

Short CV of Prof. Max Gutierrez

Max is a cell biologist originally from Mendoza, Argentina. In 2005, he obtained a PhD in cell biology from the University of San Luis, Argentina. During his PhD work, he discovered a novel innate immune pathway, later named “Xenophagy”. In 2006, he moved to EMBL in Heidelberg, Germany as a postdoc in Gareth Griffiths Laboratory, first as a fellow of the Alexander von Humboldt Foundation and then as an EMBO fellow. His work in Heidelberg focused on the cell biology and imaging of macrophages; it was also in Heidelberg that he fell in love with Electron Microscopy.

In 2009, he started his independent research group at the Helmholtz Centre for Infection Research in Braunschweig, Germany as head of the Junior Research Group 'Phagosome Biology'. In 2012, he was recruited as a Programme Leader Track at the Medical Research Council's National Institute for Medical Research, which became part of the Francis Crick Institute in 2015. Since 2018, he is a Senior Group Leader at the Francis Crick Institute.

As a cell biologist trained in microbiology, how intracellular pathogens evolved strategies to survive within host cells has always fascinated me and clearly shaped my scientific career. My long-standing interest is the cellular mechanisms that regulate the interactions between *Mycobacterium tuberculosis* and host cells. I aim to better understand the host cell factors that contribute to *M. tuberculosis* control as well as the *M. tuberculosis* factors that this pathogen uses to hijack host cells. To this end, my lab is developing a variety of cutting-edge imaging technologies in high containment combined with various model systems and approaches at the single cell level.

Host cell environments and antibiotic efficacy in tuberculosis

To cause disease and disseminate to other hosts, *M. tuberculosis* needs to replicate within human cells. Work in the last decades has shed light into some aspects of tuberculosis pathogenesis, however, we still do not understand how *M. tuberculosis* manages to survive within eukaryotic cells and why some cells are able to eradicate this lethal pathogen. This surprising gap in knowledge is in part due to the lack of appropriate imaging technologies that have precluded comprehensive understanding of the fundamental biology that underpins *M. tuberculosis*-host cell interactions. Our research focuses on the fundamental molecular and cellular mechanisms that regulate the interactions between *M. tuberculosis* and host cells. We aim to dissect the host cell factors that contribute to *M. tuberculosis* control as well as the *M. tuberculosis* factors that this pathogen uses to hijack host cells. To this end, we use a variety of cutting-edge imaging approaches and model systems. In this seminar, I will present some recent data from our group regarding the environments where *M. tuberculosis* survives in human cells and the barriers that these environments represent for therapy.