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Role of Protein Kinase C in Cd47-Mediated Phosphatidylserine Expression Pathway by Bric 126 in Leukaemia Cell Line

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ABSTRACT

Treatment failure in T-cell acute lymphoblastic leukaemia (T-ALL) occurs when leukemic blasts acquire resistance to chemotherapeutic agents. Current research efforts are focused on the search for targets for the development of more effective and less toxic anti-leukemic drugs. CD47 has been suggested to be involved in chemo-resistance and cell metastasis. Although several potential mechanisms were suggested to explain the therapeutic effect of CD47-targeting; the downstream effectors which lead to different effects by CD47 are still not well understood. In this study, the role of Protein kinase C (PKC) in CD47-mediated phosphatidylserine (PS) expression pathway in jurkat T cells was investigated. Jurkat T cells were incubated with anti-CD47 mAb (BRIC 126), anti-CD44 mAb (BRIC 235) or control in the presence or absence of Bisindolylmaleimide I, hydrochloride (PKC inhibitor). Cells were stained with annexin-V FITC. Flow cytometry analysis was used for measurement of fluorescence intensity. Cell viability was detected using trypan blue exclusion test. PKC inhibition enhanced phosphatidylserine expression in CD47 receptor-mediated leukaemia cells apoptotic pathway. This indicates that PKC may be involved in CD47-mediated PS exposure pathway in jurkat T cells. The observations from this study identify CD47 and PKC as novel functional proteins in jurkat T cells with promising therapeutic potential. This study would provide insight for targeted therapy against T-ALL disease.

Keywords: Jurkat T-cell, Acute lymphoblastic leukaemia, CD47, PKC, Phosphatidylserine, Apoptotosis, Flow cytometry

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