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Poxel Announces Participation at the H.C. Wainwright 3rd Annual NASH Investor Conference

- **Presentation will include an overview of Poxel's two clinical-stage programs for the treatment of NASH**

LYON, France--(BUSINESS WIRE)-- [POXEL S.A.](#) (Euronext – POXEL - FR0012432516), a biopharmaceutical company focused on the development of innovative treatments for metabolic disorders, including type 2 diabetes and non-alcoholic steatohepatitis (NASH), announced today that it will be featured as a presenting company at the H.C. Wainwright 3rd Annual NASH Investor Conference and the Company will provide an overview of its two clinical stage candidates, PXL770, a direct adenosine monophosphate-activated protein kinase (AMPK) activator and PXL065, a mitochondrial pyruvate carrier (MPC) inhibitor, being developed for the treatment of NASH. Clinical results for PXL770 and PXL065 are expected in the fourth quarter of 2019.

The H.C. Wainwright 3rd Annual NASH Investor Conference is being held on October 21, 2019 at The St. Regis Hotel in New York City. The Company will present an overview of its NASH programs on Monday, October 21st from 4:00-4:20 PM ET.

The presentation at the H.C. Wainwright 3rd Annual NASH Investor Conference will be webcast live. To access the webcast, please visit the following link <http://wsf.com/webcast/hcw6/poxel.pa/>. The webcast replay will remain available for 90 days following the live presentation.

For PXL770, two clinical trials are currently underway. One trial is a Phase 2a clinical study examining the efficacy and safety of PXL770 with results expected in the second quarter of 2020. In parallel, a pharmacokinetic (PK) and pharmacodynamic (PD) trial is ongoing to assess the full PK profile and PD effect on target pathways and metabolic parameters. Results from this trial are expected in the fourth quarter of 2019.

For PXL065, a Phase 1b multiple ascending dose (MAD) trial is underway and results are expected in the fourth quarter of 2019. The Phase 1b MAD trial is designed to evaluate safety, tolerability and PK and support dose selection for the registration program.

About PXL770

PXL770 is a first-in-class direct adenosine monophosphate-activated protein kinase (AMPK) activator. AMPK is a central regulator of multiple metabolic pathways leading to the control

of lipid metabolism, glucose homeostasis and inflammation. Based on its central metabolic role, targeting AMPK offers the opportunity to pursue a wide range of indications to treat chronic metabolic diseases, including diseases that affect the liver, such as non-alcoholic steatohepatitis (NASH)¹.

About PXL065

PXL065 is deuterium-stabilized R-pioglitazone. Pioglitazone is the most extensively studied drug for NASH and has demonstrated “resolution of NASH without worsening of fibrosis” in a Phase 4 trial². Pioglitazone is the only drug recommended for biopsy-proven NASH patients by the Practice Guidelines published by the American Association for the Study of Liver Diseases (AASLD) and the European Association for the Study of the Liver (EASL)³. Pioglitazone’s use for NASH, however, has been limited due to the PPAR γ -related side effects, which include weight gain, bone fractures and fluid retention.

Pioglitazone is a 1:1 mixture of two mirror-image compounds (R- and S-stereoisomers) that interconvert *in vivo*. Using deuterium, we stabilized each stereoisomer and characterized their dramatically different pharmacological properties. In *in vitro* studies, PXL065 has been shown to target mitochondrial pyruvate carrier (MPC) as an inhibitor. In preclinical models, PXL065 exhibits the anti-inflammatory activity and NASH efficacy associated with pioglitazone with little or no weight gain or fluid retention, side effects which are associated with the S-stereoisomer. Based upon preclinical and Phase 1 results to date, PXL065 is expected to exhibit a better therapeutic profile than pioglitazone for NASH.

About Poxel SA

Poxel is a **dynamic biopharmaceutical company** that uses its extensive expertise in developing **innovative drugs for metabolic diseases**, with a focus on **type 2 diabetes** and **non-alcoholic steatohepatitis (NASH)**. In its mid-to-late stage pipeline, the Company is currently advancing three drug candidates as well as earlier-stage opportunities. **Imeglimin**, Poxel’s first-in-class lead product, targets mitochondrial dysfunction. Together, with its partner Sumitomo Dainippon Pharma, Poxel is conducting the Phase 3 **Trials of IMeglimin for Efficacy and Safety (TIMES)** program for the treatment of type 2 diabetes in Japan. Poxel also established a partnership with Roivant Sciences for Imeglimin’s development and commercialization in countries outside of the partnership with Sumitomo Dainippon Pharma, including the U.S. and Europe. **PXL770**, a first-in-class direct adenosine monophosphate-activated protein kinase (AMPK) activator, is in a Phase 2a proof-of-concept program for the treatment of NASH. PXL770 could also have the potential to treat additional metabolic diseases. **PXL065** (deuterium-stabilized R-pioglitazone), a mitochondrial pyruvate carrier (MPC) inhibitor, is in Phase 1 clinical testing and being developed for the treatment of NASH. Poxel also has additional earlier-stage programs targeting metabolic, specialty and rare diseases. The Company intends to generate further growth through strategic partnerships and pipeline development. Listed on Euronext Paris, Poxel is headquartered in Lyon, France, and has subsidiaries in Boston, MA, and Tokyo, Japan. For more information, please visit: www.poxelpharma.com.

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and other words and terms of similar meaning or the negative thereof. Forward-looking statements are subject to inherent risks and uncertainties beyond the Company's control that could cause the Company's actual results or performance to be materially different from the expected results or performance expressed or implied by such forward-looking statements.

¹ Source: Smith B. K et al., (2016) Am J Physiol Endocrinol Metab 311, E730 – E740.

² Cusi, et al., Ann Intern Med. 2016, 165(5), 305-315).

³ J Hepatol. 2016, 64(6),1388-402; Hepatology 2018, 67, 328-357.

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