B7-H6-mediated downregulation of NKp30 in NK cells contributes to ovarian carcinoma immune escape

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In this study the phenotype and function of tumor-associated NK cells from peritoneal fluids of a selected cohort of patients with seropapillary ovarian carcinoma were analyzed. In >50% of these patients the expression of the activating receptor NKp30 (1) in tumor-associated NK cells was substantially reduced as compared to autologous peripheral blood NK cells. The impaired expression of this receptor was associated with the presence of one of its cellular ligands (B7-H6) (2), which was detectable as a surface/cytosolic molecule in tumor cells and as a soluble molecule in the peritoneal fluid. NK cells from patients expressing this NKp30low phenotype displayed an impaired IFNγ production and cytolytic function when tested against target cells expressing surface B7-H6. Our data also suggest that in these patients the defective expression and function of NKp30 may be induced by the chronic engagement of this receptor by soluble B7-H6 or by tumor cells expressing this ligand. The impairment of NK cell functions described herein could represent a novel mechanism by which the tumor microenvironment may contribute to the escape from immune surveillance.

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References:

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