As a Medical Doctor and resident in Internal Medicine, I started my scientific career in a clinical environment mainly focused on viral hepatitis research. During my undergraduate studies and the Internal Medicine Residency, I started developing a strong interest towards basic molecular mechanisms of disease, and inflammation in particular. As a post-doctoral fellow at UCSD, I started working on signal transduction by transcription factors that control expression of inflammatory genes. I started my research activity as independent investigator at the Institute for Research in Biomedicine in Switzerland in 2000, where I re-focused my attention and main research topic from signaling to the mechanisms linking chromatin organization to the control of inflammatory gene expression in cells of the innate immune system. Insofar, the most important contribution to knowledge of my laboratory is the understanding of how the regulatory information contained in mammalian genomes controls in a cell type- and stimulus-specific manner the deployment of the inflammatory gene expression program. These data allowed us to nail down concepts that were extensively validated and are now commonly accepted, particularly the notion that transcription factors that control differentiation of innate immune cells determine where transcription factors activated in response to inflammatory stimulation land in the genome, thus laying the ground for cell type-specific inducible gene expression. A second, more recent research endeavor in my lab relates to the molecular understanding of cellular heterogeneity in human pancreatic cancer, which underlies the incurability of this highly lethal cancer predicted to become the second most common cause of cancer deaths in the Western World by 2030.