Alejo Efeyan – Biosketch:

Alejo Efeyan has studied oncogenic pathways since his undergraduate Thesis work at the University of Buenos Aires. As a PhD student in the Manolo Serrano group, Alejo Efeyan studied how the tumor suppressor protein p53 exerts its cancer protection. By means of genetically engineered mice, his PhD worked unequivocally showed the dramatic importance of oncogenic signaling, compared to that of the DNA damage response. For his postdoctoral studies, Alejo moved to the laboratory of David M Sabatini at the Whitehead Institute. He focused to the study of a master regulator of cell growth, the mechanistic target of rapamycin (mTOR), involved in the pathogenesis of cancer and metabolic disorders. During his studies on nutrient sensing and signaling, Alejo decided to expand a field that was onlyCV approached from a biochemical and cell biological angle into physiology. His work showed: 1) the Rag GTPase / nutrient sensing pathway is essential for embryonic development and for adult life; 2) the regulation of RagA activity is key for coordinating fasting responses, for the regulation of autophagy, and for enduring the neonatal starvation period; 3) the Rag GTPases work as multi-node nutrient sensors, signaling both amino acid and glucose sufficiency to mTORC1, and 4) coordinated and dynamic control of mTORC1 by nutrient signaling is essential for B cell activation during the humoral response.

Alejo started his own laboratory at the CNIO in January 2016. His team studies the connections between nutrients and nutrient-evoked signaling, cell and organismal metabolism, aging, and cancer development, with a strong biochemical background supported by the use of novel genetically engineered mice.

Selected publications:

1. Oncogenic Rag GTPase signaling licenses B cell activation and drives follicular lymphoma development in mice. Ortega-Molina A, Deleyto-Seldas N, Carreras J, Sanz A, Menendez C, Marín-Arraiza L, Fernández-Ruiz B, Vandenberg A, Caleiras E, de Martino A, Troule K, Piñeiro-Yáñez E, Nakamura N, Araf S, Victora GD, Okosun J, Fitzgibbon J, **Efeyan A.** *(submitted)*.
2. Germinal center selection and affinity maturation require dynamic regulation of mTORC1. Ersching J*, **Efeyan A*** et al, *Immunity*. 2017 Jun 20;46(6):1045-1058 (*: equal contribution).
3. RagA, but not RagB, is essential for embryonic development and adult life. **Efeyan A** et al. *Dev. Cell*. 2014 May 12;29(3):321-9.
4. Regulation of mTORC1 by the Rag GTPases is necessary for neonatal autophagy and survival. **Efeyan A** et al. *Nature*. 2013 Jan 31;493(7434):679-83
5. Induction of p53-dependent senescence by the MDM2 antagonist nutlin-3a in mouse cells of fibroblast origin. **Efeyan A** et al. *Cancer Res*. 2007 Aug 1;67(15):7350-7.
6. Policing of oncogene activity by p53. **Efeyan A** et al. *Nature*. 2006 Sep 14;443(7108):159.
7. Genetic dissection of the role of p21Cip1/Waf1 in p53-mediated tumour suppression. **Efeyan A** et al. *Oncogene*. 2007 Mar 8;26(11):1645-9.
8. Establishment of two hormone responsive mouse mammary carcinoma cell lines derived from a metastatic mammary tumor line. **Efeyan A** et al. *Breast Cancer Res Treat*. 2004 Feb;83(3):233-44.

9. Nutrient-sensing mechanisms and pathways. **Efeyan A**, Comb WC, Sabatini DM. *Nature*. 2015 Jan 15; 517(7534):302-310.
10. mTOR: from growth signal integration to cancer, diabetes and ageing. Zoncu R*, **Efeyan A***$, Sabatini DM$. *Nat Rev Mol Cell Biol*. 2011 Jan;12(1):21-35. (*: equal contribution; $: corresponding authors)
11. mTOR and cancer: many loops in one pathway. **Efeyan A**, Sabatini DM. *Curr Opin Cell Biol*. 2010 Apr;22(2):169-76.