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

December 2020

Commission file number: 001-36288

Akari Therapeutics, Plc

(Translation of registrant's name into English)

75/76 Wimpole Street
London W1G 9RT
United Kingdom
(Address of principal executive offices)

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 Regulations S-T Rule 101(b)(1): _____

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

CONTENTS

On December 3, 2020, Akari Therapeutics, Plc (the "Company") issued a press release announcing a publication in the American Journal of Pathology highlighting the potential of nomacopan in the treatment of uveitis and retinal disease.

A copy of the press release is attached hereto as Exhibit 99.1 and incorporated herein by reference.

The information in paragraph one of Exhibit 99.1 is hereby incorporated by reference into all effective registration statements filed by the Company under the Securities Act of 1933.

Exhibit No.

99.1 Press release dated December 3, 2020

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.

Akari Therapeutics, Plc
(Registrant)

By: /s/ Clive Richardson
Name: Clive Richardson
Chief Executive Officer and Chief Operating Officer

Date: December 3, 2020

Akari Therapeutics Announces Publication in *American Journal of Pathology* Highlighting Potential of Nomacopan in Treatment of Uveitis and Retinal Disease

- *New data highlight role of leukotriene B4 (LTB4) in the pathophysiology of retinal inflammation and degeneration*
- *In the experimental allergic uveitis (EAU) disease model nomacopan reduced Th17 effector T cell and macrophage activity in addition to known downregulation of neutrophils*
- *Long-acting forms of nomacopan were at least as effective in downregulating disease in EAU model as standard of care dexamethasone, which is associated with steroid related side effects*
- *Akari is investigating the potential of two long-acting forms of nomacopan which inhibit, (1) LTB4 and C5 combined or (2) LTB4 alone for treatment of back of the eye diseases*

NEW YORK and LONDON, December 3, 2020 – Akari Therapeutics, Plc (Nasdaq: AKTX), a biopharmaceutical company focused on innovative therapeutics to treat orphan autoimmune and inflammatory diseases where the complement and/or leukotriene systems are implicated, today announces the publication of the results of a two-year research collaboration with the University College of London (UCL) Institute of Ophthalmology. The results showed that the therapeutic intravitreal (IVT) administration of long-acting nomacopan mitigated both the severity and progress of retinal damage in two models of autoimmune uveitis, a severe inflammatory eye disease where steroids are the primary treatment option. In addition, results showed the presence of inflammatory cells expressing both complement C5 and LTB4 receptors in retinal tissue from donor patients with uveitis as compared to healthy donor eyes.

These data point to the potential importance of LTB4 in the development of inflammatory retinal diseases which potentially include proliferative age-related macular degeneration (wet AMD), diabetic retinopathy, diabetic macular oedema and autoimmune uveitis.

While complement C5 activation has previously been shown to have a causative role in late stage EAU[1,2], the authors of the paper focused on the less well understood contribution of LTB4 to uveitis disease pathology.

Key findings were that IVT administration of the long-acting engineered form of nomacopan that only binds to LTB4 was at least as effective as the steroid dexamethasone in reducing retinal inflammation. Nomacopan significantly decreased proliferation, differentiation and infiltration of Th17 and Th1/17 effector CD4[3] T cells and reduced activated macrophage cell populations, each of which is reported as having a causative role in uveitis disease pathology.

Intravitreal LTB4 and C5a was elevated in response to EAU and the level rose from initiation until the disease peaked at Day 21. Few LTB4 or C5a receptors were detected in healthy human donor eyes, but significant numbers of infiltrating cells displaying these receptors were detected in uveitic eyes of EAU mice and in post-mortem retinal sections from patients with uveitis, supporting the likely role of LTB4 and C5 in the disease pathology.

Corticosteroids such as dexamethasone are the current standard of care for uveitis but may be associated with significant steroid related side effects such as increased risk of raised intraocular pressure, cataract formation, keratopathy and macular oedema, hence an equally effective and safer alternative treatment would be well received.

In conclusion, the new data highlight the likely importance of LTB4 in the aetiology of retinal inflammation and effectiveness of nomacopan's LTB4 inhibitory activity in the model of uveitis. The data also show that inhibition of LTB4 decreases Th17 effector cell differentiation and proliferation and activation of macrophages, which, along with complement inhibition, may not only be relevant in uveitis but also for other autoimmune diseases for which nomacopan is being developed, including bullous pemphigoid.

Dr. Miles Nunn, Chief Scientific Officer of Akari Therapeutics, said, "This study confirms the important role of LTB4 in uveitis and likely other autoimmune diseases by downregulating granulocytes, T cells and macrophages that may cause and direct inflammation and tissue damage. Complement dysregulation is implicated in several back of the eye diseases including dry AMD and as such the combined inhibition of C5 and LTB4 offers a potential unique treatment alternative."

Professor Virginia Calder of UCL institute of Ophthalmology said, “There is a need for safer, localized treatments for patients with uveitis since steroids are especially problematic for the eye. These new findings point to LTB4 playing a key role during disease, and the anti-inflammatory effect of nomacopan in EAU demonstrates an exciting and novel potential therapeutic option.”

[1] D. A. Copland, K. Hussain, S. Baalasubramanian, T. R. Hughes, B. P. Morgan, H. Xu, A. D. Dick and L. B. Nicholson. Systemic and local anti-C5 therapy reduces the disease severity in experimental autoimmune uveoretinitis. British Society for Immunology, Clinical and Experimental Immunology, 159: 303–314. 2009.

[2] S. Reinehr, S.C. Gomes, C.J. Gassel, M. Ali Asaad, Gesa Stute, M. Schargus, H. Burkhard Dick, S.C. Joachim. Intravitreal Therapy Against the Complement Factor C5 Prevents Retinal Degeneration in an Experimental Autoimmune Glaucoma Model. Frontiers in Pharmacology. December 2019, vol 10, article 1381.

[3] Eskandarpour M, Chen YH, Nunn MA, Coupland SE, Weston-Davies W, Calder VL. Leukotriene B4 (LTB4) and its receptor in Experimental Autoimmune Uveitis (EAU) and in human retinal tissues: clinical severity and LTB4-dependence of retinal Th17 cells. Am J Pathol. 2020 Nov 4:S0002-9440(20)30491-0. doi: 10.1016/j.ajpath.2020.10.010. Epub ahead of print. PMID: 33159884.

About Akari Therapeutics

Akari is a biopharmaceutical company focused on developing inhibitors of acute and chronic inflammation, specifically for the treatment of rare and orphan diseases, in particular those where the complement (C5) or leukotriene (LTB4) systems, or both complement and leukotrienes together, play a primary role in disease progression. Akari’s lead drug candidate, nomacopan (formerly known as Coversin), is a C5 complement inhibitor that also independently and specifically inhibits leukotriene B4 (LTB4) activity.

Cautionary Note Regarding Forward-Looking Statements

Certain statements in this press release constitute “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995. You should not place undue reliance upon the Company’s forward looking statements. Except as required by law, the Company undertakes no obligation to revise or update any forward-looking statements in order to reflect any event or circumstance that may arise after the date of this press release. These forward-looking statements reflect our current views about our plans, intentions, expectations, strategies and prospects, which are based on the information currently available to us and on assumptions we have made. Although we believe that our plans, intentions, expectations, strategies and prospects as reflected in or suggested by those forward-looking statements are reasonable, we can give no assurance that the plans, intentions, expectations or strategies will be attained or achieved. Furthermore, actual results may differ materially from those described in the forward-looking statements and will be affected by a variety of risks and factors that are beyond our control. Such risks and uncertainties for our company include, but are not limited to: needs for additional capital to fund our operations, our ability to continue as a going concern; uncertainties of cash flows and inability to meet working capital needs; an inability or delay in obtaining required regulatory approvals for nomacopan and any other product candidates, which may result in unexpected cost expenditures; our ability to obtain orphan drug designation in additional indications; risks inherent in drug development in general; uncertainties in obtaining successful clinical results for nomacopan and any other product candidates and unexpected costs that may result therefrom; difficulties enrolling patients in our clinical trials; our ability to enter into collaborative, licensing, and other commercial relationships and on terms commercially reasonable to us; failure to realize any value of nomacopan and any other product candidates developed and being developed in light of inherent risks and difficulties involved in successfully bringing product candidates to market; inability to develop new product candidates and support existing product candidates; the approval by the FDA and EMA and any other similar foreign regulatory authorities of other competing or superior products brought to market; risks resulting from unforeseen side effects; risk that the market for nomacopan may not be as large as expected; risks associated with the COVID-19 pandemic; risks associated with the SEC investigation; inability to obtain, maintain and enforce patents and other intellectual property rights or the unexpected costs associated with such enforcement or litigation; inability to obtain and maintain commercial manufacturing arrangements with third party manufacturers or establish commercial scale manufacturing capabilities; the inability to timely source adequate supply of our active pharmaceutical ingredients from third party manufacturers on whom the company depends; unexpected cost increases and pricing pressures and risks and other risk factors detailed in our public filings with the U.S. Securities and Exchange Commission, including our most recently filed Annual Report on Form 20-F filed with the SEC. Except as otherwise noted, these forward-looking statements speak only as of the date of this press release and we undertake no obligation to update or revise any of these statements to reflect events or circumstances occurring after this press release. We caution investors not to place considerable reliance on the forward-looking statements contained in this press release.

Investor Contact:
Peter Vozzo
Westwicke
+1 (443) 213-0505
peter.vozzo@westwicke.com

Media Contact:
Sukaina Virji / Lizzie Seeley
Consilium Strategic Communications
+44 (0)20 3709 5700
Akari@consilium-comms.com