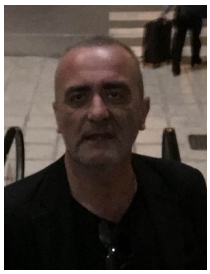
Georgios T. Stathopoulos MD PhD



Georgios is a Research Group Leader at the Comprehensive Pneumology Center of the Helmholtz Zentrum Munich and an Associate Professor of Physiology at the Faculty of Medicine of the University of Patras, Greece. He obtained his MD in Patras (1989-1995), and his Pneumology board certification (1997-2002) and PhD (2003-2007), in Athens. He worked as clinical consultant to the Greek National Health System (2006-2009) and as Research Fellow (2003-2004) and Assistant Professor (2009-2010) at Vanderbilt University, Nashville, TN. In 2011 he was appointed to his Patras position where he founded the Laboratory for Molecular Respiratory Carcinogenesis and in 2015 to Munich where he initiated the group of Lung Carcinogenesis.

Georgios' collaborators are looking into the pathobiology of lung and pleural malignancies, including early carcinogenesis and late dissemination. They are using fate models to dig into chest tumor origins and modular cell-animal systems to investigate oncogene-immunity interactions. They top this by teaching respiratory physiology and research to medical and graduate students.

Georgios and co-workers have published their findings in important biomedical journals including the Journal of Clinical Investigation, Nature Communications, and EMBO Molecular Medicine, among others. They garnered European Respiratory Society Maurizio Vignola (2009) and Romain Pauwels (2013) Awards, European Research Council Starting (2010) and Proof-of-Concept (2015) Grants, and Research Scholarships from the European Respiratory Society, the Hellenic Thoracic Society, the Hellenic Scholarship Foundation, and the Hellenic Society for Molecular Cancer Research (2006-2014).

Pulmonary repair and mutagenesis in response to tobacco carcinogens

Lung adenocarcinoma is the number one cancer killer in the world and is mainly caused by environmental tobacco and radon exposures. Despite significant efforts, the respiratory epithelial cells that suffer mutations and provide the cellular source of lubg adenocarcinoma remain unknown. In addition, the mode of mutation acuisition of these cells over time has not been explored prospectively. We have developed mouse models to mark respiratory epithelial lineages and to follow their fate and genomic lesions after single carcinogen exposure. We have also developed unique cell lines derived from carcinogen-induced lung adenocarcinomas that feature an amazing mutation spectrum. These models have allowed us to longitudinally study how the airway and alveolar epithelia are repaired upon carcinogenic insults, how transcription factors and tumor suppressors determine the survival or death of mutated epithelial cells of the lungs, and how point mutations and copy number alterations accumulate over time in developing lung tumors. The lessons learnt from mice, we translate to our human cohort of 377 loco-regional lung adenocarcinomas, which is carefully phenotyped,and is currently being genomically analyzed. The ultimate aim of these ongoing studies is to identify addiction partners of driver oncogenes and to target them for lung adenocarcinoma prevention and therapy.