Report from active participation in 1st EMBO meeting on "Cellular signaling and cancer therapy", 23-27th May 2014, Cavtat, Croatia – Malgorzata Bajor

The 1st EMBO meeting on „Cellular signaling and cancer therapy" in Cavtat marks the start of a new series of meetings focusing on cancer signaling and, most importantly, translational aspects. The detailed knowledge of cancer signaling pathways gained so far - particularly from high throughput genomic and proteomic techniques - unravels the complexity of processes controlling cancer initiation and progression. However, despite certain advances in cancer therapy, the translational impact of this enormous wealth of knowledge mostly stayed behind the high-flying expectations of the post-genomic era. Today, the challenges for future anticancer treatments are evident, and need to be met by scientific breakthroughs. In bridging the so-called innovation gap, pharma industry more and more relies on academia. Therefore, the goal of this meeting was to provide a platform for sharing newest results in cancer signaling, and bring together leading experts from all over the world. I had a pleasure to present our data during poster session "Oncogenic signaling networks II". Professor Manolis Pasparakis from University of Cologne Institute for Genetics in Germany was moderating this session.

Title of the poster: **Evaluation of adenanthin as an intracellular signaling modulator and potential therapeutic agent in estrogen receptor positive breast cancer**
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Dr Bajor at the poster during poster session

Breast cancer is a heterogeneous disease driven by a continuum of mutations and abnormal gene/protein expression. Roughly 70% of breast cancers express estrogen receptor ERα, which is a main driver of tumorigenicity in a majority of ER-positive cases. Understanding of the processes underlying the ER alpha regulation is a key to find better therapeutic approaches to this disease. Burgeoning evidence indicates that in tumors, oxidative stress is involved in the progression of ER-

positive breast cancer. The increase in cellular oxidants, such as reactive oxygen species (ROS), above critical levels contribute to uncontrolled proliferation and genomic instability, which in turn can facilitate progressive transformation of normal cells into cancer cells. On the other hand, an acute exaggeration of oxidative stress in cancer cells is a mechanism of action of numerous antitumor modalities, including radiotherapy and a range of chemotherapeutic agents. Therefore, it is crucial to evaluate newly identified inducers of oxidative stress as either potential antitumor agents or tumorigenicity promoters in breast cancer.

Adenanthin is a recently described inhibitor of peroxiredoxin (PRDX)-1 and -2, both endogenous cellular scavengers of hydrogen peroxide. Recent results from our group strongly suggest that PRDX-1 acts as a potent protector of ERα presence and function in breast cancer cells subjected to oxidative stress. Therefore, in our study, we intended to assess the actions of adenanthin in *in vitro* models of ER-positive breast cancers.

In summary, our data show that adenanthin can act as an inducer of excess oxidative stress in breast cancer, which results in potent modulation of intracellular signaling, especially in the context of estrogen receptor functions. This potential pro-tumorigenic role of adenanthin must be elucidated, especially in light of adenanthin being considered as a therapy in other cancer types.