BASTION
FROM BASIC TO TRANSLATIONAL RESEARCH IN ONCOLOGY

Capacities/Research Potential
FP7-REGPOT-2012-2013-1

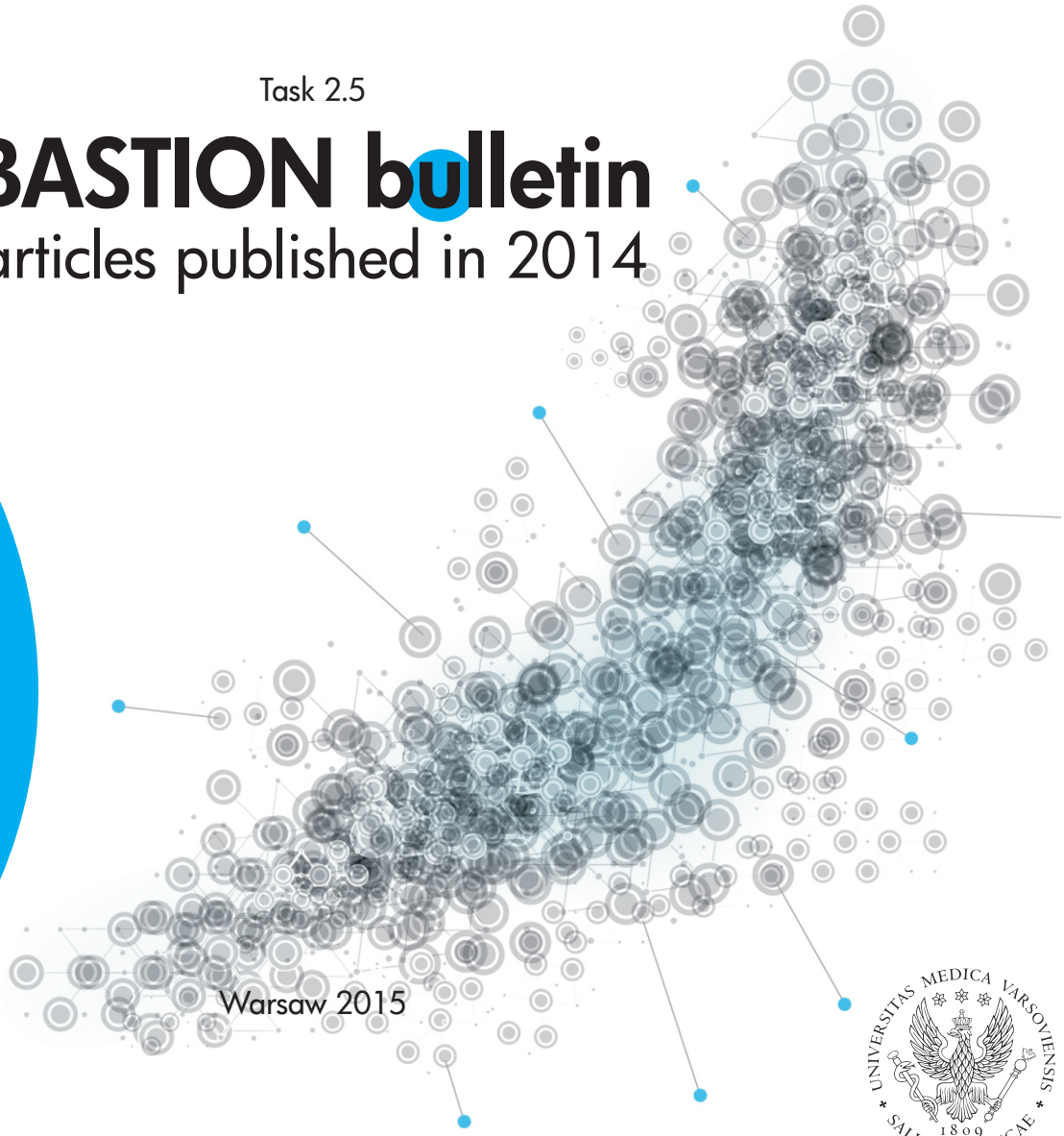
Project No. 316254

BASTION

"From Basic to Translational Research in Oncology"

Task 2.5

BASTION bulletin
articles published in 2014



Warsaw 2015

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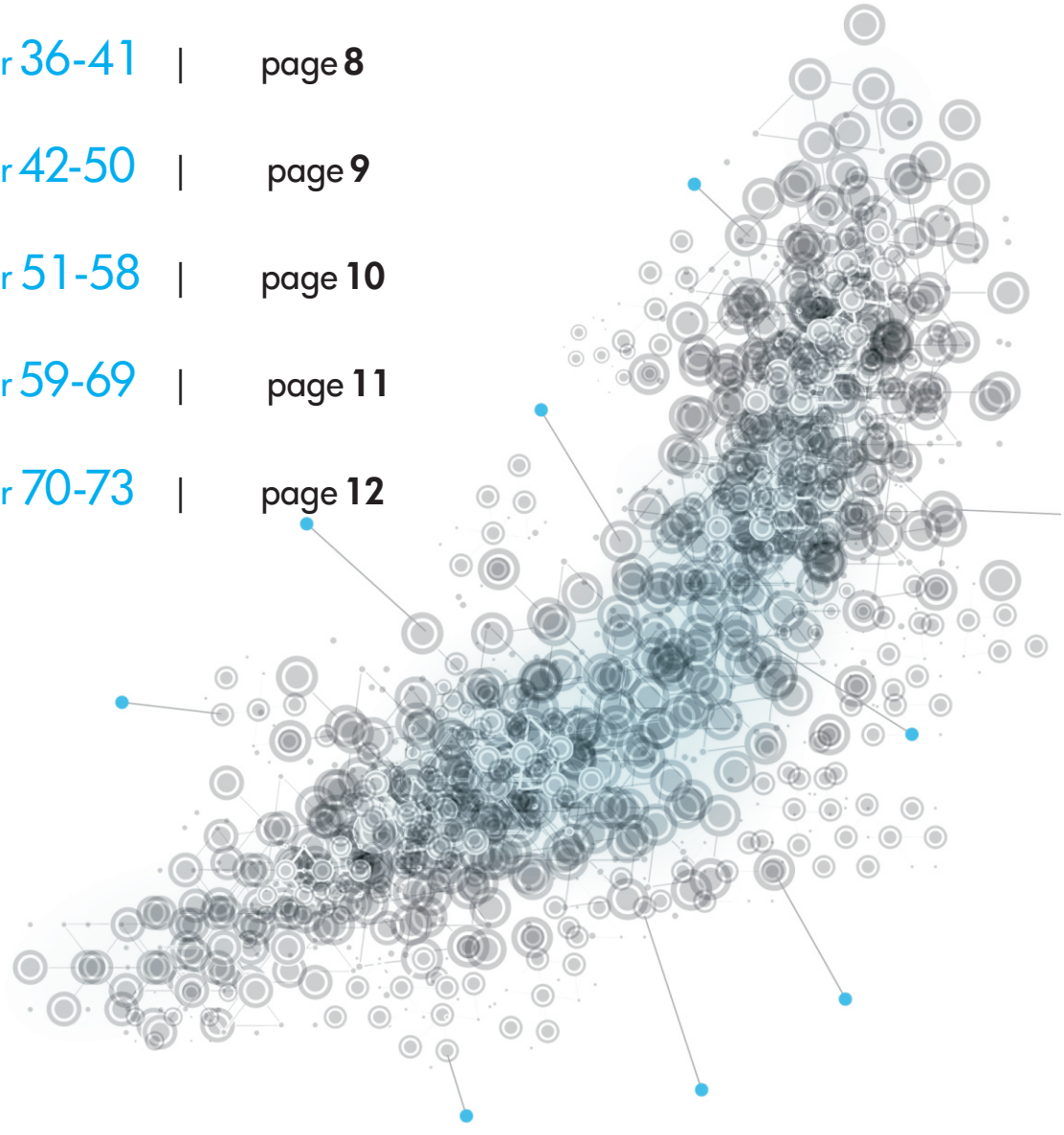
Publications:



Publications

1. Klimczak D, Pączek L, Jażdżewski K, Kuch M. MicroRNAs: powerful regulators and potential diagnostic tools in cardiovascular disease. Kardiol Pol. 2014 Nov 5.

2. Wojcicka A, Swierniak M, Kornasiewicz O, Gierlikowski W, Maciag M, Kolanowska M, Kotlarek M, Gornicka B, Koperski L, Niewinski G, Krawczyk M, Jazdzewski K. Next generation sequencing reveals microRNA isoforms in liver cirrhosis and hepatocellular carcinoma. Int J Biochem Cell Biol. 2014 Aug;53:208-17.

Liver malignancies, including hepatocellular carcinoma (HCC) are the third cause of death from cancer worldwide. Tumorigenic changes in hepatic cells potentially result from aberrant expression of microRNAs. We employed next-generation sequencing to comprehensively analyze the sequence and expression of microRNAs in HCC tumors and in cirrhotic liver.
The study showed that hepatic tissue exhibits expression of 374 microRNAs, and levels of 64 miRs are aberrant in HCC. Moreover, many miRs are expressed in numerous length variants what leads to severe changes in their regulatory role. The study gives a solid basis for the future work on the microRNA-mediated regulation of gene expression in liver. Tissue-specific microRNA profiles can be further used for elaboration of molecular tools for diagnostics and therapies of liver cancer.

3. Wojcicka A, Czetwertynska M, Swierniak M, Długosinska J, Maciag M, Czajka A, Dymecka K, Kubiak A, Kot A, Płoski R, de la Chapelle A, Jażdżewski K. Variants in the ATM-CHEK2-BRCA1 axis determine genetic predisposition and clinical presentation of papillary thyroid carcinoma. Genes Chromosomes Cancer. 2014 Jun;53(6):516-23.

In a search for genetic variations predisposing to papillary thyroid carcinoma (PTC)we conducted an association study in 1781 patients and 2081 healthy controls, genotyping polymorphisms in DNA repair genes: ATM, CHEK2 and BRCA1. The study showed that the rare variant of CHEK2 causes over 2.2-fold increased risk of developing thyroid cancer. We also showed important association of the SNPs with clinical outcome: in males, the rare variant of BRCA1 was associated with increased frequency of nodular metastases and higher stage of cancer. In contrast, rare variant of ATM alleviated the severity of the disease.Ourfindingsimplicate the role of ATM-CHEK2- BRCA1axis in modification of geneticpredisposition to PTC and itsclinicaloutcome, potentially contributing to elaboration of novel diagnostic and prognostic tools for thyroid cancer.

4. Wojcicka A, Piekielko-Witkowska A, Kedzierska H, Rybicka B, Poplawski P, Boguslawska J, Master A, Nauman A. Epigenetic regulation of thyroid hormone receptor beta in renal cancer. PLoS One. 2014 May 21;9(5):e97624.

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Clinical trials with SRC family kinase (SFK) inhibitors used alone or in a combination with anti-CD20 monoclonal antibodies (mAbs) are currently underway in the treatment of B-cell tumors. However, molecular interactions between these therapeutics have not been studied so far. We observed that SFK inhibitors strongly affect CD20 expression at the transcriptional level, leading to inhibition of anti-CD20 mAbs binding and increased resistance of tumor cells to complement- and antibody-dependent cellular cytotoxicity. The results of our studies indicate that development of optimal combinations of novel treatment modalities with anti-CD20 mAbs should be preceded by detailed preclinical evaluation of their effects on target cells.

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This study demonstrates that B-cell receptor inhibitors strongly down-regulate CD20 expression in tumor cells, leading to decreased binding of anti-CD20 mAbs to the surface of tumor cells and impairment of complement-dependent cytotoxicity (CDC) as well as antibody-dependent cell-mediated cytotoxicity (ADCC) mechanisms that mediate antitumor effects of anti-CD20 mAbs in vivo. Our observations strongly imply that before investigating novel therapeutic combinations in cancer patients, extensive pre-clinical studies should be carried out to evaluate possible interactions between drugs at the molecular level.

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Breast cancer is a heterogeneous disease, and its various forms differ markedly regarding the progression rate and responsiveness to the treatment. Increased oxidative stress conditions within the mammary tumor have recentlybeen shown to correlate with more aggressive breast cancer subtypes. It is therefore important to elucidate how different pro- and antioxidant factors can influence the behavior of breast cancer. This paper identifies a particular role for one of the most prominent antioxidant enzymes, peroxiredoxin 1 (PRDX1), in human breast cancer. Our studies, analyzing combined data from more than 1200 breast cancer patients imply that PRDX1 protects breast cancer cells to retain a less aggressive, estrogen-receptor driven, form. We further corroborate this assumption by a set of in vitro experiments with established breast cancer cell lines. In summary, our results suggest that under conditions of oxidative stress PRDX1 can act as a tumor suppressor and biomarker of favorable outcome in this breast cancer. Further studies are needed to identify potential applications of this knowledge in breast cancer treatment.

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Tyrosine kinase inhibitors (TKIs) have profoundly changed therapy of chronic myeloid leukemia (CML) and transformed this disease into truly chronic ailment for more than a half of CML patients. Unfortunately, success of TKI is shadowed by the development of resistance to therapy in a significant number of patients. As shown in this study, statins increased intracellular concentration of imatinib in primary CML cells and cell lines and enhanced antileukemic activity of imatinib. Statin-induced inhibition of membrane efflux transporters, ABCB1 and ABCG2, was responsible for these effects. Importantly, no cumulative cytotoxic effects of such combination were observed in normal CD34+ cells. This work presents a potential and feasible approach to overcome drug resistance to imatinib in selected group of CML patients and provides a rationale for a controlled, prospective clinical trial.

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51. Wachowska M, Gabrysiak M, Muchowicz A, Bednarek W, Barankiewicz J, Rygiel T, Boon L, Mroz P, Hamblin MR, Golab J. 5-Aza-2'-deoxycytidine potentiates antitumour immune response induced by photodynamic therapy. Eur J Cancer. 2014 May;50(7):1370-81.

Photodynamic therapy (PDT) has been shown to induce strong immunity against tumor cells expressing exogenous tumor-associated antigens (TAAs). Cancer cells can evade the immune system by epigenetic silencing of TAAs, while DNA methyltransferase inhibitors, such as 5-aza-2'-deoxycytidine (5-aza-dC) can restore the expression of silenced or down-regulated TAA. In the article we demonstrate that epigenetic remodeling with 5-aza-dC combined with PDT can elicit robust and durable antitumor immunity in 4 different tumor models in experimental mice. Taken together, these findings show that PDT leads to strong specific antitumor immune responses, and that epigenetic modification of tumor antigens levels may be a novel approach to further enhance the effectiveness of PDT. The present results provide a strong rationale for clinical development of this therapeutic approach.

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