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 August 2017

Commission File Number: **001-37384**

GALAPAGOS NV
(Translation of registrant's name into English)

Generaal De Wittelaan L11 A3
2800 Mechelen, Belgium
(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 [X] 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): _

Note: Regulation S-T Rule 101(b)(1) only permits the submission in paper of a Form 6-K if submitted solely to provide an attached annual report to security holders.

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): _

Note: Regulation S-T Rule 101(b)(7) only permits the submission in paper of a Form 6-K if submitted to furnish a report or other document that the registrant foreign private issuer must furnish and make public under the laws of the jurisdiction in which the registrant is incorporated, domiciled or legally organized (the registrant's "home country"), or under the rules of the home country exchange on which the registrant's securities are traded, as long as the report or other document is not a press release, is not required to be and has not been distributed to the registrant's security holders, and, if discussing a material event, has already been the subject of a Form 6-K submission or other Commission filing on EDGAR.

The information contained in this report on Form 6-K, including the Exhibit 99.1, except for the quotes of Dr. Piet Wigerinck and Dr. Toby Maher contained in Exhibit 99.1, is hereby incorporated by reference into the Company's Registration Statements on Forms F-3 (File No. 333-211765) and S-8 (File Nos. 333-204567, 333-208697, 333-211834, 333-215783, and 333-218160).

On August 9, 2017, the Registrant issued a press release, a copy of which is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

(c) Exhibit 99.1. Press release dated August 9, 2017

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.

GALAPAGOS NV
(Registrant)

Date: August 10, 2017

/s/ Xavier Maes
Xavier Maes
Company Secretary

EXHIBIT 99.1

GLPG1690 halts disease progression in IPF patients in FLORA Phase 2a trial

- **Forced vital capacity (FVC) in lungs stabilized over the 12-week treatment period, placebo arm showed expected decline**
- **Functional respiratory imaging (FRI) confirms FVC data with statistical significance**
- **GLPG1690 was generally well tolerated**
- **First autotaxin inhibitor to show effect in IPF patient trial**
- **GLPG1690 expected to progress to late stage trial**

Webcast presentation of the results to be held tomorrow 10 August, 14.00 CET/8 AM EDT, +32 2 404 0659, access code 2084135; more call number info further down

Mechelen, Belgium; 9 August 2017; 22.01 CET; regulated information - Galapagos NV (Euronext & NASDAQ: GLPG) announces positive topline results with its autotaxin inhibitor GLPG1690 in patients with idiopathic pulmonary fibrosis (IPF) in the FLORA Phase 2a trial.

FLORA was an exploratory, randomized, double-blind, placebo-controlled trial investigating a once-daily oral dose of GLPG1690. The drug candidate was administered for 12 weeks in 23 IPF patients, 17 of whom received GLPG1690 and 6 placebo. Primary objectives of the trial were to assess safety, tolerability, pharmacokinetics and pharmacodynamics of GLPG1690 in an IPF patient population. Secondary objectives included the evaluation of lung function, changes in disease biomarkers, FRI, and quality of life. The IPF diagnosis was confirmed by central reading. The baseline characteristics of the recruited population were in line with published data in similarly conducted studies and were balanced between active and placebo. Patients with previous experience on nintedanib or pirfenidone were required to have discontinued treatment with either agent for at least 4 weeks prior to initiating treatment with GLPG1690.

Over the 12-week period, patients receiving GLPG1690 showed an FVC increase of 8 mL, while patients on placebo showed an FVC reduction of 87 mL (mean from baseline). Such reductions in FVC in the placebo arm were in line with expectations based on similarly conducted third-party studies in IPF patients. In addition to the demonstrated absence of lung function decline over the 12 week period, more sensitive functional respiratory imaging (FRI) confirmed disease stabilization in the GLPG1690 arm, versus disease progression in the placebo arm, reaching statistical significance on two specific parameters.

Patients on GLPG1690 treatment showed a clear reduction of serum LPA18:2, a biomarker for autotaxin inhibition, as expected based on the mechanism of action of GLPG1690. Thus, the level of target engagement observed in Phase 1 with healthy volunteers was confirmed in IPF patients in FLORA.

GLPG1690 was found to be generally well tolerated in this Phase 2 trial. Rates of discontinuation due to adverse events, as well as serious adverse event rates, were similar between patients on GLPG1690 and placebo.

Galapagos plans to rapidly progress GLPG1690 in a late stage trial and had already discussions with regulators regarding trial design.

"Galapagos' results with GLPG1690 are extremely exciting and exceed those of previous studies. This brings hope to patients with idiopathic pulmonary fibrosis that new effective treatment may be on the horizon. Importantly, some patients even showed an increase of lung function within only 12 weeks of treatment, and the drug was well tolerated. The results from FLORA beg the question how patients will fare with longer treatment. I urge Galapagos and the IPF community to progress to the next phase of clinical trials as rapidly as possible," said Dr. Toby Maher, Professor of Interstitial Lung Disease at Imperial College, London and Consultant Physician at Royal Brompton Hospital, London.

"Not only does GLPG1690 show early promise as a potential therapy for IPF, but it also marks an important milestone for Galapagos as a company: proof of concept in patients of a second mechanism of action coming from our target discovery platform. Galapagos has shown that this platform continues to deliver novel mechanisms of action beyond JAK1 in inflammation. The stabilization of FVC over 12 weeks upon GLPG1690 treatment is a major milestone in IPF, where, by way of reference, the currently approved treatments show a decrease of approximately 30 mL over the same treatment period," added Dr. Piet Wigerinck, Chief Scientific Officer of Galapagos.

Galapagos plans to report the FLORA study results at a future medical conference.

Conference call and webcast presentation

Galapagos will conduct a conference call open to the public tomorrow, 10 August 2017, at 14:00 CET / 8 AM EDT, which will also be webcasted. To participate in the conference call, please call one of the following numbers ten minutes prior to commencement:

Confirmation Code: **2084135**

Belgium: +32 2 404 0659

France: +33 1 7677 2274

Netherlands: +31 20 721 9251

United Kingdom: +44 330 336 9411

United States: +1 719 325 2226

A question and answer session will follow the presentation of the results. Go to www.glpg.com to access the live audio webcast. The archived webcast, PDF of the slides, and a transcript will also be available on the Galapagos website later in the day.

About GLPG1690

GLPG1690 is a small molecule, selective autotaxin inhibitor which is fully proprietary to Galapagos. Galapagos identified the autotaxin target using its proprietary target discovery platform and developed molecule GLPG1690 as an inhibitor of this target. GLPG1690 showed promising results in relevant pre-clinical models for IPF, and there is growing evidence in scientific literature that autotaxin plays a role in this disease. GLPG1690 successfully completed a Phase 1 trial in 2015, showing favorable findings relating to safety and tolerability, and high target engagement in healthy volunteers. Galapagos received orphan drug designation for GLPG1690 in IPF from the U.S. Food & Drug Administration (FDA) and European Commission (EC). GLPG1690 is an investigational drug and its efficacy and safety have not been established.

For information about the studies with GLPG1690: www.clinicaltrials.gov

For more information about GLPG1690: www.glpg.com/glpg-1690

About IPF

IPF is a chronic, relentlessly progressive fibrotic disorder of the lungs that typically affects adults over the age of 40. There are approximately 200,000 patients with IPF in the U.S. and Europe, with 75,000 newly diagnosed patients per year. As such, IPF is considered a rare disease. The clinical prognosis of patients with IPF is poor as the median survival at diagnosis is 2 to 5 years. Currently, no medical therapies have been found to cure IPF. The medical treatment strategy aims to slow the disease progression and improve the quality of life. Lung transplantation may be an option for appropriate patients with progressive disease and minimal comorbidities.

Regulatory agencies have approved Esbriet®[1] (pirfenidone) and Ofev®[2] (nintedanib) for the treatment of IPF. Both pirfenidone and nintedanib have been shown to slow the rate of lung function decline in IPF and are likely to become the standard of care worldwide. These regulatory approvals represent a major breakthrough for IPF patients; yet neither drug improves lung function, and the disease continues to progress in the majority of patients despite treatment. Moreover, the adverse effects associated with these therapies include diarrhea, liver function test abnormalities with nintedanib, nausea and rash with pirfenidone. Therefore, there is still a large unmet medical need as IPF remains a major cause of morbidity and mortality.

About Galapagos

Galapagos (Euronext & NASDAQ: GLPG) is a clinical-stage biotechnology company specialized in the discovery and development of small molecule medicines with novel modes of action. Our pipeline comprises Phase 3, Phase 2, Phase 1, pre-clinical, and discovery programs in cystic fibrosis, inflammation, fibrosis, osteoarthritis and other indications. We have discovered and developed filgotinib: in collaboration with Gilead we aim to bring this JAK1-selective inhibitor for inflammatory indications to patients all over the world. Galapagos is focused on the development and commercialization of novel medicines that will improve people's lives. The Galapagos group, including fee-for-service subsidiary Fidelta, has approximately 550 employees, operating from its Mechelen, Belgium headquarters and facilities in The Netherlands, France, and Croatia. More information at www.glpg.com.

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This press release contains inside information within the meaning of Regulation (EU) No 596/2014 of the European Parliament and of the Council of 16 April 2014 on market abuse (market abuse regulation).

Forward-looking statements

This release may contain forward-looking statements, including statements regarding Galapagos' strategic ambitions, the potential activity of GLPG1690, the anticipated timing of future clinical studies with GLPG1690, the progression and results of such studies, and Galapagos' interactions with regulatory authorities. Galapagos cautions the reader that forward-looking statements are not guarantees of future performance. Forward-looking statements involve known and unknown risks, uncertainties and other factors which might cause the actual results, financial condition and liquidity, performance or achievements of Galapagos, or industry results, to be materially different from any historic or future results, financial conditions and liquidity, performance or achievements expressed or implied by such forward-looking statements. In addition, even if Galapagos' results, performance, financial condition and liquidity, and the development of the industry in which it operates are consistent with such forward-looking statements, they may not be predictive of results or developments in future periods. Among the factors that may result in differences are the inherent uncertainties associated with competitive developments, clinical trial and product development activities and regulatory approval requirements (including that data from the ongoing and planned clinical research programs may not support registration or further development of GLPG1690 due to safety, efficacy or other reasons), Galapagos' reliance on collaborations with third parties, and estimating the commercial potential of Galapagos' product candidates. A further list and description of these risks, uncertainties and other risks can be found in Galapagos' Securities and Exchange Commission (SEC) filings and reports, including in Galapagos' most recent annual report on form 20-F filed with the SEC and subsequent filings and reports filed by Galapagos with the SEC. Given these uncertainties, the reader is advised not to place any undue reliance on such forward-looking statements. These forward-looking statements speak only as of the date of publication of this document. Galapagos expressly disclaims any obligation to update any such forward-looking statements in this document to reflect any change in its expectations with regard thereto or any change in events, conditions or circumstances on which any such statement is based or that may affect the likelihood that actual results will differ from those set forth in the forward-looking statements, unless specifically required by law or regulation.

[1] Esbriet® (pirfenidone) is indicated for the treatment of IPF by Roche/Genentech.

[2] Ofev® (nintedanib) is indicated for the treatment of IPF by Boehringer Ingelheim.