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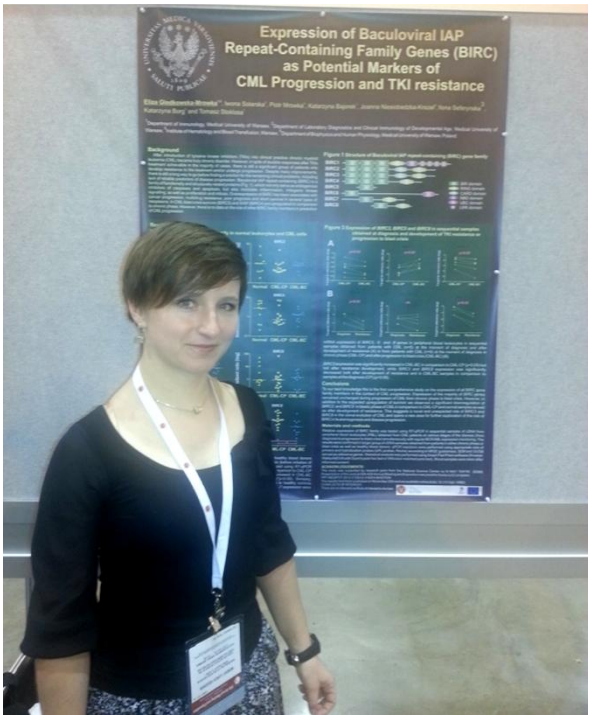
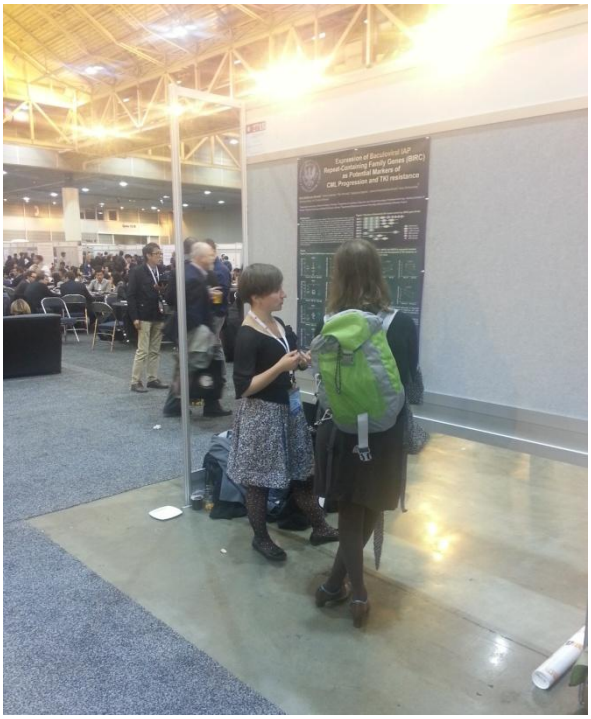
Report from active participation in 55th American Society of Hematology Annual Meeting and Exposition, New Orleans, LO, USA, December 6-10, 2013 - Eliza Glodkowska-Mrowka

American Society of Hematology Annual Meeting and Exposition is held annually in early December. It is the major scientific meeting in the field of hematology covering novel discoveries and achievements in both therapy and research. This year's 55th ASH Meeting and Exposition in New Orleans gathered more than 20 thousands of clinicians, scientists and corporate partners from all over the world. I had an opportunity to present the results of our research project during poster session *Chronic Myeloid Leukemia: Biology and Pathophysiology, excluding Therapy*. I had a unique possibility to present my data to broad audience and discuss my results with many experts in my field of research.





Abstract/poster title: Differential Expression of BIRC Family Genes In The Course Of Chronic Myeloid Leukemia – BIRC3 and BIRC8 As Potential New Candidates To Identify Disease Progression

Authors: Eliza Glodkowska-Mrowka (presenting author), Iwona Solarska, Piotr Mrowka, Katarzyna Bajorek, Joanna Niesiobedzka-Krezel, Ilona Seferynska, Katarzyna Borg, and Tomasz Stoklosa

The presented study focused on BIRC (baculoviral IAP repeat-containing) family of genes that includes eight functionally- and structurally-related proteins. Most of them are believed to serve as endogenous inhibitors of apoptosis and their overxpression is associated with cancer progression, multidrug resistance, poor prognosis and short survival. In brief, we presented the first comprehensive analysis of the expression of all eight BIRC genes in the course of CML. In addition to the previously described upregulation of BIRC5, we observed significant downregulation of BIRC3 and BIRC8 associated with TKI-resistance and also with progression to accelerated or blastic phase. Our results suggest novel and unexpected role of BIRC3 and BIRC8 in the clonal evolution of CML and open a new area for further exploration of the role of BIRC in CML progression.