
**UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549**

FORM 6-K

REPORT OF FOREIGN PRIVATE ISSUER PURSUANT TO RULE 13a-16 OR 15d-16 UNDER THE SECURITIES EXCHANGE ACT OF 1934

For the month of November 2019

Commission File Number: 001-35776

ACASTI PHARMA INC.
(Translation of registrant's name into English)

**545 Promende du Centropolis
Suite 100
Laval, Québec
Canada H7T 0A3**
(Address of principal executive office)

Indicate by check mark whether the registrant files or will file annual reports under cover of Form 20-F or Form 40-F.
Form 20-F Form 40-F

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1):

Indicate by check mark if the registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7):

If "Yes" is marked, indicate below the file number assigned to the registrant in connection with Rule 12g3-2(b): N/A

This Report on Form 6-K including the exhibits hereto shall be deemed to be incorporated by reference into Acasti Pharma Inc.'s registration statement on Form S-8 (File No. 333-191383) and to be a part thereof from the date on which this report is furnished, to the extent not superseded by documents or reports subsequently filed or furnished.

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, thereunto duly authorized.

ACASTI PHARMA INC.

Date: November 18, 2019

By: /s/ Jan D'Alvise
Name: Jan D'Alvise
Title: Chief Executive Officer

EXHIBIT INDEX

Exhibit Number **Description**

99.1 Press Release dated November 18, 2019

Acasti Pharma Releases Preliminary New Animal Data, and Gains Insights Into CaPre's Novel Mechanism of Action in Diabetes

- *Data from a new preclinical mouse study suggests a unique mechanism of CaPre compared to metformin and icosapent ethyl (VASCEPA®) in a diet-induced obesity animal model*
- *Preliminary, statistically significant findings show that CaPre may promote insulin secretion, and showed a significant increase in plasma levels of 17S-HDHA and PDX as compared to metformin and icosapent ethyl*
- *Despite the lower concentration of EPA in CaPre's composition, the actual levels of 18RS-HEPE (a metabolite of EPA) reached in the blood were higher for CaPre than levels produced by icosapent ethyl. 18RS-HEPE and Resolvin E1 are both involved in the resolution of inflammation that is triggered in many chronic diseases including obesity and diabetes.*

LAVAL, Québec, Nov. 18, 2019 (GLOBE NEWSWIRE) -- Acasti Pharma Inc. ("Acasti or the "Company") (NASDAQ: ACST – TSX-V: ACST), a biopharmaceutical innovator focused on the research, development and commercialization of its prescription drug candidate CaPre® (omega-3 phospholipid) for the treatment of severe hypertriglyceridemia (HTG), released some preliminary new animal data today which provides additional insights into CaPre's potential mechanism of action in diabetes. In the Company's Phase 2 studies in humans, a statistically significant reduction of hemoglobin A1c (HbA1c) was seen in the 4 gram treatment arm of the COLT study. This is the same dose that is currently being tested in Acasti's TRILOGY Phase 3 program in humans. This positive HbA1c result in COLT was surprising at the time, and potentially unique to CaPre, as other therapeutic OM3s had previously shown no effect on glucose metabolism in a diabetic or pre-diabetic population. The main objective for this new mechanistic diabetes mouse study was to assess if CaPre acts on glucose and/or insulin in some unique manner, and to compare results head-to-head with icosapent ethyl (VASCEPA), a marketed omega-3 therapeutic, and metformin, a widely prescribed diabetic medication. Acasti collaborated with Professor André Marette, Ph.D. who conducted the study. Dr. Marette, who is the Director of the Pfizer Chair to study the pathogenesis of insulin resistance and cardiometabolic diseases at the University Laval, Quebec, conducted the study for Acasti in a widely used and well accepted animal model in diet-induced obese C57BL6 mice to compare the mechanisms of action of CaPre versus icosapent ethyl and metformin on insulin resistance and type 2 diabetes. A second, still ongoing study will also compare these same drugs in a fatty liver disease animal model. Dr. Marette is a highly regarded researcher of cardiometabolic disease, and he has published numerous papers in prestigious journals such as Nature Medicine.

The preliminary findings obtained for the diabetes mouse study showed that CaPre may promote insulin secretion as seen by statistically significant results produced in a standard glucose challenge test, thus suggesting a mechanism of action different and unique when compared to metformin, which does not promote insulin secretion. Furthermore, icosapent ethyl showed no effect on insulin or any improvement in glucose metabolism or management.

Key additional findings from this diabetic mouse study are:

- CaPre increased insulin production in association with increased c-peptide levels, suggesting that this effect is linked to greater insulin secretion by β cells. This was also associated with a tendency for lower glucose responses during a glucose challenge test. CaPre exhibited a dose response, where the higher the dose the more insulin was secreted.
- Both CaPre and icosapent ethyl significantly increased plasma 18RS-HEPE, (a metabolite of EPA and a precursor of Resolvin E1) as compared to the untreated control and metformin groups. Despite the lower levels of EPA in CaPre's composition, the actual levels of 18RS-HEPE reached in the blood were higher for CaPre than levels produced by icosapent ethyl. Again, a dose response effect was seen with CaPre. 18RS-HEPE and Resolvin E1 are both resolving mediators of OM3s, and particularly EPA, and they are involved in the resolution of inflammation that is triggered in many chronic diseases including obesity and diabetes.
- Both high dose (HED or human equivalent dose of 4 grams/day), and low dose (HED of 2 grams/day) of CaPre significantly increased plasma levels of 17S-HDHA and PDX (two metabolites of DHA) as compared to the untreated control group. The effects of high dose CaPre on PDX was very robust and significant, and much greater than those of icosapent ethyl, which showed virtually no response. Research has shown that increased levels of PDX improves insulin sensitivity in various models of insulin resistance and diabetes by several mechanisms, including by limiting inflammation in metabolic tissues, as well as by enhancing skeletal muscle IL-6 secretion, AMP activated protein kinase (AMPK) activation and glucose uptake, and by enhancing insulin's ability to suppress hepatic glucose production, which is also elevated in diabetic patients.

Data from the diabetic mouse study are still being compiled and finalized. The second study underway in a fatty liver/NASH disease model, will further confirm the findings of the diabetes study, and may potentially provide more insight into the mechanism of action of CaPre on the plasma lipid profile, and in fatty liver disease by further comparing the impact of CaPre on plasma TGs, LDL-C and HDL-C, as well as on hepatic lipid accumulation versus that of icosapent ethyl and metformin. Based on the findings from both of these study phases, Acasti and Dr. Marette plan to submit the data to a peer review journal for publication. Acasti may also file additional patents covering unique aspects and applications of this expanded understanding of CaPre's mechanism of action.

Dr. Marette commented, "Our initial pre-clinical studies with CaPre are very promising. The effect of CaPre on insulin secretion may suggest preservation of beta-cell function early in the development of type 2 diabetes. The robust increase in plasma PDX levels with CaPre treatment is also of marked interest given the pleiotropic action of this key anti-inflammatory and pro-metabolic molecule. We have not seen this in previous studies with other OM3s using similar pre-clinical models of diabetes."

Pierre Lemieux, Ph.D., COO and CSO of Acasti, added, “We are very pleased to collaborate with Dr. Marette. He has been a leader in the omega-3 research field, and especially in elucidating the importance and the role of resolvins and protectins (PDX) in the management of inflammation related to insulin resistance and glucose management in diabetes. These studies may also further reinforce and explain some of the unique and positive results reported in our Phase 2 human clinical trials. Furthermore, based on this data, we now plan to expand our list of exploratory markers to be evaluated in our TRILOGY Phase 3 program to include the resolvins and protectins such as PDX and other related pro-resolution molecules.”

About CaPre (omega-3 phospholipid)

Acasti’s prescription drug candidate, CaPre, is a highly purified omega-3 phospholipid concentrate derived from krill oil, and is being developed to treat severe hypertriglyceridemia, a metabolic condition that contributes to increased risk of cardiovascular disease and pancreatitis. Its omega-3s, principally EPA and DHA, are either “free” or bound to phospholipids, which allows for better absorption into the body. Acasti believes that EPA and DHA are more efficiently transported by phospholipids sourced from krill oil than the EPA and DHA contained in fish oil that are transported either by triglycerides (as in dietary supplements) or as ethyl esters in other prescription omega-3 drugs, which must then undergo additional digestion before they are ready for transport in the bloodstream. Clinically, the phospholipids may not only improve the absorption, distribution, and metabolism of omega-3s, but they may also decrease the synthesis of LDL cholesterol in the liver, impede or block cholesterol absorption, and stimulate lipid secretion from bile. In two Phase 2 studies, CaPre achieved a statistically significant reduction of triglycerides and non-HDL cholesterol levels in patients across the dyslipidemia spectrum from patients with mild to moderate hypertriglyceridemia (patients with TG blood levels between 200mg/dl and 500mg/dl) to patients with severe hypertriglyceridemia (those with TG levels above 500mg/dl). Furthermore, in the Phase 2 studies, CaPre demonstrated the potential to actually reduce LDL, or “bad cholesterol”, as well as the potential to increase HDL, or “good cholesterol”, especially at the therapeutic dose of 4 grams/day. The Phase 2 data also showed a significant reduction of HbA1c at a 4 gram dose, suggesting that due to its unique omega-3/phospholipid composition, CaPre may actually improve long-term glucose metabolism. Acasti’s TRILOGY Phase 3 program is currently underway.

About Acasti Pharma

Acasti Pharma is a biopharmaceutical innovator advancing a potentially best-in-class cardiovascular drug, CaPre® (omega-3 phospholipid), for the treatment of hypertriglyceridemia, a chronic condition affecting an estimated one third of the U.S. population. Since its founding in 2008, Acasti Pharma has focused on addressing a critical market need for an effective, safe and well-absorbing omega-3 therapeutic that can make a positive impact on the major blood lipids associated with cardiovascular disease risk. The company is developing CaPre in a Phase 3 clinical program in patients with severe hypertriglyceridemia, a market that includes 3 to 4 million patients in the U.S. The addressable market may expand significantly if omega-3s demonstrate long-term cardiovascular benefits in on-going third party outcomes studies. Acasti may need to conduct at least one additional clinical trial to support FDA approval of a supplemental New Drug Application to expand CaPre’s indications to this segment. Acasti’s strategy is to commercialize CaPre in the U.S. and the company is pursuing development and distribution partnerships to market CaPre in major countries around the world. For more information, visit www.acastipharma.com.

Forward Looking Statements

Statements in this press release that are not statements of historical or current fact constitute “forward-looking information” within the meaning of Canadian securities laws and “forward-looking statements” within the meaning of U.S. federal securities laws (collectively, “forward-looking statements”). Such forward-looking statements involve known and unknown risks, uncertainties, and other unknown factors that could cause the actual results of Acasti to be materially different from historical results or from any future results expressed or implied by such forward-looking statements. In addition to statements which explicitly describe such risks and uncertainties, readers are urged to consider statements labeled with the terms “believes,” “belief,” “expects,” “intends,” “anticipates,” “potential,” “should,” “may,” “will,” “plans,” “continue,” “targeted” or other similar expressions to be uncertain and forward-looking. Readers are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date of this press release. Forward-looking statements in this press release include, but are not limited to, information or statements about Acasti’s strategy, future operations, prospects and the plans of management; Acasti’s ability to conduct all required clinical and non-clinical trials for CaPre, including the timing and results of those trials; the timing and the outcome of licensing negotiations; CaPre’s potential to become the “best-in-class” cardiovascular drug for treating severe Hypertriglyceridemia (HTG), Acasti’s ability to commercially launch CaPre, CaPre’s potential to meet or exceed the target primary endpoint of reducing triglycerides by 20% compared to placebo, CaPre’s potential mechanism of action in diabetes, and Acasti’s ability to fund its continued operations.

The forward-looking statements contained in this press release are expressly qualified in their entirety by this cautionary statement, the “Cautionary Note Regarding Forward-Looking Information” section contained in Acasti’s latest annual report on Form 20-F and most recent management’s discussion and analysis (MD&A), which are available on SEDAR at www.sedar.com, on EDGAR at www.sec.gov/edgar/shtml, and on the investor section of Acasti’s website at www.acastipharma.com. All forward-looking statements in this press release are made as of the date of this press release. Acasti does not undertake to update any such forward-looking statements whether as a result of new information, future events or otherwise, except as required by law. The forward-looking statements contained herein are also subject generally to assumptions and risks and uncertainties that are described from time to time in Acasti’s public securities filings with the Securities and Exchange Commission and the Canadian securities commissions, including Acasti’s latest annual report on Form 20-F and most recent MD&A.

Neither NASDAQ, the TSX Venture Exchange nor its Regulation Services Provider (as that term is defined in the policies of the TSX Venture Exchange) accepts responsibility for the adequacy or accuracy of this release.

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