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GUEST EDITOR

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Dr AJ Capobianco

AJ Capobianco is an associate professor of Cellular and Molecular Oncogenesis at the Wistar Institute and is a Wistar associate professor of Biochemistry and Biophysics at the University of Pennsylvania School of Medicine. Dr Capobianco has 20 years experience researching the biochemistry and genetics of oncogenes.

Dr Capobianco's research interest has long been focused on the molecular mechanisms that drive tumorigenesis. In the fall of 1988, Dr Capobianco joined the lab of Dr Thomas Gilmore at Boston University as a first year graduate student to understand the function of the retroviral oncogene *v-rel* and how it is related to the cellular homologue c-rel. Dr Capobianco had several key papers while a graduate student that contributed significantly to our understanding of Rel/nuclear factor-κB function and its role in cellular transformation.

After receiving a PhD from Boston University in 1993, Dr Capobianco joined the laboratory of Dr J Michael Bishop at UCSF. While in the lab, Dr Capobianco became interested in a relatively unknown protein called Notch, named in 1917 for its 'notched' wing phenotype in *Drosophila*. At the time, nearly all studies on notch were performed in *Drosophila* and it was thought that notch functioned as a factor involved in cell fate determination. Dr Capobianco, based on evidence in the literature, thought that Notch should act as an oncogene. So with his experience in molecular oncology, Dr Capobianco set out to prove this hypothesis. His research established that Notch is capable of transforming cells in culture, and thus provided direct evidence that Notch functions as an oncogene.

In 1997, Dr Capobianco joined the faculty of the University of Cincinnati College of Medicine as an assistant professor where he focused his laboratory on the elucidation of the Notch signaling pathway and how deregulation of this pathway leads to neoplasia. In 2003, Dr Capobianco moved to the Wistar Institute as an associate professor and a Wistar associate professor of Biochemistry and Biophysics at the University of Pennsylvania School of Medicine.

His research has had a major impact on our understanding of the Notch pathway and its role in tumorigenesis. His lab was first to demonstrate that Notch regulates the cell cycle and that this is critical for cellular transformation. His lab established the first mouse model for Notch-induced tumorigenesis. Using this model he demonstrated that Notch critically controls the levels of p53 and that Ikaros functions as a tumor suppressor for the Notch pathway. His lab demonstrated that DSL (Delta-Serrate-lag) proteins, which function as Notch ligands, can signal through a PDZ-dependent mechanism and he proposed a bidirectional-signaling model for the DSL/Notch pathway. Now his interests include exploiting the labs knowledge of Notch signaling to identify small molecule inhibitors of the Notch pathway.