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### NOVEL BCR-ABL PROTEIN PARTNERS AS PROMISING THERAPEUTIC TARGETS IN CHRONIC MYELOID LEUKEMIA

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**Background/Aim.** CML is driven by the Bcr-Abl oncoprotein, with its BCR region playing a key role in oncogenic signaling. This study aimed to identify novel Bcr-Abl partner proteins as potential therapeutic targets in CML. **Methods.** The K562 cell line was used. Protein complexes were studied using co-immunoprecipitation, GST-precipitation, His-tag pull-down analysis methods, the results obtained were studied using western blot analysis. Immunofluorescence analysis with subsequent confocal microscopy was also used. All experiments were performed in three independent biological replicates. **Results.** The presence of an interaction between full-length Bcr-Abl and USP1/GLG1 proteins in K562 cells was established. It was shown that the Bcr-Abl/USP1 protein complex is formed due to the PH domain and is localized in the nuclei of K562 cells. Colocalization of the Bcr-Abl and GLG was detected in the Golgi complex. A relationship was found between the deubiquitinating activity of USP1 and the level of the Bcr-Abl in cells. It was shown that inhibition of USP1 activity using pharmacopoeial drugs, in particular pimozide, promotes apoptosis of K562 cells. It was found that cortactin colocalizes with the PH domain and with full-length Bcr-Abl in the centrosome region and in the premembrane region at actin branching sites, and the involvement of clathrin in this colocalization was also shown. GST-precipitation and Western blotting demonstrated direct interaction of the PH domain of the BCR protein with cortactin and FBNP1. Colocalization of the Bcr-Abl and FBNP1 was demonstrated, and the latter was involved in the process of phagocytosis. **Conclusions.** Novel signaling pathways involving the Bcr-Abl and its protein partners USP1, GLG1, cortactin, and FBNP1 were identified, suggesting that these pathways may be dysregulated by the constitutive tyrosine kinase activity of the Bcr-Abl. The results obtained deepen the understanding of the mechanisms of stabilization of the Bcr-Abl level, its participation in intracellular transport, and remodeling of the actin cytoskeleton. A new approach to reducing the Bcr-Abl levels by inhibiting the deubiquitinating activity of USP1 has been identified. Thus, the identified Bcr-Abl partner proteins are important for further investigation as potential therapeutic targets for the development of new approaches to the treatment of CML. **Grants.** This work was supported by the National Research Foundation of Ukraine grant № 0126U003794.

**Keywords:** Chronic myeloid leukemia, Bcr-Abl, USP1, GLG1, cortactin, FBNP1.

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