

Post-doctoral position in Cell Biology @ Institut Jacques Monod – Paris, France

A 1.5-year post-doctoral position, funded by the French National Research Agency (ANR), is available in Sebastien Léon's lab at the Institut Jacques Monod in Paris, France.

Work in the lab aims at understanding the molecular mechanisms leading to cellular adaptation to nutritional challenges. The funded project specifically aims at identifying novel mechanisms leading to nutrient-dependent remodeling of the plasma membrane proteome using yeast as a model system, following upon our previous studies (see refs. below). For more information, please visit <u>http://tinyurl.com/leonlab</u>.

The key goals are to elucidate the mechanisms in charge of the nutrient-regulated ubiquitylation of transporters, with emphasis on the regulation of arrestin-related proteins. The approaches used range from cell biology techniques (live cell microscopy, microfluidics, BiFC), genetics and biochemistry (recombinant protein purification, *in vivo* and *in vitro* ubiquitylation, interaction proteomics) to systems-wide approaches (robotized genetic screens, high-throughput imaging, quantitative proteomics). Confirmation of key results in mammalian cell cultures will be possible in the lab.

Applicants must have a PhD in Life Sciences, a strong background in cell biology or biochemistry, and an excellent track record. Candidates must be rigorous, self-motivated and have a strong team spirit. Experience with yeast is not a requirement. Start is at earliest convenience.

The IJM (<u>http://www.ijm.fr</u>) is one of the largest research centers in fundamental biology in Paris, and a multidisciplinary working environment. It gathers 30 teams and state-of-the-art imaging/proteomics facilities that are among the best in the country.

Applications in the form of a CV and motivation letter, including the contact information for 2 referees, should be sent to Sebastien Leon, <u>sebastien.leon@ijm.fr</u>.

References.

I Hovsepian et al. 2018. The yeast arrestin-related protein Bul1 is a novel actor of glucose-induced endocytosis. Mol Biol Cell. 29:1012-1020. I Hovsepian et al. 2017. Multilevel regulation of an alpha-arrestin by glucose depletion controls hexose transporter endocytosis. J Cell Biol. 216:1811-1831. I Becuwe & Léon. 2014. Integrated control of transporter endocytosis and recycling by the arrestin-related protein Rod1 and the ubiquitin ligase Rsp5. eLife. 3:03307. I Becuwe et al. (2012). A molecular switch on an arrestin-like protein relays glucose signaling to transporter endocytosis. J Cell Biol 196, 247-259. I