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Title: Induced expression of B7-H3 on the lung cancer cells and macrophages suppresses tumor-specific T cell immunity

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Body: Macrophages are prominent components of solid tumors and have complex dual functions in their interaction with cancer cells. Strong evidence suggests that TAM is part of inflammatory circuits that promote tumor progression. B7-homologue 3 (B7-H3), a recently identified homologue of B7.1/2 (CD80/86), has been described to exert co-stimulatory and immune regulatory functions. Here, we showed that a fraction of macrophages in tumoral stroma expresses surface B7-H3 molecules in lung cancer carcinoma model. Normal macrophages activated by tumor cell strongly express B7-H3 proteins. Although a lung cancer cell line constitutively expressed B7-H3 mRNA and protein in plasma, primary tumor cell isolated from the transplanted lung carcinoma model expressed B7-H3 on the surface. Interestingly, in transplanted lung carcinoma model, the expression of membrane-bound B7-H3 in tumor cells was increased as prolonging of tumor transformation. Interleukin 10 released from TAM could stimulate cancer cell expression of membrane bound B7-H3. Furthermore, Lung cancer and TAM-related B7-H3 was identified as a strong inhibitor of T-cell effect. B7-H3 expression may significantly influence the outcome of T cell immune response and TAM-tumor cell interactions induced membrane-bound B7-H3 represents a novel immune escape mechanism which links the proinflammatory response to immune tolerance in the tumor milieu.