

**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) **October 15, 2014**

Tekmira Pharmaceuticals Corporation

(Exact name of registrant as specified in its charter)

British Columbia, Canada
(State or other jurisdiction
of incorporation)

001-34949
(Commission File Number)

980597776
(IRS Employer Identification No.)

**100-8900 Glenlyon Parkway
Burnaby, British Columbia
Canada**
(Address of principal executive offices)

V5J 5J8
(Zip Code)

Registrant's telephone number, including area code: **(604) 419-3200**

(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))
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Item 8.01. Other Events.

On October 15, 2014 the Registrant issued a press release, a copy of which is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

Item 9.01. Financial Statements and Exhibits.

Exhibit 99.1. Press release dated October 15, 2014

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.

Tekmira Pharmaceuticals Corporation

(Registrant)

/s/ **BRUCE G. COUSINS**

October 15, 2014

(Date)

Bruce G. Cousins

Executive Vice President and Chief Financial Officer

Exhibit Index

99.1 Press release dated October 15, 2014

Tekmira Presents Results of Preclinical Studies With Hepatitis B Therapeutic

Demonstrates Robust Knockdown of HBV Surface Antigen

VANCOUVER, British Columbia, Oct. 15, 2014 (GLOBE NEWSWIRE) -- Tekmira Pharmaceuticals Corporation (Nasdaq:TKMR) (TSX:TKM), a leading developer of RNA interference (RNAi) therapeutics, announced today the presentation of preclinical results characterizing TKM-HBV, a therapeutic agent targeting human hepatitis B virus (HBV), at the 10th Annual Meeting of the Oligonucleotide Therapeutics Society. Tekmira's Chief Technical Officer, Dr. Ian MacLachlan delivered a podium presentation titled, "*Update on the Preclinical Development of an LNP-Based HBV Therapeutic.*" The conference is taking place in San Diego, California, from October 12 – 15, 2014.

Among the results reported is the potent and rapid reduction in hepatitis B surface antigen (HBsAg) demonstrated by TKM-HBV in several preclinical models including the chronically infected humanized (chimeric) mouse.

"These results reflect the rigorous approach we have taken to the design and characterization of our novel hepatitis B therapeutic," said Dr. Mark J Murray, President and CEO, Tekmira Pharmaceuticals. "TKM-HBV employs a unique combination of three RNAi triggers, in a third generation LNP formulation, which results in broad and effective knockdown of viral mRNAs and viral proteins including hepatitis B surface antigen, our primary therapeutic target. This data supports the utility of TKM-HBV as a new therapeutic option for treating patients with chronic HBV infection. Our plan is to file an IND, or equivalent document, by the end of this year, and initiate clinical trials in early 2015."

Key summary points from the presentation include:

- Preclinical studies employed several well validated models including true-infection models that support the full viral replication/re-infection cycle and the production of covalently closed circular DNA (cccDNA).
- In these models, TKM-HBV treatment resulted in rapid and potent reductions in both intrahepatic and serum HBsAg, as well as reductions in HBV DNA, cccDNA, HBeAg and HBcAg.
- A rapid 1 log reduction in serum HBsAg was achieved with a single 1 mg/kg dose of TKM-HBV in the chronically infected humanized mouse model, which mimics human hepatitis B infection. 1-2 log viral reductions from similar single-dose LNP treatments in two other true-infection animal models have also been demonstrated.
- Tekmira's data suggests inclusion of three RNAi triggers results in a more broadly effective knockdown of viral products than a single trigger alone.
- By targeting three distinct and highly conserved sites on the HBV genome:
 - TKM-HBV is designed to achieve "universal" activity in chronic HBV patients infected with genotypes A through H. Studies conducted on infected primary human hepatocytes showed that TKM-HBV had robust and consistent activity against different viral strains representing the major clinical genotypes A, B, C and D.
 - TKM-HBV is designed to achieve a higher threshold against development of antiviral resistance. Preclinical investigation targeting the closely related woodchuck hepatitis virus suggests that the use of a single stand-alone RNAi trigger can result in viral resistance.
- The mode of action of TKM-HBV complements standard of care nucleoside/nucleotide (NUC) therapy, and lack of drug antagonism has been demonstrated with entecavir, lamivudine and tenofovir on infected primary human hepatocytes.
- The drug mechanism of action is well-understood and RNAi-mediated site-specific cleavage of viral mRNA by TKM-HBV has been confirmed using RLM-RACE sequence analysis.

A copy of Tekmira's presentation from the 10th Annual Meeting of the Oligonucleotide Therapeutics Society Meeting will be available on the Tekmira website on the "Events" section at: <http://investor.tekmirapharm.com/events.cfm>

About RNAi and Tekmira's LNP

RNAi therapeutics have the potential to treat a number of human diseases by "silencing" disease causing genes. The discoverers of RNAi, a gene silencing mechanism used by all cells, were awarded the 2006 Nobel Prize for Physiology or Medicine. RNAi trigger molecules often require delivery technology to be effective as therapeutics. Tekmira believes its LNP technology represents the most advanced and widely adopted delivery technology for the systemic delivery of RNAi triggers. Tekmira's LNP platform is being utilized in multiple clinical trials in various disease areas by Tekmira and its partners. Tekmira's LNP technology (formerly referred to as stable nucleic acid-lipid particles or SNALP) encapsulates RNAi triggers with high efficiency in uniform lipid nanoparticles that are effective in delivering these therapeutic compounds to disease sites. Tekmira's LNP formulations are manufactured by a proprietary method which is robust, scalable and highly reproducible, and LNP-based products have been

reviewed by multiple regulatory agencies for use in clinical trials. LNP formulations comprise several lipid components that can be adjusted to suit the specific application.

About Tekmira

Tekmira Pharmaceuticals Corporation is a biopharmaceutical company focused on advancing novel RNAi therapeutics and providing its leading lipid nanoparticle (LNP) delivery technology to pharmaceutical and biotechnology partners. Tekmira has been working in the field of nucleic acid delivery for over a decade, and has broad intellectual property covering its delivery technology. Further information about Tekmira can be found at www.tekmira.com. Tekmira is based in Vancouver, Canada and Seattle, USA.

About TKM-HBV

TKM-HBV is designed to eliminate hepatitis B surface antigen expression in chronically infected patients. Blocking HBV surface antigen – and reducing the pathology associated with its expression may enable patients to raise antibody against the virus. The ability of TKM-HBV to collectively reduce several viral elements in addition to surface antigen, allows effective targeting of the hepatitis B virus at multiple critical nodes in the infection cycle.

TKM-HBV is being developed as a multi-component RNAi therapeutic that simultaneously targets three sites on the HBV genome. Because HBV is a viral infection of the liver, TKM-HBV will employ a liver-centric LNP formulation that is more potent than any LNP currently in clinical development.

About HBV

The hepatitis B virus (HBV) is a DNA virus belonging to the Hepadnaviridae family of viruses. Approximately 350 million individuals in the world and one million in the United States are chronically infected with HBV, many of whom appear healthy but can spread the virus to others. Chronic hepatitis B may lead to cirrhosis, liver cancer or liver failure. Approximately 15% to 25% of people with chronic infection will die prematurely as a result of the infection, and the disease typically progresses gradually over the course of several decades. Current treatments are able to suppress HBV if taken indefinitely, but these do not typically lead to a functional cure, and viral rebound is observed upon treatment stoppage. (www.medicinenet.com/hepatitis_b)

Forward-Looking Statements and Information

This news release contains "forward-looking statements" or "forward-looking information" within the meaning of applicable securities laws (collectively, "forward-looking statements"). Forward-looking statements in this news release include statements about Tekmira's strategy, future operations, clinical trials, prospects and the plans of management; the results reported by Tekmira on TKM-HBV in preclinical models; plans to file an IND, or equivalent document, by the end of this year and initiate clinical trials in early 2015 for TKM-HBV; the effects and potency of Tekmira's product TKM-HBV on chronic Hepatitis B infection; and estimations of unmet demands for TKM-HBV.

With respect to the forward-looking statements contained in this news release, Tekmira has made numerous assumptions regarding, among other things: LNP's status as a leading RNAi delivery technology; the effectiveness of RNAi therapeutics in the treatment of Hepatitis B virus; and the results of true-infection animal models. While Tekmira considers these assumptions to be reasonable, these assumptions are inherently subject to significant business, economic, competitive, market and social uncertainties and contingencies.

Additionally, there are known and unknown risk factors which could cause Tekmira's actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements contained herein. Known risk factors include, among others: RNAi based therapeutics may not prove to be effective in the treatment of Hepatitis B virus as currently anticipated, compared to other therapeutics, or at all; Tekmira may not file an IND, or equivalent document, or initiate clinical trials for TKM-HBV as currently anticipated, or at all; the FDA or other regulatory agencies may refuse to approve Tekmira's products, or place restrictions on Tekmira's ability to commercialize its products; anticipated pre-clinical and clinical trials may be more costly or take longer to complete than anticipated, and may never be initiated or completed, or may not generate results that warrant future development of the tested drug candidate; future operating results are uncertain and likely to fluctuate; economic and capital market conditions; and the possibility that Tekmira may not have sufficiently budgeted for expenditures necessary to carry out planned activities.

A more complete discussion of the risks and uncertainties facing Tekmira appears in Tekmira's Annual Report on Form 10-K and Tekmira's continuous disclosure filings, which are available at www.sedar.com and www.sec.gov. All forward-looking statements herein are qualified in their entirety by this cautionary statement, and Tekmira disclaims any obligation to revise or update any such forward-looking statements or to publicly announce the result of any revisions to any of the forward-looking statements contained herein to reflect future results, events or developments, except as required by law.

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