Oncology
and Radiotherapy ©
16 (7) 2022: 001 • CONFERENCE PROCEEDINGS

Mechanism of Bcr-Abl/GLG1 protein complex formation and its role in the pathogenesis of chronic myeloid leukaemia

Antonenko Svitlana

PhD, Institute of Molecular Biology and Genetics National Academy of Sciences of Ukraine, Ukraine

ABSTRACT

Statement of the Problem: The development of chronic myeloid leukaemia is the result of a reciprocal translocation between chromosome 9 and chromosome 22 due to the emergence of Philadelphia chromosome. The product of this mutation is a hybrid onco-protein Bcr-Abl. According to the results of mass spectrometric analysis, GLG1 protein was identified as a potential candidate for interaction with Bcr-Abl. GLG1 protein occupies a privileged position in the signalling pathways of the Golgi complex, participates in the regulation of adhesion, migration, motility processes in cell. We believe that the Bcr-Abl phosphorylates GLG1 and thus deregulates its activity and disrupts numerous downward signalling pathways, leading to the progression of chronic myeloid leukaemia.

Methodology & Theoretical Orientation: K562 cells were selected for the experiment. The studies were performed using co-immunoprecipitation, Western blot, immunofluorescence staining, confocal microscopy. The results were analysed quantitatively and statistically.

Findings: The interaction between Bcr-Abl and GLG1 in K562 cells has been established. It was found that K562 cells are characterized by the presence of three isoforms of the GLG1 protein, with only one of them forming a complex with the Bcr-Abl onco-protein. It has been shown that the GLG1 protein isoform which forms a complex with the Bcr-Abl onco-protein is phosphorylated at tyrosine sites. It was detected that the formation of the Bcr-Abl/GLG1 protein complex occurs in the Golgi apparatus.

Conclusion & Significance: A new protein complex Bcr-Abl/GLG1 was detected in the Golgi apparatus in CML cells. It was found that during the formation of the Bcr-Abl/GLG protein complex, the onco-protein phosphorylates GLG1 at tyrosine sites. We believe that uncontrolled phosphorylation of GLG1 deregulates its properties, which causes disruption of the descending signalling pathways, processes of mobility, adhesion, cell migration and promotes disease progression.

Key words: Tumour, phyllodes, lymph node, metastasis, malignant

Address for correspondence:

Antonenko Svitlana, PhD, Institute of Molecular Biology and Genetics National Academy of Sciences of Ukraine, Ukraine, Email: antonenkoimbg@gmail.com

Word count: 391 **Tables:** 00 **Figures:** 00 **References:** 00

Received: - 09 July, 2022, Manuscript No. OAR-22-85717

Editor assigned:- 11 July, 2022, PreQC No. OAR-22-85717 (PQ)

Reviewed:- 25 July, 2022, QC No. OAR-22-85717 (Q)

Revised:- 27 July, 2022, Manuscript No. OAR-22-85717 (R)

Published:- 29 July 2022, Invoice No. J-85717