A study of CD180 and CD32 expression profile on Chronic Lymphocytic Leukaemia cells and MEC1 cell line.

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Chronic lymphocytic leukemia (CLL) is the most common leukaemia in the US and in Europe, including Georgia. Chronic Lymphocytic Leukemia (CLL) presents with clonal expansion and accumulation of CD5+CD19+CD23+ cells in peripheral lymphoid organs and tissues and in bone marrow.CLL is supposedly driven by exogenous or endogenous (auto)antigen(s) and there isincreasing evidence that CLL cellsreceive microenvironmental signals which support their growth,survival and expansion *in vivo*. We have previously shown that powerful signals are received by CLL cells through CD180 orphan toll-like receptor. Additional accessory signals could be generated through FcγRII (CD32), and both are expressed on CLL cells as well as on control B cells. Here we studied correlation of expression of CD32 and CD180 on CLL cells as well as on MEC1 cell line.

Peripheral blood mononuclear cells (PBMC) from CLL patients and age-matched healthy volunteers were separated, stained with appropriate antibodies to CD19, CD32 and CD180 and analysed by flow cytometry. CD32 and CD180 expression on MEC1 cells was studied at different time-points. The data was statistically analysed using the Mann-Whitney non-parametrical test.

Our data indicates that expression of CD32 is significantly increased on CLL cells compared to control B cells as well as in long-term MEC1 cell culture. In contrast, CD180 expression on MEC1 cells significantly decreased throughout 0-96h of MEC1 cell culture. We have recently shown that CD180 ligation can redirect sIgM-mediated signaling from pro-survival to pro-apoptotic. This data indicates that a drop in the expression of CD180 on cycling CLL cells might lead to a weakening of this effect and enhance further survival and expansion of CLL cells in proliferative centres of lymphoid tissues. Since MEC1 cells are derived from a CLL patient with mutated *IGVH* genes (M-CLL) negative correlation between CD180 and CD32 expression on cycling MEC1 cells could be limited to M-CLL.