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

January 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):_____

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On January 27, 2020, Akari Therapeutics, Plc (the “Company”) issued a press release announcing new preclinical data indicating that nomacopan significantly reduced both retinal inflammation and Vascular Endothelial Growth Factor (VEGF). 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 January 27, 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
Title: Chief Executive Officer and Chief Operating Officer

Date: January 27, 2020

Exhibit 99.1

Akari Therapeutics Announces Preclinical Ophthalmic Data Showing Nomacopan Reduces Both Vascular Endothelial Growth Factor (VEGF) and Retinal Inflammation Supporting Nomacopan as a Potential Treatment Option for Back-of-the-Eye Diseases

- The new data show PAS-nomacopan (the long acting form of Akari's lead drug, nomacopan) significantly reduced VEGF levels and retinal inflammation, both of which are associated with permanent damage in many back-of-the-eye diseases
    - PAS-nomacopan lowered intraocular levels of VEGF as effectively as an anti-VEGF antibody; 74% and 68% reduction respectively, compared to saline control
    - PAS-nomacopan also significantly reduced retinal inflammation compared to saline, unlike the anti-VEGF antibody
- Previously reported data showed cell-activating receptors for complement-C5a and leukotriene B4 (LTB4) in the retina. Nomacopan inhibits C5a and LTB4 signalling via these receptors.
- By inhibiting VEGF and complement, and reducing retinal inflammation, nomacopan has the potential to treat multiple pathways implicated in back-of-the-eye diseases.
- PAS-nomacopan, with its longer half-life, offers potential for less frequent injections (intravitreal dosing), which is critical in the treatment of back-of-the-eye diseases.

NEW YORK and LONDON, January 27, 2020– Akari Therapeutics, Plc (Nasdaq: AKTX), a biopharmaceutical company focused on innovative therapeutics to treat autoimmune and inflammatory diseases where the complement and/or leukotriene systems are implicated, announces new preclinical data indicating that nomacopan significantly reduced both retinal inflammation and intraocular VEGF.

Treatment of back-of-the-eye diseases, including angiogenic ocular diseases with new blood vessel growth such as diabetic retinopathy and wet age-related macular degeneration (AMD), is a multi-billion dollar market. VEGF promotes angiogenesis in these latter conditions, and inhibition of VEGF with antibodies such as *Lucentis*® and antagonists such as *Macugen*® and *Eyelea*® form the mainstay of treatment of wet AMD. In contrast, complement inhibitors are being explored as a treatment for dry AMD.

"Despite the successful use of VEGF inhibitors, many patients with AMD and other back-of-the-eye diseases remain sub-optimally treated," said Professor Virginia Calder, University College of London (UCL), Institute of Ophthalmology. "This reflects the complex natural history and pathology of these retinal diseases. An agent such as nomacopan that appears to combine anti-inflammatory activity with VEGF and complement inhibition has the potential to improve treatment options, highlighting the importance of these pathways and the degree to which their dysregulation is implicated in different sight-threatening conditions."

Professor Calder and colleagues at UCL have undertaken new work comparing the therapeutic efficacy of nomacopan, PAS-nomacopan, and a monoclonal anti-VEGF antibody all administered intravitreally. Their work utilized an established 26-day model of severe experimental autoimmune uveitis (EAU) in mice which results in elevated VEGF and retinal inflammation mediated by influx of lymphocytes which thereby mimics the pathology seen in several back-of the eye diseases. Drug treatments were administered on day 15 once disease was established and ongoing.

PAS-nomacopan was found to reduce intraocular VEGF levels by as much as the anti-VEGF antibody with 74% (p=0.04) and 68% (p=0.05) reductions respectively, compared to saline control. Furthermore, while clinically assessed inflammation increased in both the control and anti-VEGF groups by 49% and 33%, respectively, PAS-nomacopan treatment showed a 9% reduction in inflammation assessed by retinal fundoscopy (p=0.02), supporting nomacopan's therapeutic activity across multiple pathogenic pathways.

PAS-nomacopan, which has a very large functional molecular weight of 670kDa, due to use of the PASylation^{TM} technology (Kuhn et al. 2016[1]), was more effective than nomacopan (17kDa) in reducing both VEGF levels and inflammation, probably reflecting longer residency time of PAS-nomacopan in the eye.

The rationale for using nomacopan as a treatment for the back-of-the-eye was initiated by the discovery that cell-activating receptors (C5aR1 and BLT1) that bind to complement C5a and LTB4 are expressed by retinal inflammatory cells in the back of the eye. Nomacopan inhibits C5 activation preventing formation of C5a and captures LTB4, and thereby has the potential to inhibit C5a and LTB4 signalling via the receptors identified in the retina.

Furthermore, as previously reported in an EAU model (April 26, 2019), nomacopan significantly reduced inflammatory markers such as the cytokine IL-17 together with clinical and histological inflammation. Nomacopan's activity appeared to be equivalent to potent corticosteroids, the standard of care treatment for acute posterior non-infective uveitis.

These results suggest the potential for nomacopan as a first-line treatment for multiple back-of-the-eye diseases in that the single drug has now been demonstrated to mitigate three pathways implicated in uveal, macular and retinal diseases:

- Inhibition of VEGF release, a key driver of wet AMD
- Reduced expression of inflammatory mediators such as the cytokine IL-17
- Inhibition of the complement pathway, implicated in dry AMD

Wynne Weston-Davies, Medical Director, Akari Therapeutics, said, "We have an ongoing Phase II trial in atopic keratoconjunctivitis (a surface of the eye disease) with nomacopan and are pleased to report new encouraging pre-clinical data in the back of the eye with the differentiated long-acting PAS-nomacopan. We are now actively exploring other pre-clinical models in diseases such as AMD and how best to develop these findings in the clinic where there remains a significant unmet need."

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). Nomacopan is currently being clinically evaluated in four indications: bullous pemphigoid (BP), atopic keratoconjunctivitis (AKC), thrombotic microangiopathy (TMA), and paroxysmal nocturnal hemoglobinuria (PNH). Akari believes that the dual action of nomacopan on both C5 and LTB4 may be beneficial in AKC and BP. Akari is also developing other tick derived proteins, including longer acting versions.

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[1]*Nomacopan, formerly Coversin, accepted as International Non-proprietary Name by World Health Organization.*

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 regarding, among other things, statements related to the offering, the expected gross proceeds and the expected closing of the offering. 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; our ability to enter into collaborative, licensing, and other commercial relationships and on terms commercially reasonable to us; difficulties enrolling patients in our clinical trials; 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 departure of our former Chief Executive Officers and other executive officers; 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.

For more information

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