

11th SWISS BRIDGE AWARD: Honouring three outstanding cancer researchers

Summaries of the three supported research projects

Andrea Alimonti, M.D., Laboratory of Experimental Oncology, Oncology Institute of Southern Switzerland (IOSI), Bellinzona, receives 250,000 Swiss francs for the project entitled:

Manipulation of senescence pathways for cancer therapy: from experimental models to clinic

Various studies demonstrate the relevance of cellular biological aging in restricting the development of new tumour cells and thus open up new potential for opportunities in cancer treatment. Andrea Alimonti and his team examine a certain form of the biological aging known as PICS (PTEN loss induced cellular senescence): The ability to induce biological aging in cells by targeting PTEN signalling (via deactivation of the tumour suppressor PTEN), without a requirement for hyper replication and DNA damage, opens up the possibility of targeting quiescent cells including quiescent cancer initiating cells. This offers a new therapeutic approach especially to target prostate cancer cells. If it is possible to activate PICS cellular senescence through drugs and bioactive compounds in prostate cells, then dormant cancer cells could also be inactivated. Alimonti's research team has tested various compounds in the laboratory in order to find out if and to what degree PICS cell aging can be activated. Such a compound could eventually lead to a new anticancer drug to treat prostate cancer.

Ronit Satchi-Fainaro, Ph.D., Department of Physiology and Pharmacology, Sackler School of Medicine, Tel Aviv University, Israel, receives 125 000 Swiss francs for the project entitled:

Deciphering the molecular mechanism of tumour dormancy using bone-targeted polymer therapeutics

Before tumours spread, they remain dormant and microscopic and do not expand over prolonged periods of time. Once a tumour is fed with nutrients and oxygen by newly built blood vessels, it can grow and develop metastases. The ability of a tumour to progress from a dormant to a fast-growing state is central to the progression of cancer and is termed the «angiogenic switch». Not much is known on the inactive phase of tumours and this angiogenic switch. Ronit Satchi-Fainaro and her team investigate the molecular and genetic changes that trigger inactive tumours via the angiogenic switch to become growing and spreading tumours. Satchi-Fainaro's research group has tested various anticancer or anti-angiogenic used in the treatment of bone tumours and bone metastases. The researchers will evaluate the ability of these compounds to delay the angiogenic switch to keep dormant tumours inactive for longer periods of time or, alternatively, to regress fast-growing angiogenic tumours to a dormant state.

Anna Sablina, Ph.D., VIB Department of Molecular and Developmental Genetics, University of Leuven, Belgium, receives 125,000 Swiss francs for the project entitled:

The role of the RalA signalling pathway in human cancer

The RAS gene family plays an essential role in the formation of many tumours. Mutations in these genes lead to the activation of cell growth and cell division, leading to cancer cell development. Oncogenic mutations of RAS family members have been identified in up to 30% of human cancers. Many cancer therapies work insufficiently or not at all for patients with RAS-mutated tumours. Thus, there is pressing clinical need for new therapies specifically for patients with RAS-mutated tumours and this is the motivation for Anna Sablina and her research team. They want to find out how the enzyme RalA GTPase influences cancer development as a result of an RAS-mediated tumorigenesis. Compounds that activate or switch off the RalA GTPase are being sought after in the laboratory. With their work, the researchers hope they can identify novel molecular targets for cancer therapies effective in the treatment of patients with RAS-mutated tumours.