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): December 9, 2015

Intra-Cellular Therapies, Inc.
(Exact name of registrant as specified in its charter)

Commission File Number: 001-36274

Delaware	**36-4742850**
(State or other jurisdiction of incorporation)	**(IRS Employer Identification No.)**

430 East 29th Street
New York, New York 10016
(Address of principal executive offices, including zip code)

(212) 923-3344
(Registrant's telephone number, including area code)

Not applicable
(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.

On December 9, 2015, Intra-Cellular Therapies, Inc. (the "Company") announced additional data from the first Phase 3 clinical trial of its lead drug candidate, ITI-007 (ITI 007-301), and the ITI-007 Positron Emission Tomography ("PET") study in patients with schizophrenia.

The Company's press release announcing additional data from the PET study and the Phase 3 clinical trial of ITI-007-301, and the related posters presented at the 54th Annual Meeting of the American College of Neuropsychopharmacology, are filed as Exhibits 99.1, 99.2 and 99.3, respectively, to this Current Report on Form 8-K and are incorporated herein by reference.

ITEM 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Description
99.1	Press release dated December 9, 2015
99.2	Poster presentation, entitled "Clinical Development of ITI-007 for the Treatment of Schizophrenia"
99.3	Poster presentation, entitled "Further Characterizing Brian Receptor Occupancy with ITI-007: Results from a Positron Emission Tomography (PET) Study in Patients with Schizophrenia"

SIGNATURES

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.

INTRA-CELLULAR THERAPIES, INC.

By: /s/ Lawrence J. Hineline
Lawrence J. Hineline
Vice President of Finance, Chief Financial Officer, Treasurer and Assistant Secretary

Date: December 10, 2015

Exhibit 99.1

Intra-Cellular Therapies Presents Additional Efficacy and Safety Data From the Positive Phase 3 Clinical Trial of ITI-007 for the Treatment of Schizophrenia and From the Positron Emission Tomography Study

Intra-Cellular Therapies to Host a Conference Call Thursday, December 10, 2015 at 8:30 am EST to Discuss the Data

NEW YORK, December 9, 2015 (GLOBE NEWSWIRE) — Intra-Cellular Therapies, Inc. (Nasdaq:ITCI), a biopharmaceutical company focused on the development of therapeutics for central nervous system (CNS) disorders, today announced additional data from the first Phase 3 clinical trial of ITI-007 for the treatment of patients with schizophrenia (ITI-007-301) and the ITI-007 Positron Emission Tomography (PET) study in patients with schizophrenia at the 54th annual meeting of the American College of Neuropsychopharmacology (ACNP) in Hollywood, Florida.

Poster W166 entitled "Clinical Development of ITI-007 for the Treatment of Schizophrenia" described additional data from the ITI-007-301 trial, topline results of which were announced in September 2015.

ITI-007 60 mg improved symptoms of schizophrenia and met the primary endpoint demonstrating statistically significant superiority over placebo at Day 28 as measured by the Positive and Negative Syndrome Scale (PANSS) total score. The 40 mg dose approximated the trajectory of improvement seen with the 60 mg dose, but the effect with 40 mg did not reach statistical significance on the PANSS total score.

Both the 60 mg and 40 mg doses of ITI-007 significantly reduced the PANSS positive symptom subscale score versus placebo at study endpoint and at earlier time points.

ITI-007 60 mg met the key secondary endpoint demonstrating statistically significant improvement on the Clinical Global Impression scale for Severity of Illness (CGI-S). ITI-007 40 mg also demonstrated a statistically significant improvement versus placebo on the CGI-S. The CGI-S is a well-established and clinically useful rating tool which provides a clinician's view of the patient's global level of illness severity.

Moreover, ITI-007 significantly improved social function as evidenced by improvements in the PANSS-derived Prosocial Factor and the Personal and Social Performance Scale. ITI-007 qualitatively improved the PANSS negative symptoms subscale score in this acute patient population.

ITI-007 was well-tolerated and demonstrated a safety profile that did not differ from placebo. This study had a high percentage of patients completing treatment, with time to treatment discontinuation (due to any reason) being statistically significantly better with 60 mg ITI-007 than with placebo. The only treatment-emergent adverse events considered at least possibly related to ITI-007, administered orally once daily in the morning, occurring in greater than 5% of patients and at least twice the rate of placebo were somnolence, sedation, and fatigue, all predominantly mild.

ITI-007's motoric, metabolic, and cardiovascular profile was similar to placebo, and there were no clinically significant changes in akathisia, extrapyramidal symptoms, prolactin, body weight, glucose, insulin, and lipids.

Second generation antipsychotic drugs (SGAs) for schizophrenia expose patients to increased risk of diabetes and other associated diseases including cardiovascular disease, resulting in a significant burden on our healthcare system. SGAs also have motoric adverse events impacting patient quality of life, often leading to poor medication adherence. The Company believes existing data suggest that ITI-007 does not impact these metabolic, cardiovascular and motoric parameters in patients with schizophrenia.

"The Phase 3 trial demonstrated that ITI-007 is efficacious in the treatment of patients with schizophrenia while possessing a favorable safety and tolerability profile particularly in relation to metabolic, motoric and cardiovascular parameters," said Dr. Sharon Mates, Chairman and CEO of Intra-Cellular Therapies. "These data are

consistent with prior results seen in our Phase 2 study and add further evidence to our belief that ITI-007 may represent an improvement on existing therapies for patients with schizophrenia."

Poster W174 entitled "Further Characterizing Brain Receptor Occupancy with ITI-007: Results from a Positron Emission Tomography (PET) Study in Patients" highlighted data from the ITI-007 PET study in patients with schizophrenia.

ITI-007 was safe and well tolerated in this study. In this study, mean striatal D2 receptor occupancy at an effective antipsychotic dose of 60 mg ITI-007 was about 40%. This PET study in patients with stable schizophrenia further adds to the information gleaned regarding brain receptor occupancy levels from a prior PET study in healthy volunteers. ITI-007 demonstrates relatively low striatal D2 receptor occupancy at an antipsychotic efficacious dose, and has a decreased risk for induction of D2 mediated side effects, including extrapyramidal side effects, akathisia, and hyperprolactinemia. Together, these data suggest that ITI-007 may represent an exciting new first-in-class treatment for schizophrenia.

Conference Call and Webcast Details

The Company will host a live conference call and webcast December 10, 2015 at 8:30 AM Eastern Standard Time to discuss the additional data presented at ACNP. The live webcast and subsequent replay may be accessed by visiting the Company's website at www.intracellulartherapies.com. Please connect to the Company's website at least 5-10 minutes prior to the live webcast to ensure adequate time for any necessary software download. Alternatively, please call 1-844-835-6563 (U.S.) or 1-970-315-3916 (international) to listen to the live conference call. The conference ID number for the live call is 2009462. Please dial in approximately 10 minutes prior to the call. The webcast will be available on the Company's website until December 14, 2015.

About ITI-007

ITI-007 is our lead drug development candidate with mechanisms of action that, we believe, have the potential to yield a first-in-class antipsychotic therapy. In our pre-clinical and clinical trials to date, ITI-007 combines potent serotonin 5-HT2A receptor antagonism, dopamine receptor phosphoprotein modulation (DPPM), glutamatergic modulation and serotonin reuptake inhibition into a single drug candidate for the treatment of acute and residual schizophrenia. At dopamine D2 receptors, ITI-007 has been demonstrated to have dual properties and to act as both a post-synaptic antagonist and a pre-synaptic partial agonist. ITI-007 has also been demonstrated to stimulate phosphorylation of glutamatergic NMDA GluN2B receptors in a mesolimbic specific manner. We believe that this regional selectivity in brain areas thought to mediate the efficacy of antipsychotic drugs, together with serotonergic, glutamatergic, and dopaminergic interactions, may result in antipsychotic efficacy for positive, negative, affective and cognitive symptoms associated with schizophrenia. The serotonin reuptake inhibition could allow for antidepressant activity for the treatment of schizoaffective disorder, co-morbid depression, and/or as a stand-alone treatment for major depressive disorder. We believe ITI-007 may also be useful for the treatment of bipolar disorder and other psychiatric and neurodegenerative disorders, particularly behavioral disturbances associated with dementia, autism and other CNS diseases.

About Schizophrenia

Schizophrenia is a disabling and chronic mental illness affecting over 1% of the world's population. Schizophrenia is characterized by multiple symptoms during an acute phase of the disorder that can include so-called "positive" symptoms, such as hearing voices, disorganized thinking, grandiose beliefs and suspiciousness or paranoia. These symptoms can be accompanied by additional, harder-to-treat symptoms, such as social withdrawal and blunted emotional response and expression, collectively referred to as "negative" symptoms, difficulty concentrating or cognitive

impairment, depression, and insomnia. Such residual symptoms often persist even after the acute positive symptoms subside, and contribute substantially to the social and employment disability associated with schizophrenia. Current antipsychotic medications provide some relief for the symptoms associated with the acute phase of the disorder, but they do not effectively treat the residual phase symptoms and psychosocial impairment associated with chronic schizophrenia. Currently available medications used to treat acute schizophrenia are limited in their use due to side effects that can include movement disorders, weight gain, metabolic disturbances, and cardiovascular disorders. There is an unmet medical need for new therapies that have improved side effect and efficacy profiles.

About Intra-Cellular Therapies

Intra-Cellular Therapies is developing novel drugs for the treatment of neuropsychiatric and neurodegenerative diseases and diseases of the elderly, including Parkinson's and Alzheimer's disease. The Company is developing its lead drug candidate, ITI-007, for the treatment of schizophrenia, bipolar disorder, behavioral disturbances in dementia, depression and other neuropsychiatric and neurological disorders. ITI-007, a first-in-class molecule, is in Phase 3 clinical development for the treatment of schizophrenia. The Company is also utilizing its phosphodiesterase platform and other proprietary chemistry platforms to develop drugs for the treatment of CNS and other disorders.

Forward-Looking Statements

This news release contains "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995 that involve risks and uncertainties that could cause actual results to be materially different from historical results or from any future results expressed or implied by such forward-looking statements. Such forward-looking statements include statements regarding, among other things, our clinical and non-clinical development plans; the progress, timing and results of our clinical trials; the safety and efficacy of our product development candidates; our beliefs about the potential uses and benefits of ITI-007; and our research and

development efforts and plans under the caption “About Intra-Cellular Therapies.” All such forward-looking statements are based on management’s present expectations and are subject to certain factors, risks and uncertainties that may cause actual results, outcome of events, timing and performance to differ materially from those expressed or implied by such statements. These risks and uncertainties include but are not limited to the following: our current and planned clinical trials, other studies for ITI-007, and our other product candidates may not be successful or may take longer and be more costly than anticipated; product candidates that appeared promising in earlier research and clinical trials may not demonstrate safety and/or efficacy in larger-scale or later clinical trials; our reliance on collaborative partners and other third parties for development of our product candidates; and the other risk factors discussed under the heading “Risk Factors” contained in our Annual Report on Form 10-K for the year ended December 31, 2014 filed with the Securities and Exchange Commission (SEC), as well as any updates to those risk factors filed from time to time in our periodic and current reports filed with the SEC. All statements contained in this press release are made only as of the date of this press release, and we do not intend to update this information unless required by law.

Contact:

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Vice President
Corporate Communications and Investor Relations of Intra-Cellular Therapies, Inc.
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Exhibit 99.2

Clinical Development of ITI-007 for the Treatment of Schizophrenia

Kimberly E Vanover, Robert E Davis, Cedric O'Gorman, Jelena Saillard, Michal Weingart, Sharon Mates
Intra-Cellular Therapies Inc., New York, NY

ABSTRACT

Schizophrenia is a devastating and serious mental illness afflicting approximately 1 percent of the population resulting in high rates of disability to patients and a high burden to their caregivers. It also exerts an enormous toll in terms of healthcare costs. Schizophrenia ranks in the top 10 leading causes of disability in the world. Despite the introduction of neuroleptics in the 1950s and the advance of atypical antipsychotic therapy since the introduction of clozapine, there still remains an unmet need for newer treatments which address a broad spectrum of schizophrenia symptoms including positive, negative and depressive symptoms without concomitant high rates of motor disturbances, metabolic syndrome, and/or cardiovascular risk.

ITI-007 is an investigational new drug in late-stage clinical development for schizophrenia. Through synergistic actions via serotonergic, dopaminergic and glutamatergic systems, ITI-007 represents a novel approach to the treatment of schizophrenia and other neuropsychiatric disorders. ITI-007 is a potent antagonist at 5-HT2A receptors, a mesolimbic/mesocortical dopamine phosophoprotein modulator (DPPM) with activity as a pre-synaptic partial agonist and post-synaptic antagonist at dopamine D2 receptors, a mesolimbic glutamate GluN2B receptor phosphoprotein modulator and a serotonin reuptake inhibitor[1]. This unique pharmacology has been predicted to translate clinically, in a dose dependent manner, into broad antipsychotic efficacy for the treatment of positive and negative symptoms with improved cognition, affective symptoms, and sleep.

Methods The ITI-007 schizophrenia program includes three randomized, double-blind, placebo-controlled clinical trials in patients with acute schizophrenia: ITI-007-005, ITI-007-301, and ITI-007-302.

In the Phase 2 trial ITI-007-005, patients were randomized to receive one of four oral treatments once daily for 4 weeks: 60 mg ITI-007, 120 mg ITI-007, 4 mg risperidone (positive control) or placebo in a 1:1:1:1 ratio. In the first Phase 3 trial ITI-007-301, patients were randomized to receive one of three oral treatments once daily for 4 weeks: 60 mg ITI-007, 40 mg ITI-007, or placebo in a 1:1:1 ratio. In the second Phase 3 trial ITI-007-302 (clinical conduct ongoing), patients are randomized to receive one of four oral treatments once daily for 6 weeks: 60 mg ITI-007, 20 mg ITI-007, 4 mg risperidone (positive control) or placebo in a 1:1:1:1 ratio.

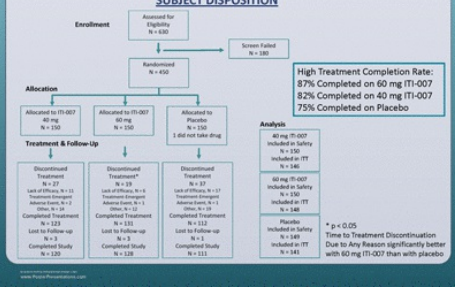
The Phase 2 trial ITI-007-005 was completed in November 2013 with 335 patients randomized. The first Phase 3 trial ITI-007-301 was completed in July 2015 with 450 patients randomized. The second Phase 3 ITI-007-302 trial is ongoing. In all studies the primary efficacy endpoint is change from baseline in the total PANSS score versus placebo at end of treatment.

Results In Phase 2, ITI-007 60 mg significantly improved schizophrenia symptoms on the primary endpoint (least squares [LS] mean change -13.2 points versus -7.4 points; P=0.017, MMRM, ES=0.4)[2]. ITI-007 120 mg did not significantly separate from placebo on the total PANSS at Day 28 (LS mean change -8.3 versus -7.4; P=0.708). Risperidone (4 mg) differed from placebo on the total PANSS demonstrating assay sensitivity (least squares [LS] mean change -13.4 points versus -7.4 points; P=0.013, MMRM, ES=0.4). ITI-007 was safe and well-tolerated, comparable to placebo on safety measures in this trial. Secondary analyses indicated improved negative symptoms and symptoms of depression, particularly in pre-specified subgroups with prominent negative symptoms and depression at baseline. Data analysis for the first Phase 3 trial ITI-007-301 are presented here.

Discussion ITI-007 represents a new approach for the treatment of schizophrenia with unique pharmacology as well as a differentiating clinical profile. Data from the ongoing late-stage schizophrenia program for ITI-007 continue to further characterize ITI-007's novel mechanism of action as well as the potential clinical benefits, in terms of efficacy and safety for patients.

[1]Snyder et al., 2015 Psychopharmacology 232:605-621
[2]Lieberman et al., 2015 Biological Psychiatry online, ahead of print

SUBJECT DISPOSITION



High Treatment Completion Rate:
87% Completed on 60 mg ITI-007
82% Completed on 40 mg ITI-007
75% Completed on Placebo

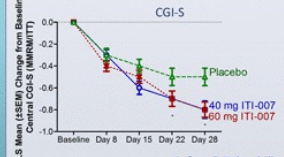
* p < 0.05
Time to Treatment Discontinuation Due to Any Reason significantly better with 60 mg ITI-007 than with placebo

RESULTS

60 mg ITI-007 Met Primary and Key Secondary Endpoints

60 mg ITI-007 separated from placebo as early as week 1, and maintained efficacy at every time point on PANSS total score.

* p < 0.05 versus placebo

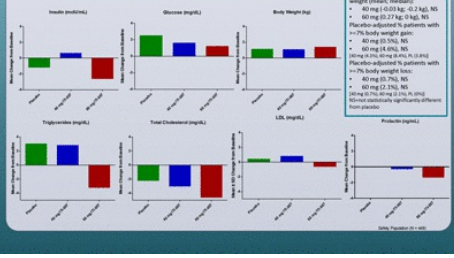


Both 40 mg and 60 mg were statistically significantly superior to placebo on CGI-S at study day 22 and 28.

* p < 0.05 versus placebo

Safety & Tolerability

ITI-007 had a favorable safety and tolerability profile as evidenced by motoric, metabolic, and cardiovascular characteristics similar to placebo.



Placebo-adjusted change in body weight (means; median):
- 40 mg (-0.03 kg; -0.2 kg), NS
- 60 mg (0.27 kg; 0 kg), NS

Placebo-adjusted % patients with >=7% body weight gain:
- 40 mg (0.5%), NS
- 60 mg (4.6%), NS

(40 mg (4.3%; 40 mg (8.4%); PI: (1.6%))

Placebo-adjusted % patients with >=7% body weight loss:
- 40 mg (0.7%), NS
- 60 mg (2.1%), NS

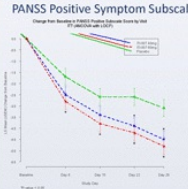
(40 mg (0.7%; 40 mg (1.1%; PI: (0%))

NS=not statistically significantly different from placebo

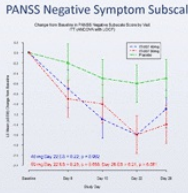
Safety Population (N = 445)

ITI-007 Significantly Improves Positive Symptoms and Social Function and Qualitatively Improves Negative Symptoms in the Acute Patient Population

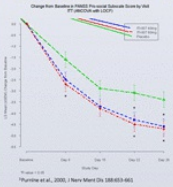
PANSS Positive Symptom Subscale



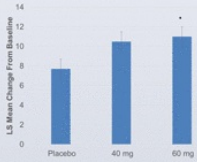
PANSS Negative Symptom Subscale



PANSS Prosocial Symptom Factor[3]



PSP for Psychosocial Function



[3]Purdon et al., 2000, J Neuropsychiatry Clin Neurosci 12:65-661

CONCLUSIONS

- ITI-007 60 mg met the primary endpoint at Week 4 as measured by PANSS Total Score, and showed significant antipsychotic efficacy as early as week 1, which was maintained at every time point throughout the study
- ITI-007 60 mg met the key secondary endpoint of statistically significant improvement on the CGI-S and demonstrated significant improvement in psychosocial functioning as measured by PANSS Derived Prosocial Factor and the PSP
- ITI-007 showed a dose-related improvement in symptoms of schizophrenia; 40 mg ITI-007 significantly improved CGI-S, Positive Subscale, and Prosocial Factor
- The only treatment-emergent adverse events considered at least possibly related to ITI-007 administered orally once daily in the morning that occurred in greater than 5% of patients and at least twice the rate of placebo were somnolence, sedation, and fatigue, all predominantly mild
- There was a significantly higher completion rate with 60 mg ITI-007 compared to placebo; despite the higher completion rate with 60 mg, there was no significant difference in weight gain from placebo
- ITI-007 was safe and well-tolerated with motoric, metabolic, and cardiovascular characteristics similar to placebo

DISCLOSURES

KEV, RED, CO'G, JS, MW and SM are full time employees of Intra-Cellular Therapies, Inc. (ITI).

Contact: cogorman@intracellulartherapies.com

Further Characterizing Brain Receptor Occupancy with ITI-007: Results from a Positron Emission Tomography (PET) Study in Patients with Schizophrenia

Kimberly E Vanover[1], Robert E Davis[1], Yun Zhou[2], Weiguo Ye[2], Cedric O'Gorman[1], Jelena Saillard[1], Michal Weingart[1], Robert Litman[3], Sharon Mates[1], Dean Wong[2]

[1]Intra-Cellular Therapies Inc., New York, NY; [2]Johns Hopkins University School of Medicine, Baltimore, MD; [3]CBH Health, Rockville, MD

ABSTRACT

All medications approved in the USA to treat schizophrenia, to date, have to a varying extent, dopamine D2 receptor occupancy (D2RO) as a feature of their pharmacology. Nonetheless, different antipsychotics exhibit different threshold levels of striatal D2RO. Most antipsychotics are D2 receptor antagonists, both pre- and post- synaptically, and have demonstrated antipsychotic efficacy in association with about a 65 - 80% striatal D2RO, while only slightly higher striatal D2RO (~80%) has been associated with the development of extrapyramidal side effects and hyperprolactinemia. Dopamine receptor partial agonists differ in this regard. For example, aripiprazole, which is both a pre- and post- synaptic partial agonist, demonstrates higher (>80%) D2RO in association with efficacy. At therapeutic doses in patients with schizophrenia, clozapine is an exception, with efficacy associated with relatively lower D2RO occupancy (<50%).

ITI-007 is a first-in-class dopamine receptor phosphoprotein modulator (DPPM), acting as a presynaptic partial agonist and postsynaptic antagonist[1]. This allows for reduced release of dopamine presynaptically with ITI-007 compared to pre-synaptic antagonists along with blockade of dopamine postsynaptically for more efficient reduction of dopaminergic signaling than most other antipsychotic drugs. ITI-007 also benefits from potent serotonin 5-HT2A receptor antagonism, serotonin transporter (SERT) inhibition, and increased phosphorylation of glutamatergic N-methyl-D-aspartate (NMDA) GluN2B receptors likely downstream of dopamine D1 receptor activation in mesolimbic brain regions[1]. Together, this unique pharmacology predicts more efficient dopamine modulation with antipsychotic efficacy at relatively low levels of D2RO.

Positron emission tomography (PET) data in healthy volunteers (Clinical trial ITI-007-003) was previously presented[2]. The results from this PET study indicated ITI-007 (10-40 mg) was safe and well tolerated and rapidly entered the brain with long-lasting and dose-related occupancy. ITI-007 (10 mg) demonstrated high occupancy (~80 %) of cortical 5-HT2A receptors and low occupancy of striatal D2 receptors (~12%). D2RO increased with dose and significantly correlated with plasma concentration. ITI-007 (40 mg) resulted in mean occupancy of 29% and peak occupancy up to 39% of striatal D2R and peak occupancy up to 33% of striatal serotonin transporters. ITI-007 (60 mg) was projected to have ~50% striatal D2RO.

The primary objective of the present study (ITI-007-008) was to determine the striatal D2RO of ITI-007 in patients with schizophrenia at a dose of 60 mg that has previously demonstrated antipsychotic efficacy[1].

Methods Patients with stable schizophrenia (N=14) volunteered and were washed off their antipsychotic medications for participation in this inpatient open-label study. After a drug-free period of at least two weeks, patients received a baseline scan followed by administration of 60 mg ITI-007 once daily for two weeks and up to three subsequent post-treatment scan(s). Carbon-11-Raclopride was used as the radiopharmaceutical for imaging striatal D2 receptors. Brain regions of interest were outlined using magnetic resonance tomography (MRT) with cerebellum as the reference region. Binding potentials were estimated using a simplified reference tissue model. D2RO was expressed as percent change in the binding potentials before and after ITI-007 administration.

Results Mean striatal D2RO levels observed with the dose of 60 mg ITI-007 was ~40%, with peak occupancy up to 51%. ITI-007 was safe and well tolerated in this study.

Discussion This PET study in patients with stable schizophrenia further adds to the information gleaned regarding brain receptor occupancy levels from a prior PET study in healthy volunteers. ITI-007 demonstrates relatively low striatal D2RO at the antipsychotic efficacious dose of 60 mg. In this regard, ITI-007 is more clozapine-like with efficacy at relatively low D2RO. Yet, ITI-007 demonstrates a more favorable safety profile. Consistent with low striatal D2RO, ITI-007 has a lesser liability for D2 mediated side effects, such as extrapyramidal side effects, including akathisia, and hyperprolactinemia than many antipsychotic drugs. Together, ITI-007 represents an exciting new potential treatment for schizophrenia.

BACKGROUND

ITI-007 has a first-in-class pharmacological profile via serotonergic, dopaminergic and glutamatergic pathways[1]

- 5-HT2A receptor antagonist
- Dopamine phosphoprotein modulator (DPPM)
- Glutamatergic phosphoprotein modulator
- Serotonin reuptake inhibitor

In Vitro Receptor Binding Affinities		
Target	ITI-007 Ki (nM)	IC200131 Ki (nM)
5-HT2A	0.5	61
D2	32	574
D1	52	>1000
SERT	62	~70

Note: IC200131 is the major circulating active metabolite of ITI-007

[1]Snyder et al., 2015 Psychopharmacology 232:605-621
[2]Davis et al., 2015 Psychopharmacology 232:2863-2872
[3]Lieberman et al., 2015 Biological Psychiatry online, ahead of print

RESULTS

60 mg ITI-007

~40% Mean Peak Striatal D2 Receptor Occupancy

At an effective antipsychotic dose, ITI-007 demonstrated relatively low striatal D2RO

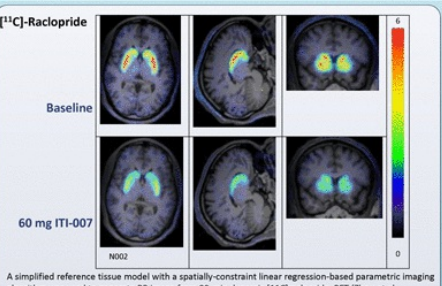
		Baseline BP_{ND}		%D_2RO			
Subject[a]	Start Time of Scan	Caudate	Putamen	Caudate	Putamen	Mean for Striatum	Group Mean ± SD
N001[b]	1 h	3.04	3.149	17.9	18	18	39% ± 12%
N002		4.033	5.234	43.1	39.2	41	
N003		4.216	5.519	35.5	34	35	
N004		3.399	4.556	49.8	47.5	49	
N005		3.641	4.619	52.3	50	51	
N006		3.684	4.074	41	34.1	38	
N011	3 h	3.872	4.476	36.8	36.1	36	34% ± 8%
N012		2.939	3.419	24.1	21.5	23	
N013		3.965	4.6	35.7	36	36	
N014		3.079	3.653	41.8	40.3	41	
N011	7.5 h	3.872	4.476	16	16.1	16	19% ± 10%
N012		2.939	3.419	12.8	10.8	12	
N013		3.965	4.6	13.8	16	15	
N014		3.079	3.653	36	31.2	34	
N011	24-27 h	3.872	4.476	4.7	5.9	5	7% ± 7%
N012		2.939	3.419	0.5	-3.6	negligible	
N013		3.965	4.6	9.6	12.5	11	
N014		3.079	3.653	12.9	14.7	14	

[a] An additional 2 subjects were evaluated at 20 mg ITI-007 and an additional 2 subjects were evaluated at 120 mg ITI-007, but baseline binding potentials were low with no measureable D2RO post-dose in one subject at each dose (N008 and N010); therefore these data (N=1/dose) are considered exploratory: In subject N009, a mean of 27% striatal D2RO was measured 1 h after a dose of 20 mg ITI-007; in subject N007, a mean of 47% striatal D2RO was measured 1 h after a dose of 120 mg ITI-007.

[b] N001 showed low plasma levels at the start of the scan, indicating the dose may have been missed; a reanalysis without this low outlier confirmed the initial analysis result with mean striatal D_2RO approximately 40% with 60 mg ITI-007, but, as would be expected, with less variability (43% ± 7%; range 35% to 51%; 41% median).

SAFETY & TOLERABILITY

- ITI-007 was safe and well tolerated in this study.
- There were no clinically significant changes in vital signs, ECGs, or clinical chemistry laboratory values.
- The most frequent adverse events (occurring in more than 2 patients) that were reported to be at least possibly related to ITI-007 were mild headache and mild sedation.
- There were no adverse event reports of akathisia or other extrapyramidal side effects.
- Mean values of motor function as measured by BARS and SAS indicated no motor disturbances with ITI-007 treatment.



A simplified reference tissue model with a spatially-constraint linear regression-based parametric imaging algorithm was used to generate BP image from 90-min dynamic [11C]raclopride PET (Zhou et al., NeuroImage, 2003, pp. 975-989).

CONCLUSIONS

- At an effective antipsychotic dose of 60 mg, ITI-007 demonstrated relatively low (~40%) mean peak striatal D2RO in patients with schizophrenia at plasma steady state.
- Time course data revealed peak brain occupancy as early as 1 h post-dose that was sustained through the 3 h post-dose measure and gradually tapered off over the course of 24 h.
- Consistent with low striatal D2RO, ITI-007 has a relatively low liability for D2 mediated side effects, such as extrapyramidal side effects, including akathisia, and hyperprolactinemia; in efficacy studies, 60 mg ITI-007 was not different from placebo on such side effects.
- ITI-007 represents a first-in-class new potential treatment for schizophrenia and other psychiatric and neurological disorders.
- ITI-007 is currently in Phase 3 clinical development for the treatment schizophrenia and bipolar depression.
- A low dose strategy for the treatment of behavioral disturbances associated with dementia is being pursued, for which even lower (5 – 10%) striatal D2RO may be beneficial without the accompanying adverse effects associated with many antipsychotic drugs.

DISCLOSURES

KEV, RED, CO'G, JS, MW and SM are full time employees of Intra-Cellular Therapies, Inc. (ITI).

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