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Increased expression of CD24 in Glioblastoma Cell Lines after treatment with Temozolomide and Bortezomib

Dilara Akcora-Yildiz[1], Tulin Ozkan[2], Klara Dalva[2], Hasan Caglar Ugur[2], Asuman Sunguroglu[2] and **Seyma Ozkanca[3]**
[1]Mehmet Akif Ersoy University, Turkey
[2]Ankara University, Turkey
[3]Bogazici University, Turkey

Glioblastoma Multiforme (GBM), is the most prevalent and aggressive type of primary brain tumour with a median survival of only 15 months due to recurrence of tumour after surgical resection and acquisition of resistance to radiotherapy or chemotherapy. Temozolomide (TMZ) an oral alkylating agent leading to the occurrence of DNA damage has been still used for GBM treatment. Other than TMZ, Bortezomib (BZ), currently in clinical use for the treatment of myeloma by achieving proteasome inhibition, has been revealed to induce apoptosis and growth inhibition in GBM cells. Our purpose was to examine the role of potential cancer stem cell markers including CD133, CD38, CD24, CD70 and DR6 in cell survival after either TMZ or bortezomib treatment. U118 GBM cell line and other GBM cell lines including U87, U138 and T98 were incubated with TMZ and BZ for 48 hr, respectively. Real Time Cancer Stem Cell, Integrin, Apoptosis and Cell Adhesion PCR Arrays (Bio-Rad) were performed in U118 cells treated with TMZ for 48 hr. Flow cytometry assay was used to determine the protein amounts of the genes of interest after BZ treatment. Treatment with TMZ led to an increase in mRNA expression of CD38 and CD24 but not in CD70 and DR6. BZ decreased the expression of CD133 and CD38, whereas CD24 expression was found to be increased in a dose-dependent manner in all GBM cell lines. Furthermore, CD70 protein expression was elevated, while DR6 protein expression was reduced with the increase of the dose of BZ. Our results suggest that CD24 seems to be involved in GBM cell survival after either TMZ or BZ treatment, indicating inhibition of its expression might benefit to overcome chemo resistance. This research has been supported by The Scientific and Technological Research Council of Turkey (No: 114S189).

Biography

Dilara Akcora Yildiz is an Assistant Professor at Biology Department, Mehmet Akif Ersoy University. She has been the Vice Director at the Institute of Science and Technology since 2016. She graduated from the Biology Department, Faculty of Science, Ege University, Turkey in 2004. She received her master's degree from the Medical Biology Department, Faculty of Medicine, Ankara University, Turkey in 2007 and studied the effect of T315I, E255K and M351T mutations in imatinib resistance in chronic myeloid leukaemia patients. In the same year she was awarded with a Postgraduate Education Scholarship in Australia by the Ministry of National Education of Turkey (MEB) (2008-2012). She then earned her Ph.D. degree in intestinal stem cell biology at Department of Pathology at The University of Melbourne in 2012 under the supervision of Prof. Dr. Robert G. RAMSAY. During her doctoral studies, she characterized the role of colony stimulating factor 1 receptor-ligand pair (Cfms/CSF1) and granulocyte macrophage colony-stimulating factor (GM-CSF) in intestinal biology. She was the principal investigator of a research project titled as 'The effect of WRN and MGMT proteins which play a role in DNA repair on drug resistance occured in Multiple Myeloma disease' and supported by The Scientific And Technological Research Council Of Turkey (TUBITAK - 3501 National Young Investigator Career Development Program). She is currently working as an investigator in other projects focused on brain tumors, cancer stem cells and antibody production. Dr. Akcora Yildiz was a participant at 9th HOPE Meeting with Nobel Laureates in 2017. Her research interests include stem cell biomarkers, DNA repair mechanisms, apoptosis and autophagy signaling in cancer biology.

dilaraakcora@mehmetakif.edu.tr

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