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Effect of Bortezomib in L1210 Cells

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Abstract

During chemotherapy, leukemic cells can develop resistance to a wide variety of cytotoxic agent with different structures or mechanisms of action. This phenomenon is also referred to multidrug resistance and it is often mediated by proteins called ABC transporters. One of the most studied members of this protein family is P-glycoprotein (P-gp), which expression seems to be secondary associated with changes in some metabolic and regulatory pathways. Protein homeostasis is regulated through degradation of either central lifespan regulators or abnormally folded proteins regulated through ubiquitin-proteasome system. However, ubiquitination can also stimulate endocytosis or even intracellular trafficking. The main goal of our experiments was to characterise an effect of bortezomib (proteasome inhibitor) on polyubiquitin chains linkage through lysines, K63 and K48 expression of selected molecular chaperones in P-gp positive (R, T) and P-gp negative (S) variants of L1210 cells. We have observed that P-gp positive (R and T) cells were less sensitive to bortezomib. Further, we detected increased expression of molecular chaperones Grp78, Hsp70 and Hsp90α in R and T cells, either in the absence or presence of bortezomib when compared with S cells. Moreover, bortezomib after 24 hours induced differences in content and profile of polyubiquitinated protein through K48 linkage between P-gp positive and P gp negative cells. K63 linked ubiquitination was unchanged in all three variants of L1210 cells. R and T cells seem to restore process of ubiquitination after cultivation with bortezomib, but study of this feature is necessary.

Keywords: MDR; molecular chaperones; ubiquitination; bortezomib

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