

Long term activation of natural killer cells results in modulation of beta 1-integrin expression and function

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Abstract

Integrin expression and function is largely modulated by cell activation. Here we provide evidence that long term activation of human NK cells results in a marked modulation of beta 1-integrin expression and adhesive functions. By flow cytometry and immunochemical analysis we have detected induction of alpha 1 beta 1 and alpha 2 beta 1, increased expression of alpha 4 beta 1 and alpha 5 beta 1, and decline of alpha 6 beta 1 on CD3-CD56+ NK cells generated from 10-day coculture of nonadherent PBMC with irradiated RPMI 8866 EBV+ lymphoblastoid B cell line. Adhesion assays performed on extracellular matrix-coated plates showed that, unlike fresh NK cells, long term-activated NK cells bind to native collagen I via alpha 2 beta 1 and to heat-denatured collagen I in an RGD-dependent manner, although they lose the ability to bind to laminin. In regard to the adhesion to FN, no major quantitative changes are observed after long term NK cell activation. However, whereas alpha 4 beta 1 and alpha 5 beta 1 completely mediate the adhesion of fresh NK cells to fibronectin, binding of activated NK cells is only partially beta 1-dependent and seems to involve also non-beta 1-integrin(s) recognizing and RGD sequence. The modulation of beta 1-integrin expression and the acquisition of new adhesive properties on long term-activated NK cells may be relevant for their traffic and tissue localization during inflammation and immune response.