people in the US and more than 5 million worldwide have the disease

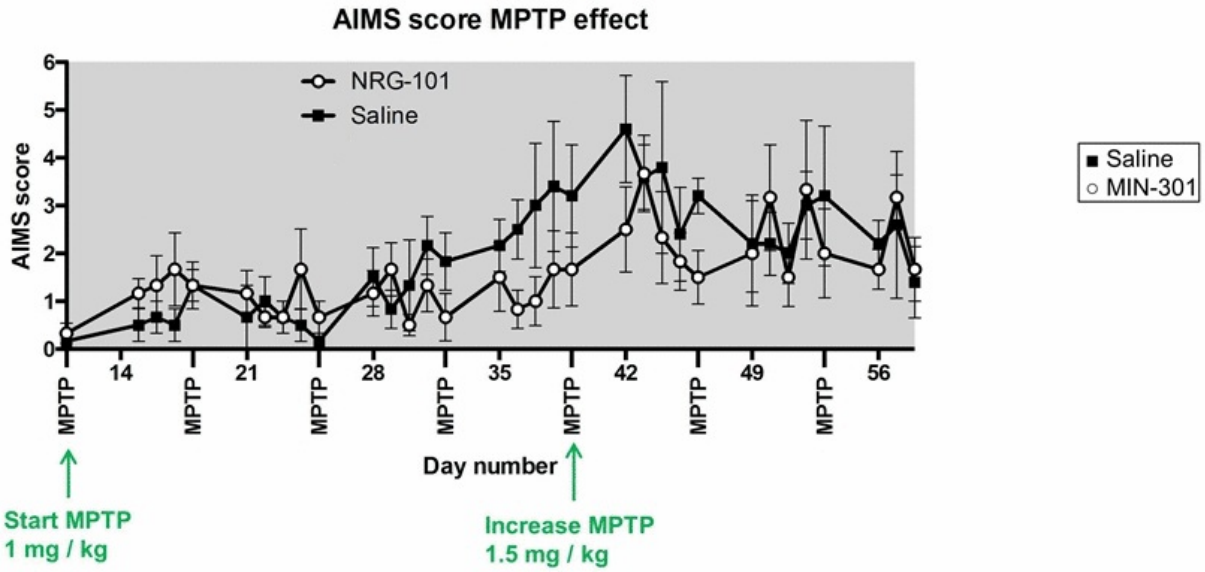
What do we need?

Treatments that;

- **Are disease modifying**
- **Have less side effects**
- **Treat all symptoms particularly cognitive decline and not just the motor impairment**

Animal models (non-human primates):
Effect of treatment on abnormal involuntary movements scale (AIMS)

MINERVA NEUROSCIENCES, INC.



- **Clear MPTP-induced increase in AIMS scores**
- **Scores in MIN-301-treated animals lower during low MPTP (< 1 mg/kg) induction as compared to placebo**

Upcoming milestones and value drivers

MINERVA NEUROSCIENCES, INC.

Program	Primary Indication	Status
MIN-101	Schizophrenia	• End of Phase II meeting with FDA in early Q2 2017 • Initiation of pivotal Phase III trial planned in Q3 2017
MIN-202	Primary Insomnia and Major Depressive Disorder	• Phase II trial preparation underway • Next trials in insomnia disorder and MDD planned in 2017
MIN-117	Major Depressive Disorder	• Planning underway for next phase of clinical trials expected to begin in 2017
MIN-301	Parkinson's Disease	• IND or IMPD, with Phase I expected to initiate thereafter

Financial position

MINERVA NEUROSCIENCES, INC.

- ~$91.9 million cash balance (cash, cash equivalents and marketable securities) at September 30, 2016
  - sufficient to fund operations into 2018
- 2016 quarterly R&D expense (Q1 – Q3) : approx. $2.7m - $5.9m
- Shares outstanding at October 28, 2016: ~34.8 million (~40.8 million fully diluted)