Paolo M. Comoglio MD, Prof
Curriculum vitae

Academic and Research Career

1963-1969	Doctor in Medicine (MD), University of Torino School of Medicine, IT
1970-1972	Postdoctoral Research Fellow, Washington University, St. Louis, USA
1973	EMBO Fellow, University of Pennsylvania, Philadelphia, USA
1974-1980	Associate Professor of Histology, University of Trieste, IT
1980-1983	Full Professor (as above).
1984-2015	Full Professor of Histology, University of Torino School of Medicine, IT
1996-2008	Director, Division of Molecular Oncology, Candiolo Cancer Institute, IT
2000–2017	Scientific Director, Candiolo Cancer Institute IRCCS, IT
Present	Dean, Candiolo Cancer Institute; Director of Molecular Therapeutics and Exploratory Research Division, IT
2016-	Co-Founder and Scientific Advisor of OCTIMET Oncology NV, Geel, BE
2017-	Co-Founder and President of METIS Precision Medicine SB, Torino, IT

Paolo Comoglio has a long and distinguished record in the field of research on tyrosine kinase receptors and related oncogenes (*Comoglio et al, Nature Reviews Cancer, 18:341-358, 2018*). He developed the first anti-phosphotyrosine antibody (*Comoglio et al, The EMBO Journal 3:483-489, 1984*); through this tool he identified the protein tyrosine kinase encoded by the rearranged BCR-ABL oncogene, responsible for the onset of Chronic Myeloid Leukemia (*Naldini et al, Molecular and Cellular Biology 6: 1803-1811, 1986*) and discovered the protein tyrosine kinase receptor for Hepatocyte Growth Factor (HGF) encoded by the MET oncogene (*Giordano et al, Nature 339: 155-156, 1989; Naldini et al, Oncogene 6: 501-504, 1991*). He identified and elucidated the functions of two other kinase receptors, encoded by genes structurally related to MET: RON (*Gaudino et al, The EMBO Journal 13: 3524-3532, 1994*), and a homologous kinase-dead receptor, ROR (*Gentile et al, Cancer Research 71: 3132-3141, 2011*). Paolo Comoglio expounded and introduced a number of insights that are now largely accepted and widespread, notably the concept of 'invasive growth', a genetic program otherwise 'physiological' but 'usurped' by cancer cells to progress toward metastasis (*Trusolino and Comoglio, Nature Reviews Cancer 2: 289-300, 2002*). Among others, two advances are worthy of mention. First, Comoglio's group was able to show that the well-known but poorly understood role of hypoxia in promoting tumor invasiveness is mediated by activation of MET (*Pennacchietti et al, Cancer Cell 3: 347-361, 2003*). Second, malignant transformation driven by the MET oncogene, is associated with venous thrombosis via over-expression of the PAI-1 and COX-2 genes. This provides a long-sought link between cancer and thrombo-embolism (Trousseau's sign or para-neoplastic thrombosis. *Boccaccio et al, Nature 434: 396-400, 2005*). He recently discovered that MET inhibition overcomes radiation resistance of glioblastoma stem cells (*De Bacco et al, EMBO Molecular Medicine 8: 550-568, 2016*) and that withdrawal of therapeutic tyrosine kinase inhibitors accelerates disease progression by unleashing a 'rebound' effect (the 'flare effect'. *Pupo et al, Cancer Research 76:5019-5029, 2016*).

Link to list of publications: https://www.ncbi.nlm.nih.gov/pubmed/?term=Comoglio+P*