Professor Dr Gerry Melino

University of Rome Tor Vergata, Italy; Oxford University, London, UK

Professor Gerry Melino currently works as Department Director and Professor of Molecular Biology at the University of Rome "Tor Vergata" in Italy (Email: melino@uniroma2.it). He is also Programme Leader for the Medical Research Council (MRC) Toxicology Unit, in Leicester, UK His training originated in Italy and in particular Rome, where he obtained his M.D. (1978, University of Rome) followed by clinical specialisations in Paediatrics (1981, University of Rome) and Clinical Oncology (1985, University of Rome). He obtained his Ph.D in 1984 at the University of London in the Chemical Pathology Department, Charing Cross & Westminster Medical School. Upon graduation, he worked as Research Fellow, then Lecturer and later Senior Lecturer (Honorary Consultant) until 1987. Professor Melino returned to Italy in 1988 to the University of Rome as a Lecturer before becoming a full Professor in 1994. Professor Melino has acted as Consultant and Scientific Advisor for several companies and government institutions. He also has significant Editorial activity as Founder and Editor-in-Chief of the Nature-Publishing-Group journals Cell Death and Disease (www.nature.com/cddis) and Cell Death and Differentiation (Impact Factor 9.050. www.nature.com/cdd) as well as serving on the editorial boards of several other scientific journals. The Scientific Interests of Professor Melino are focused on Programmed Cell Death or apoptosis, in neural and epidermal models. Originally, he worked on the Molecular mechanisms of cell death in the skin, a process known as cornification or formation of the cornified envelope. The molecular events were investigated in vitro and in animal models as well as in human genetic pathologies. The role of transglutaminases (type 1, 2, 3, and 5) and their substrates (SPRs, loricrin, keratins) were investigated at biochemical and genetic levels. While still keeping an interest on these aspects, his current work is focused on The p53/p63/p73 family. DNA damage elicit repair mechanisms involving the tumour suppressor gene p53 and the two newer members of the same family: p63 and p73. The molecular events driven by DNA damage to elicit the function of p63/p73 and their transcriptional regulation, is investigated in vitro. The molecular mechanisms of apoptosis and their protein stability and degradation is also under investigation. Transgenic mice and knock-out work for p63 or p73 is in progress.