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): April 7, 2014

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.)**

3960 Broadway
New York, New York 10032
(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 April 7, 2014, Intra-Cellular Therapies, Inc. announced multiple presentations on ITI-007 at the 4th Biennial Schizophrenia International Research Society (SIRS) Conference.

The Company's press release announcing these presentations is filed as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

ITEM 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Description
99.1	Press release dated April 7, 2014

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 and Secretary

Date: April 7, 2014

Exhibit 99.1

Intra-Cellular Therapies Announces Multiple Presentations on ITI-007 at the 4th Biennial Schizophrenia International Research Society (SIRS) Conference

NEW YORK, April 7, 2014 /GLOBE NEWSWIRE/ — Intra-Cellular Therapies, Inc. (NASDAQ: ITCI), a biopharmaceutical company focused on the development of therapeutics for central nervous system (CNS) disorders has presented several posters and a talk at the 4th Biennial Schizophrenia International Research Society (SIRS) conference being held in Florence, Italy.

"We are delighted to present our progress in the development program of ITI-007 at SIRS. Together, we believe these presentations convey the favorable profile and differentiating properties of ITI-007 demonstrated to date. At low doses ITI-007 has been shown to act as a potent 5-HT2A receptor antagonist, and, as the dose is increased, ITI-007 gradually engages other key brain receptors with regional selectivity. In addition, IC200131's long half-life and affinity for 5-HT2A receptors and serotonin transporters (SERT) offer a boost to the underlying effects of ITI-007 which we believe are sustained by IC200131 and its back conversion to ITI-007. Clinically, we believe ITI-007's pharmacology offers a potential therapeutic alternative to presently available treatments with a favorable adverse event profile and with improvements in symptoms rarely addressed by other marketed antipsychotics" said Dr. Sharon Mates, Chief Executive Officer and Chairman.

Oral Presentation of the ITI-007-005 Phase 2 Trial Results

An oral presentation highlighted data from the ITI-007-005 trial, topline results of which were first made public in December, 2013.

Primary endpoint: ITI-007 at a dose of 60 mg demonstrated a statistically significant improvement in psychosis on the trial's pre-specified primary endpoint, which was a change from baseline on the PANSS total score, compared to placebo on Study day 28. Specifically, patients on ITI-007 at 60 mg experienced a mean 13.2 point reduction in the PANSS total score compared to a mean reduction of 7.4 points in patients on placebo ($p = 0.017$; MMRM-ITT) for an antipsychotic effect size of 0.4. Secondary assessments included weekly PANSS total score, as well as its subscales.

Secondary endpoints: ITI-007 at a dose of 60 mg significantly reduced the PANSS positive symptom subscale score over four weeks of treatment ($p \leq 0.05$ versus placebo at day 15, 22 and 28). Moreover, ITI-007 improved the PANSS negative symptom subscale score with an effect size of 0.34 in a subgroup of patients with prominent negative symptoms at baseline. Unlike risperidone, ITI-007 at 60 mg did not worsen certain negative symptoms such as blunted affect. We believe that these data suggest that ITI-007 differentiates from risperidone in two ways, by improving some negative symptoms that risperidone does not improve, and by not worsening other negative symptoms that risperidone worsens.

Social function: ITI-007, at a dose of 60 mg, significantly improved certain individual symptom items on the PANSS across the positive symptom, negative symptom and general psychopathology subscales consistent with improved social function. Specifically, patients on ITI-007 at 60 mg experienced significant improvements in the Pro-social PANSS Factor (PPF), separating from placebo as early as the first week of treatment ($p \leq 0.05$ versus placebo at day 8) and continuing to improve over time with a robust effect size of 0.6 at the completion of 4 weeks of treatment ($p < 0.001$).

Co-morbid schizophrenia and depression: Patients were evaluated for depression using the Calgary Depression Scale for Schizophrenia (CDSS) and were included in the subgroup analysis if they exhibited a score of greater than 6 at baseline. In these patients with schizophrenia and co-morbid depression, at a dose of 60 mg, ITI-007 significantly reduced depression as measured by the CDSS ($p=0.044$) and significantly improved psychosis as measured by the PANSS total score ($p=0.018$).

Safety and tolerability: ITI-007 was well tolerated and the most frequent adverse event was sedation. At 60 mg and 120 mg ITI-007 showed a favorable side effect profile compared to risperidone in the present study and compared generally to other marketed antipsychotics on measures related to movement disorders, metabolic, prolactin or cardiovascular signals. For example, ITI-007 demonstrated a favorable metabolic profile on blood glucose levels, insulin, cholesterol and triglycerides. There were no serious adverse events related to ITI-007.

The ITI-007-005 Phase 2 results were also presented in poster format today (Monday April 7, 2014) Poster # M245 titled "Positive results with ITI-007 for the treatment of schizophrenia: a randomized, double-blind, placebo- and active-controlled Phase 2 study".

The Company believes the combination of effect in treating positive symptoms combined with a favorable safety and tolerability profile, including effects on negative symptoms and symptoms consistent with enhanced patient social function (which typically are left untreated by existing antipsychotic drugs), suggests ITI-007 may have broad utility in treating schizophrenia in its acute and chronic/residual phases as a single, stand-alone drug therapy.

Poster About IC200131 ("131")

On Sunday April 6, 2014, Intra-Cellular Therapies presented a poster describing the metabolism of ITI-007. Poster #S78, "The novel pharmacology of ITI-007 is enhanced and extended by its metabolic back conversion from IC200131" detailed the metabolic pathway and pharmacological profile of 131. Acting primarily as a 5-HT2A receptor antagonist and SERT inhibitor, 131 is believed to contribute to the unique pharmacological profile of ITI-007 and its clinical differentiation. The presentation emphasized ITI-007's pharmacological profile, driven by the combined actions of ITI-007 and its major circulating metabolite 131 (itself an active-moiety). This pharmacology is complemented by 131's back conversion to ITI-007. Given its long half-life, 131 can act as a pool from which ITI-007 can be continuously extracted resulting in a sustained pharmacological effect. 131 is in pre-clinical development as a candidate for mood disorders and other neurologic and psychiatric indications.

Other Presentations Describe ITI-007's Pharmacological Characterization and its Mechanism of Action

On Sunday, April 6, 2014, Intra-Cellular Therapies presented a poster describing the pharmacology of ITI-007. Poster #S176, "Targeted molecular therapeutic for schizophrenia: characterization of ITI-007 in in vitro and in vivo models" integrated the differentiated target affinity of ITI-007 at relevant receptors with the specifics of ITI-007 D2 pre-synaptic partial agonist effects and its mesolimbic/mesocortical selectivity. The presentation integrates the drug's pharmacokinetic profile, evidence of CNS penetration and D2 target engagement, and findings in preclinical behavioral models.

On Tuesday April 8, 2014, Intra-Cellular Therapies is scheduled to present a poster describing ITI-007's mechanism of action. Poster #T221 is titled "Mechanism of action of ITI-007: A novel therapy for the treatment of schizophrenia and related psychoses". Data showed ITI-007 combines potent 5-HT2A receptor antagonism (Ki = 0.54 nM) with lesser affinity for dopamine D2 receptors (Ki = 31.9 nM), D1 receptors (Ki = 52 nM), and the serotonin transporter (SERT, Ki = 62 nM). Experiments demonstrated the administration of ITI-007 increases dopamine release and enhances certain protein phosphorylation pathways in the prefrontal cortex and not in the striatum. We believe this combination suggests ITI-007 may offer a low motor side effect liability while offering a desired enhancement of dopamine mesocortical tone (activity/levels). In preclinical behavioral models, ITI-007 demonstrated a biochemical and pharmacological profile consistent with potent antipsychotic activity and antidepressant activity.

About the ITI-007-005 Phase 2 Clinical Trial Design

The Phase II clinical trial, ITI-007-005, was a randomized, double-blind, placebo- and active-controlled clinical trial in patients with an acutely exacerbated episode of schizophrenia. In this trial, 335 patients were randomized to receive one of four treatments: 60 mg ITI-007, 120 mg ITI-007, 4 mg risperidone (active control) or placebo in a 1:1:1:1 ratio. Patients received study treatment orally once daily in the morning for 28 days. Of those randomized, 311 patients were included in the intent-to-treat primary analysis.

The primary endpoint for this clinical trial was change from baseline to Day 28 on the PANSS total score. The PANSS is a well-validated, 30-item rating scale that measures the ability of a drug to reduce schizophrenia symptom severity (Kay et al., 1987, Schizophrenia Bulletin 13:261-276). The PANSS measures positive symptoms such as delusions, suspiciousness, and hallucinations; negative symptoms, such as blunted affect, social and emotional withdrawal, and stereotyped thinking; and general psychopathology, such as anxiety, tension, depression, and active social avoidance. Safety and tolerability were also assessed.

About Intra-Cellular Therapies

Intra-Cellular Therapies (the "Company") is developing novel drugs for the treatment of neuropsychiatric and neurodegenerative disease and other disorders of the central nervous system ("CNS"). The Company is developing its lead drug candidate, ITI-007, for the treatment of schizophrenia, behavioral disturbances in dementia, bipolar disorder and other neuropsychiatric and neurological disorders. In December 2013, the Company announced positive topline results from the Company's randomized, placebo- and active-controlled Phase II clinical trial of ITI-007 in patients with acutely exacerbated schizophrenia. This study showed a statistically significant improvement in symptoms associated with schizophrenia at the 60 mg dose on the trial's pre-specified primary endpoint and a favorable safety profile. The Company is also utilizing its phosphodiesterase (PDE) platform and other proprietary chemistry platforms to develop drugs for the treatment of cognitive deficits in schizophrenia and other CNS disorders. The Company has partnered its lead PDE1 compound, ITI-214, and backups from this platform with the Takeda Pharmaceutical Company. ITI-214 has finished the first Phase 1 clinical trial and is now in subsequent Phase 1 trials. The Company is also developing inhibitors against additional targets for CNS indications such as Alzheimer's disease, Parkinson's disease and depression and non-CNS indications such as cardiovascular disease.

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 expectations, plans and beliefs regarding the clinical development of ITI-007 and its potential to treat patients with schizophrenia and other CNS disorders more favorably than existing marketed antipsychotics, the potential for IC200131 to enhance ITI-007, and our plans to advance IC200131 into clinical development.

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 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 and commercialization 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, 2013 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. 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:

Juan Sanchez, M.D.
Vice President
Corporate Communications and Investor Relations of Intra-Cellular Therapies, Inc.
212-923-3344

Burns McClellan, Inc.
Lisa Burns/Angeli Kolhatkar (Investors)
Justin Jackson (Media)
jjackson@burnsmc.com
Phone 212-213-0006