UNITED STATES SECURITIES AND EXCHANGE COMMISSION

Washington, DC 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) : **October 15, 2010**

AVI BioPharma, Inc.

(Exact name of registrant as specified in its charter)

Oregon	**001-14895**	**93-0797222**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

3450 Monte Villa Parkway, Suite 101
Bothell, WA 98021
(Address of principal executive offices, including zip code)

(425) 354-5038
(Registrant's telephone number, including area code)

(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 (see General Instruction A.2. below):

☐ 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 October 15, 2010, AVI BioPharma, Inc. issued a press release announcing the results of a recently completed clinical trial of its product candidate, AVI-4658, for the treatment of Duchenne Muscular Dystrophy (DMD). The text of the press release is included as Exhibit 99.1 to this 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 October 15, 2010 announcing results of clinical trial of AVI-4658.

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.

AVI BioPharma, Inc.

By: /s/ J. David Boyle II
J. David Boyle II
Interim President and Chief Executive Officer, and Senior Vice President and Chief Financial Officer

Date: October 16, 2010

EXHIBIT INDEX

Exhibit Number	Description
99.1	Press Release dated October 15, 2010 announcing results of clinical trial of AVI-4658.

Exhibit 99.1

AVI BioPharma's Investigational Drug Candidate AVI-4658 Demonstrates Broadly Favorable Profile of Safety and Tolerability, New Dystrophin Expression, Stable Clinical Performance and Inflammatory Modulation in the Treatment of Duchenne Muscular Dystrophy

AVI-4658 Data From Phase 1b/2 Study Presented at 15th International Congress of the World Muscle Society Supports Potential as Disease Modifying Therapy

- AVI-4658 Well Tolerated in All Patients
- New, Dose Dependent, Dystrophin and Dystrophin Positive Fibers Up to 55%
- Reduction in Key Inflammatory Markers; No Immune Response to Newly Produced Dystrophin
- Restoration of the Dystrophin-Associated Glycoprotein Complex in Muscle Cells
- General Stability in Exploratory Markers of Patient Clinical Performance
- Results Support U.S. Phase 2 Trial Evaluating Higher Doses Planned to Start This Year

AVI BioPharma, Inc. (NASDAQ: AVII), a developer of RNA-based therapeutics, today announced that data from the recently completed trial of AVI-4658, the Company's investigational drug candidate for the treatment of Duchenne muscular dystrophy (DMD), demonstrated a broadly favorable therapeutic profile with promising safety, biological and exploratory clinical performance assessments that supports rapid advancement of the program into the next phase of clinical development. The data from AVI's Study 28, the completed Phase 1b/2 clinical trial conducted in the U.K., were presented at the 15th International Congress of the World Muscle Society.

Based on the Study 28 results presented today and other supportive clinical and preclinical data, AVI plans to initiate a randomized, double blind, placebo-controlled Phase 2 clinical trial this year in the U.S evaluating higher doses of AVI-4658 in patients with DMD. AVI-4658 is AVI's lead drug candidate in development as a systemically administered treatment for a substantial subgroup of patients with DMD, a genetic muscle wasting disease caused by the absence of functional dystrophin in the muscles.

"The data from this trial support the potential of AVI-4658 as a promising disease modifying therapy for DMD and its planned advancement into a Phase 2 study later this year," said Stephen B. Shrewsbury, M.D., Senior Vice President and Chief Medical Officer of AVI. "I'm encouraged by the broadly favorable profile seen to date, particularly the safety and tolerability data, new dystrophin expression, stable clinical performance, improvements in inflammatory markers and lack of an immune response. Adding to my level of confidence in our approach is the restoration of the dystrophin related protein complexes necessary for proper muscle cell function that were previously absent from these patients. I'm eager to continue our clinical program in an effort to develop a disease modifying therapy where currently no such therapies are available."

A review of the data from all patients who completed the trial establishes support for the following conclusions:

- AVI-4658 was well tolerated in all patients. Adverse events were mostly mild or moderate in intensity, not dose-related, and none were considered probably related to study drug. There were also no drug-related serious adverse events or severe adverse events.
- Substantial and novel dystrophin expression and dystrophin-positive fiber generation reaching up to 55%, although variable among patients, tended to be greatest in the highest two cohorts.
- Dystrophin expression was correctly localized and was accompanied by restoration of the Dystrophin-associated Glycoprotein Complex (DGC), a protein complex necessary for the proper function of muscle cells.
- Reductions in key inflammatory markers, including CD3, CD4 and CD8 counts, potentially suggest an alteration in the underlying degenerative disease process.
- There was no immune response to newly made dystrophin.
- There was general stability in exploratory markers of patient clinical performance, including cardiac, pulmonary and muscle functional assessments.

Phase 1b/2 Trial Results

Evaluation of safety measurements over the 26-week course of the study demonstrate that AVI-4658 was well tolerated in all patients treated, with 219 doses of AVI-4658 successfully administered in this study. Adverse events were consistent with those typically seen in chronic pediatric trials and with the underlying disease of the patients enrolled. Adverse events were generally mild or moderate. None of the adverse events, serious adverse events or the single severe adverse event reported was considered by the investigators to be probably related to treatment with AVI-4658. There was also no dose-dependent increase in the frequency or severity of any events. Laboratory assessments for this population were generally stable throughout the course of the study, notably across measurements of coagulation, renal function, clinical chemistry and hematology.

In the study, treatment with AVI-4658 also appeared to have a favorable impact on immunological and inflammatory markers. Restoration of dystrophin was followed by an absence of anti-dystrophin antibodies, suggesting no immunologic reaction to the newly produced dystrophin. There was also an overall reduction in T cell infiltration and inflammatory markers, including CD3, CD4 and CD8 cell counts, in the patient muscle biopsies from the top two cohorts.

Treatment with AVI-4658 in all eight patients in the 10 mg/kg and 20 mg/kg cohorts showed consistent skipping of exon 51, which is believed necessary to restore the messenger RNA (mRNA) reading frame leading to expression of a truncated but functional dystrophin protein in a substantial subgroup of patients with specific mutations. All patients in these cohorts also demonstrated generation of new dystrophin-positive muscle fibers as measured by immunofluorescent analysis of their muscle biopsies.

Of note, three patients, one patient in each of the 2.0, 10 and 20 mg/kg cohorts, demonstrated substantial levels of new dystrophin-positive muscle fibers, which increased from 1% to 21%, 1% to 15%, and 3% to 55% of normal, respectively, when comparing pre-treatment to post-treatment samples. These three patients also demonstrated a noted increase in dystrophin per fiber as determined by immunofluorescent analysis as well as multiple fold increases in dystrophin protein expression measured by Western blot over baseline.

Dystrophin expression in patients, although variable, tended to be greatest in the 10 mg/kg and 20 mg/kg cohorts. These cohorts, both quantitatively and qualitatively, had more uniform and widespread dystrophin-positive fiber distribution than patients who received lower doses. Additionally, the greatest response measured by Western blot was reported in the 20 mg/kg cohort.

Restoration of dystrophin was correctly localized within muscle cells in patients and was observed to be accompanied by restoration of the DGC. Loss of dystrophin results in the loss of the DGC, which is necessary for the proper function of muscle cells.

Exploratory clinical performance measurements, including the 6 Minute Walk Test, 10 Meter Walk Test, NorthStar Ambulatory Assessment and evaluations of cardiac and pulmonary function, generally demonstrated stability in patient performance. Given the size and duration limitations of this open label trial, definitive assessment of the treatment impact on measures of clinical performance will need to be further evaluated in additional clinical studies.

Phase 1b/2 Trial Design

Study 28 was a Phase 1b/2 open label, dose-ranging, clinical trial assessing the safety, tolerability, pharmacokinetics and exploratory efficacy of AVI-4658 in ambulatory patients with DMD between the ages of five and 15 who have an error in the gene coding for dystrophin that can be treated by skipping exon 51. Patients were dosed once per week for 12 weeks by slow intravenous infusion. Nineteen patients were enrolled in total and assigned to one of six dose cohorts: 0.5, 1.0, 2.0, 4.0, 10.0 or 20.0 mg/kg. After completion of dosing, patients were followed for an additional 14 weeks. Muscle biopsies were taken before treatment and at week 14, two weeks after administration of the final dose. The primary objective of the trial was to assess the safety of AVI-4658 at these doses over the 26-week duration of the trial. Eighteen of 19 patients received at least 10 of the 12 doses planned in this trial. Secondary trial objectives included assessment of plasma pharmacokinetics, urinary elimination and exploratory endpoints evaluating biological activity and clinical performance.

The clinical trial of AVI-4658 was conducted in London, UK at the UCL Institute of Child Health / Great Ormond Street Hospital NHS Trust facilities by members of the MDEX Consortium led by Professor Francesco Muntoni and by Professor Kate Bushby at the Royal Victoria Infirmary, Newcastle-Upon-Tyne, UK, which is the coordinating center for the European Network of Excellence TREAT-NMD. The clinical costs for the trial were provided in part by the UK Medical Research Council.

Phase 2 Trial Plans

AVI is currently preparing to initiate a Phase 2 clinical trial intended to evaluate higher weekly doses of AVI-4658 of 50 mg/kg and 100 mg/kg, subject to review of the trial protocol by the U.S. Food and Drug Administration (FDA) under the Company's open Investigational New Drug application (IND) and by the Institutional Review Board of Nationwide Children's Hospital, Columbus, Ohio, the planned clinical site. AVI received permission from the FDA to conduct studies in the U.S. under an IND that was opened in July 2010. The highest weekly dose evaluated in Study 28 was 20 mg/kg. The Phase 2 study is scheduled to start in late 2010.

The design for the Phase 2 clinical trial was discussed recently at an expert panel assembled by AVI to review Study 28 data and future clinical plans in DMD. As a result of the input from the expert panel, the planned study will be a randomized, double blind, placebo-controlled Phase 2 trial assessing the safety, tolerability and pharmacokinetics of AVI-4658 as well as biological activity of the compound as determined by levels of dystrophin restoration in patients. The study will also assess exploratory parameters of clinical performance. The study will be conducted in ambulatory patients with DMD between the ages of five and 15 who have an error in the gene coding for dystrophin that can be treated by skipping exon 51. Patients will be dosed once per week for 12 weeks. After completion of dosing, patients will be followed for an additional 14 weeks.

About AVI-4658

AVI-4658 is AVI's lead drug candidate being developed as a systemically administered treatment for a substantial subgroup of patients with DMD. It is an RNA-based therapeutic employing AVI's novel phosphorodiamidate morpholino oligomer (PMO) based chemistry which can work by exon skipping. AVI has exclusive worldwide rights to intellectual property under a license from the University of Western Australia. The lead inventor on the patent is Prof. Steve Wilton, Winthrop Professor, University of Western Australia, President, Australian Gene Therapy Society, and Head, Molecular Genetic Therapeutics Group, Centre for Neuromuscular and Neurological Disorders, Australian Neuromuscular Research Institute.

About Duchenne Muscular Dystrophy

Duchenne Muscular Dystrophy (DMD) is one of the most common fatal genetic disorders to affect children around the world. Approximately one in every 3,500 boys worldwide is affected with DMD. Girls are rarely affected by the disorder. DMD is a devastating and incurable muscle-wasting disease associated with specific inborn errors in the gene that codes for dystrophin, a protein that plays a key structural role in muscle fiber function. Symptoms usually appear in children by age three. Progressive muscle weakness of the legs and pelvis eventually spreads to the arms, neck, and other areas. By age 10, braces may be required for walking, and most patients require full-time use of a wheelchair by age 12. Eventually, this progresses to complete paralysis and increasing difficulty in breathing due to respiratory muscle dysfunction requiring ventilatory support, and cardiac muscle dysfunction leading to heart failure. The condition is terminal and death usually occurs before the age of 30. The outpatient cost of care for a non-ambulatory DMD patient is very high. There is currently no cure for DMD, but for the first time ever there are promising therapies in, or moving into, development.

About the MDEX Consortium

The MDEX consortium led by Professor Francesco Muntoni, is a multidisciplinary enterprise to promote translational research into muscular dystrophies, and is formed by the clinical groups of Professor Francesco Muntoni (UCL Institute of Child Health) and Professor Kate Bushby and

Professor Volker Straub (Newcastle University) and scientists from Imperial College London (Professor Dominic Wells), UCL Institute of Child Health (Dr. Jennifer Morgan), Royal Holloway University of London (Professor George Dickson), Oxford University (Dr. Matthew Wood) and University of Western Australia (Professor Steve Wilton). In addition, the charities Muscular Dystrophy Campaign (MDC), Action Duchenne and Duchenne Family Support Group also participate in the Consortium. For more information, visit www.mdex.org.uk.

About AVI BioPharma

AVI BioPharma is focused on the discovery and development of novel RNA-based therapeutics for rare and infectious diseases, as well as other select disease targets. Applying pioneering technologies developed and optimized by AVI, the Company is able to target a broad range of diseases and disorders through distinct RNA-based mechanisms of action. Unlike other RNA-based approaches, AVI's technologies can be used to directly target both messenger RNA (mRNA) and precursor messenger RNA (pre-mRNA) to either down-regulate (inhibit) or up-regulate (promote) the expression of targeted genes or proteins. By leveraging a highly differentiated RNA antisense-based technology platform, AVI has built a pipeline of potentially transformative therapeutic agents, including one in the clinical development stage for the treatment of Duchenne muscular dystrophy.

Forward-Looking Statements and Information

This press release contains statements that are forward-looking, including statements about the development of AVI 4658, other antisense-based technology and the efficacy, potency and utility of our product candidates in the treatment of rare and infectious diseases, and its potential to treat a broad number of human diseases. These forward-looking statements involve risks and uncertainties, many of which are beyond AVI's control. Known risk factors include, among others: clinical trials may not demonstrate safety and efficacy of any of AVI's drug candidates, including AVI 4658, and/or AVI's antisense-based technology platform; any of AVI's drug candidates, including AVI 4658, may fail in development, may not receive required regulatory approvals, or be delayed to a point where they do not become commercially viable. Any of the foregoing risks could materially and adversely affect AVI's business, results of operations and the trading price of its common stock. For a detailed description of risks and uncertainties AVI faces, you are encouraged to review the official corporate documents filed with the Securities and Exchange Commission. AVI does not undertake any obligation to publicly update its forward-looking statements based on events or circumstances after the date hereof.

SOURCE: AVI BioPharma, Inc.