

Conference at Pasteur Institute (Sidi Fredj)

"Dissecting inflammation metabolism and cancer in mouse

models for human diseases"

By

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Manipulation of the mouse genome to genetically engineered mouse models (GEMMs) has provided powerful tools for understanding the cellular and molecular processes governing development and pathogenesis and to discover disease-relevant targetable pathways (1). Over the last decade, we explored the role of the AP-1 transcription factor complex, formed by dimers of Jun and Fos/Fra proteins, in homeostasis and disease using a collection of AP-1-dependent GEMMs generated by state-of-the-art transgenic and Embryonic Stem (ES)-cell technology. These include broad and tissue-specific loss-of-function mutants of AP-1 as well as Doxycycline (Dox)-switchable liver-specific gain-of-function AP-1 monomer and dimer mutant mice (2-4). Specific examples will be presented to illustrate how GEMMs can be generated and exploited to mechanistically dissect the cellular and molecular events controlling inflammation, metabolism and cancer, and how they can help identifying new prognostic biomarkers and therapeutic targets, in the context of a whole mammalian organism.

- 1. Bakiri L, Wagner EF, Mol Oncol, 2013
- 2. Bakiri L et al., Cell Cycle, 2014
- 3. Hasenfuss SC et al., Cell Metabolism. 2014
- 4. Bakiri L et al., J Exp Med. 2017



Dr. Latifa Bakiri graduated from the Ecole Normale Supérieure and the Sorbonne-Nouvelle and Pierre-et-Marie-Curie Universities in Paris, France. She trained in biochemistry, cell cycle and transcription factors biology in the laboratory of Pr. M. Yaniv at the Pasteur Institute, where she completed her PhD in 2000. Since then she joined Erwin Wagner's

team, first as a post-doc and then as a staff scientist. She has worked on the *in vivo* functions of different AP-1 members in development and disease with a particular focus on using new technologies for the generation of genetically engineered mouse models and their use to study bone and liver diseases, inflammation, metabolism and cancer.