



Postdoctoral Scientist in Cancer/Senescence Biology

Applications are invited for a postdoctoral research scientist to join the “Senescence Escape Mechanisms” lab at the Cancer Research Centre of Lyon (CRCL).

The research will be targeted towards deciphering and understanding new genetic events involved in the regulation of cellular senescence and their consequences on cancer and age-related diseases. The project will involve various technologies including si/shRNA experiments, senescence characterization, primary cell culture, retroviral infection, cell imaging, in vivo experiments in mice. The candidate must have experience in some of these technologies.

Candidates should have obtained their PhD recently in an area related to cellular biology, molecular biology and genetics. The recruited candidate is expected to start as early as possible.

Highly motivated applicants are invited to submit a full CV, detailed cover letter and contact information for three potential referees to Dr David Bernard at the following address: david.bernard@lyon.unicancer.fr

Recent relevant lab work:

.Griveau A, Devailly G, Eberst L, Navaratnam N, Le Calvé B, Ferrand M, Faull P, Augert A, Dante R, Vanacker JM, Vindrieux D, Bernard D. The PLA2R1-JAK2 pathway upregulates ERRA and its mitochondrial program to exert tumor-suppressive action. *Oncogene*, 2016;35:5033-42.

.Wiel C, Gras B, Vindrieux D, Warnier M, Gitenay D, Le Calvé B, Ferrand M, Augert A, Bernard D. Multidrug Resistance Protein 3 loss promotes tumor formation by inducing senescence escape. *Oncogene*, 2016;35:1596-1601.

.Le Calvé B, Griveau A, Vindrieux D, Maréchal R, Wiel C, Svrcek M, Gout J, Azzi L, Payen L, Cros J, de la Fouchardière C, Dubus P, Guitton J, Bartholin L, Bachet JB, Bernard D. Lysyl Oxidase family activity promotes resistance of pancreatic ductal adenocarcinoma to chemotherapy by limiting the intratumoral anticancer drug distribution. *Oncotarget*, 2016;7:32100-12.

.Ferrand M, Kirsh O, Griveau A, Vindrieux D, Martin N, Defossez PA and Bernard D. Screening of a kinase library reveals novel pro-senescence kinases and their common NF- κ B-dependent transcriptional program. *Aging (Albany NY)*. 2015 Nov;7(11):986-1003.

.Wiel C et al. Endoplasmic reticulum calcium release through ITPR2 channel leads to mitochondrial calcium accumulation and senescence. *Nature Communications*, 2014; 5:3792.

.Vindrieux D et al. PLA2R1 mediates tumor suppression by activating JAK2. *Cancer Res*, 2013; 73(20):6334-6345.

.Lallet-Daher H et al. Potassium channel KCNA1 modulates oncogene-induced senescence and transformation. *Cancer Res*, 2013; 73(16):5253-65.

.Humbert N et al. Regulation of ploidy and senescence by the AMPK-related kinase NUA1. *EMBO J*, 2010;29(2):376-86.

.Humbert N et al. A genetic screen identifies Topoisomerase 1 as a regulator of senescence. *Cancer Res*, 2009;69(10):4101-6.

.Augert A et al. The M-type receptor PLA2R regulates senescence through the p53 pathway. *EMBO Reports*, 2009;10(3):271-7.