SWISS BRIDGE AWARD 2009 - WINNERS

SUMMARIES OF THE THREE SUPPORTED RESEARCH PROJECTS

Prof. Matthias Egger, MD, Head, Institute of Social and Preventive Medicine (ISPM) at the University of Berne, receives 200 000 Swiss francs for the project: **AIDS-defining Cancers in Southern Africa in the Era of ART.** In Southern Africa, where HIV infection is common, AIDS-defining cancers are an important public health issue. With the scale-up of combination antiretroviral therapy (ART) in Africa the prognosis of individuals living with HIV/AIDS has been improved substantially, with mortality rates approaching those of the general population. The implications for the burden of cancer in the HIV-infected population, and for treatment and outcome of AIDS-defining cancers like Kaposi sarcoma, non-Hodgkin's lymphoma and cervical cancer are unclear at present. Professor Matthias Egger of the Institute of Social and Preventive Medicine at the University of Berne aims to bridge these gaps. In his research projects in the area of epidemiology, health services and clinic, the prevention and treatment of AIDS-defining cancers in the region are being studied. The aim is to evaluate the incidence, the clinical stages at diagnosis and the prognosis, to examine the provision of health care to these patients and build relevant capacity. The International Epidemiological Databases to Evaluate AIDS in Southern Africa (IeDEA-SA), a large network of HIV programmes including data on ART patients in Botswana, Malawi, Mozambique, South Africa, Zambia and Zimbabwe, will serve as the research platform.

Prof. Wilhelm Krek, PhD, Institute of Cell Biology, Swiss Federal Institute of Technology Zurich, receives 150 000 Swiss francs for the project: **The impact of the VHL tumour suppressor protein on aneuploidy and cancer.** Von Hippel Lindau (VHL) cancer syndrome, a rare hereditary disease, is caused by inactivating germline mutations in the VHL tumour suppressor gene and is associated with an increased risk of developing a variety of tumours, including clear cell renal cell carcinoma. How the VHL protein exerts its tumour suppression function in healthy cells remains incompletely understood. Recent work in Professor Wilhelm Krek's laboratory at the Institute of Cell Biology of the Swiss Federal Institute of Technology in Zurich revealed a previously unrecognized function of the VHL protein in suppression of chromosomal instability. If this protein is functionally inactivated, the cell produces reduced levels of Mad2, an important regulatory protein which is responsible for the correct distribution of the chromosomes during cell division. This results in aneuploidy, i.e. an absence or an excess of chromosomes. In his research project, Professor Krek will examine the functions of the VHL and Mad2 proteins. The results derived from these studies will significantly advance the knowledge of the development of the Von Hippel Lindau syndrome and of sporadic renal cell carcinoma and perhaps will also help define fundamental mechanisms of chromosomal stability maintenance and the role of aneuploidy as an instigator of tumourgenesis.

Prof. Stephen C. West, PhD, Senior Group Leader & Deputy Director, Clare Hall Laboratories, London Research Institute, Cancer Research UK, South Mimms, UK, receives 150 000 Swiss francs for the project: **Interplay between the cancer pre-disposition disorders Fanconi Anemia, Bloom's Syndrome and BRCA2 breast cancer.** Our genetic material, the DNA, is continually subjected to damage, either from endogenous sources such as reactive oxygen species produced within the cell, or by agents in the environment such as ionising radiation or carcinogenic chemicals. Normally, cells employ elaborate and effective repair processes that are specialised to recognise different types of lesions in DNA. Some individuals, however, are genetically predisposed to crippling diseases or cancers that are the direct result of mutations in genes involved in the DNA damage response like Fanconi Anemia (bone marrow failure), Bloom's Syndrome (chromosomal breaks) and breast cancers caused by mutation of BRCA2.

Professor Stephen C. West and his research team at the London Research Institute of the charity Cancer Research UK have been working at the forefront of basic biological research in the area of DNA repair. The focus of this project is to determine the molecular interplay of the gene products responsible for the instability of the genetic material in these three inheritable cancer predisposition syndromes. These findings will allow the researchers to determine precisely how the BRCA2 tumour suppressor protein contributes to DNA repair.