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 22, 2009**

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)	(I.R.S. Employer Identification No.)

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

(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:

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

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

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

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

Item 7.01. Regulation FD Disclosure.

On December 22, 2009, AVI BioPharma, Inc. (the "Company") issued a press release announcing initial efficacy data from the ongoing Phase 1b/2 clinical trial of AVI-4658 for the systemic treatment of patients with Duchenne muscular dystrophy (DMD), a genetic muscle wasting disease caused by failure to produce dystrophin. A copy of this press release is attached hereto as Exhibit 99.1.

The information in this Item 7.01 and the press release attached as Exhibit 99.1 to this Form 8-K, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, nor shall this Item 7.01, such Exhibit 99.1, or any of the information contained therein be deemed incorporated by reference in any filing under the Securities Exchange Act of 1934 or the Securities Act of 1933, except as shall be expressly set forth by specific reference in such filing.

Item 9.01. Financial Statements and Exhibits

(d) Exhibits

Exhibit No.	Description
99.1	Press release, dated December 22, 2009, entitled "Systemic Treatment With AVI-4658 Demonstrates RNA Exon Skipping and Dystrophin Protein Expression in Duchenne Muscular Dystrophy Patients"

SIGNATURES

Pursuant to the requirements of Section 13 or 15(d) of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized, in the City of Bothell, State of Washington, on December 23, 2009.

AVI BioPharma, Inc.

By: /s/ Leslie Hudson, Ph.D.

Leslie Hudson, Ph.D.
President and Chief Executive Officer
(Principal Operating Officer)

EXHIBIT INDEX

Exhibit No.	Description
99.1	Press release, dated December 22, 2009, entitled "Systemic Treatment With AVI-4658 Demonstrates RNA Exon Skipping and Dystrophin Protein Expression in Duchenne Muscular Dystrophy Patients"

AVI Press and Investor Contact:
David A. Walsey
Senior Director, Investor Relations & Corporate Communications
425.354.5140
Investorrelations@avibio.com

Systemic Treatment with AVI-4658 Demonstrates RNA Exon Skipping and Dystrophin Protein Expression in Duchenne Muscular Dystrophy Patients

Positive RNA and Protein Signals in First Cohorts Analyzed

Conference Call Scheduled Today at 8:30 AM Eastern Time

BOTHELL, WA — December 22, 2009 — AVI BioPharma, Inc. (NASDAQ: AVII), a developer of RNA-based drugs, today announced initial efficacy data from the ongoing Phase 1b/2 clinical trial of AVI-4658 for the systemic treatment of patients with Duchenne muscular dystrophy (DMD), a genetic muscle wasting disease caused by failure to produce dystrophin. Patients in the first four (of six) cohorts completing 12 weeks of treatment with different doses of AVI-4658 (0.5, 1.0, 2.0 or 4.0 mg/kg) have had their muscles biopsied. Analysis of the post treatment biopsies found that patients in the 2 and 4 mg/kg drug-treatment cohorts (3 of 3 in total) showed correctly spliced mRNA for dystrophin. One of these patients, in the 2mg/kg cohort, showed robust expression of dystrophin protein by western blot and immunofluorescent analysis. No RNA or protein expression signal was detected in patients from the 0.5 mg/kg or 1.0 mg/kg cohorts after completing treatment. Restoration of functional dystrophin expression is considered critical for successful treatment of DMD.

Treatment with AVI-4658 in the three patients in the 2.0 and 4.0 mg/kg cohorts led to accurate skipping of exon 51, which is believed to be necessary to restore the mRNA reading frame for functional dystrophin expression in patients with this class of mutations. Analysis of post-treatment biopsies by the reverse transcription-polymerase chain reaction showed a new lower molecular weight band of RNA resulting from the intended skipping, or exclusion, of exon 51. The intensity of the higher molecular weight band (which included exon 51) was correspondingly reduced. In one of the patients at the 2.0 mg/kg dose, the appearance of skipped mRNA was accompanied by a robust increase in expression of dystrophin protein in the post treatment samples using both western blot and immunofluorescent analysis. Western blot analysis detected a fivefold increase in dystrophin expression, from 0.9% to 5.3% of normal. Immunofluorescent analysis of the muscle biopsies from this patient showed an increase in the percentage of dystrophin positive muscle fibers from 1% pre-treatment to 21% in the post-treatment biopsy. Quantitative intensity analysis of the amount of dystrophin per fiber in patient samples before and after drug treatment showed a sevenfold increase in dystrophin. When compared to the level of dystrophin in normal muscle fibers, the dystrophin content per patient fiber went from 5% pre-treatment to 37% in the post-treatment biopsy.

“I am very encouraged by the evidence of accurate skipping of exon 51 in three treated patients,” stated Prof. Francesco Muntoni, Professor of Pediatric Neurology and Head of the Dubowitz Neuromuscular Centre at the UCL Institute of Child Health, London, England and the trial’s lead investigator. “These results suggest that we are on the right path towards developing a drug that

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could play a role in the treatment of DMD. The fact that one patient at the 2 mg/kg dose showed significant expression of dystrophin protein leads us to expect greater levels of dystrophin expression following treatment with the higher doses of 10.0 mg/kg and 20.0 mg/kg of AVI-4658, which are currently underway in the trial.”

Clinical Trial Design and Update
Study 28 is a Phase 1b/2 open label, dose-ranging clinical trial assessing the safety, tolerability, pharmacokinetics and exploratory efficacy of AVI-4658 in ambulatory DMD boys between the ages of 5 and 15 years of age who have an error in the gene coding for dystrophin that could be treated by skipping exon 51. Patients are dosed once per week for 12 weeks by intravenous infusion. Nineteen patients have been 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 are followed for a further 14 weeks. The primary objective of the trial is to assess the safety of AVI-4658 at these doses over the 26-week duration of the trial.

To date, 9 of 10 patients in the first four cohorts (0.5 through 4.0 mg/kg) have completed dosing. A single patient (in the 4 mg/kg cohort) withdrew from treatment due to DMD-related cardiomyopathy (now stabilized and believed not to be drug related). An additional patient was enrolled at 4 mg/kg but has not yet completed dosing. All 8 patients in the fifth and sixth cohorts, receiving 10 or 20 mg/kg respectively, have initiated dosing.

Data from patients dosed to date demonstrate that AVI-4658 continues to be generally very well tolerated. Adverse events reported to date are mostly mild, unrelated to drug treatment and transient. In the patients who completed dosing, two serious adverse events, both deemed unrelated to AVI-4658, were reported in different patients after they completed their 12-week treatment period and during the 14-week follow-up period.

Studies Towards US IND
AVI has completed a series of 12-week preclinical studies of AVI-4658 under Good Laboratory Practice (GLP) conditions required to open an Investigational New Drug (IND) application in the US. The studies tested doses up to 960 mg/kg in both *mdx* and wild type mice, and up to 320 mg/kg in non-human primates, both doses being the maximum feasible single doses in these animals. In all cases the PMO was well tolerated at doses equivalent to 80 mg/kg and 110 mg/kg in humans respectively (based on standard allometric scaling), suggesting the potential for a wide therapeutic index.

An additional GLP study of AVI-4225 PMO, to skip exon 23, in the mdx mouse has also been completed, with similar encouraging reports of good tolerability. The histopathology is currently being reviewed but initial reports suggest that the muscles of treated mice show improvement over the 12 weeks of study.

“AVI-4658 continues to demonstrate the good safety profile associated with PMO-based drug candidates. Data from the recently completed series of preclinical studies required to open an IND in the US suggest that this good tolerability is likely to continue at higher doses,” stated Stephen B. Shrewsbury, M.D., Senior Vice President and Chief Medical Officer, AVI BioPharma, Inc. “This is critically important given that any DMD drug based on exon skipping is expected to be administered regularly over the entire course of a patient’s life.”

The clinical trial of AVI-4658 is being 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 Muntoni and by Professor Kate Bushby at the Royal Victoria Infirmary, Newcastle-Upon-Tyne, UK, which is the coordinating center for the European Treat Neuromuscular Diseases (Treat-NMD) initiative. The clinical costs for the trial are provided, in part, by the UK Medical Research Council.

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 afflicted with DMD with 20,000 new cases reported each year. It 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 male 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 are confined to a wheelchair by age 12. Eventually, this progresses to complete paralysis and increasing difficulty in breathing requiring ventilatory support. The condition is terminal and death usually occurs before the age of 30. The outpatient cost of care for a non-ambulatory DMD boy is among the highest of any disease. There is currently no cure for DMD, but for the first time ever, there are promising therapies in or moving into development.

Conference Call
AVI management will hold a conference call to review the initial data from the ongoing Phase 1b/2 clinical trial on Tuesday, December 22, 2009, at 8:30 AM Eastern time (5:30 AM Pacific Time).

Individuals interested in listening to the live conference call may do so by dialing 877-879-6209 toll free within the United States and Canada, or 719-325-4794 for international callers. A replay of the call will be available by dialing 888-203-1112 toll free within the United States and Canada, or 719-457-0820 for international callers. The passcode for the replay is 1823048. In addition, a recording of the call will be available within approximately 24 hours at www.avibio.com.

About AVI BioPharma
AVI BioPharma is focused on the discovery and development of RNA—based drugs utilizing proprietary derivatives of its antisense chemistry (morpholino-modified phosphorodiamidate oligomers or PMOs) that can be applied to a wide range of diseases and genetic disorders through several distinct mechanisms of action. Unlike other RNA therapeutic approaches, AVI's antisense technology has been used to directly target both messenger RNA (mRNA) and its precursor (pre-mRNA), allowing for both up- and down-regulation of targeted genes and proteins. AVI's RNA—based drug programs are being evaluated for the treatment of Duchenne muscular dystrophy, including an ongoing systemic Phase 1b/2 clinical trial of exon skipping with AVI-4658. AVI's antiviral programs have demonstrated promising outcomes in Ebola Zaire and Marburg Musoke virus infections and may prove applicable to other viral targets such as Junín, influenza, HCV or Dengue viruses. For more information, visit www.avibio.com.

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.

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"Safe Harbor" Statement under the Private Securities Litigation Reform Act of 1995: The statements that are not historical facts contained in this release are forward-looking statements that involve risks and uncertainties, including, but not limited to, the results of research and development efforts, the results of preclinical and clinical testing, the effect of regulation by the FDA and other agencies, the impact of competitive products, product development, commercialization and technological difficulties, and other risks detailed in the company's Securities and Exchange Commission filings