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Release of iC3b from apoptotic pancreatic tumor cells induces tolerance by binding to immature dendritic cells

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Background & Aims

Chemo- as well as immunotherapeutical approaches induce apoptosis in tumor cells. Apoptotic cells are known to activate homologous complement and to be opsonized with iC3b. Since maturation of dendritic cells (DC) can be inhibited by binding of iC3b to the complement receptor 3 (CR3, CD11b/CD18) and because immature DC induce tolerance, we investigated the induction of tolerance after pulsing DC with apoptotic cells in the presence or absence of native serum.

Methods

Apoptosis in pancreatic carcinoma cells was induced either by heat-stress, chemotherapy or anti-Her2 antibody. Monocyte-derived DC were pulsed with apoptotic cells with or without native serum. DC were analyzed for the maturation state by flow cytometry and the cytotoxic activity was determined. Tolerance was prevented by addition of substances such as anti CD11b or *N*-acetyl-D-glucosamine (NADG) which block iC3b binding to CR3.

Results

All of the former strategies for apoptosis induction resulted in iC3b release. Pulsing DC with apoptotic cells in the presence of serum prevents maturation of DC and induces finally tolerance. This tolerance could be prevented almost completely by blocking the interaction of iC3b with the CR3 receptor.