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Heat Shock Protein 70 (HSP70) induces cytotoxicity of T-helper cells

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Stress-inducible heat shock protein 70 (HSP70) has gained plenty of attention because of its potent adjuvant capability to induce antigen-specific $CD8^+$ cytotoxic T-lymphocyte (CTL) and $CD4^+$ T-helper cell (Th1) responses. In this study, we investigated the behavior of T-cell subsets stimulated with endotoxin-free recombinant HSP70 with respect to proliferation, cytokine expression, cytotoxicity against allogeneic B-lymphoblastoid cell line (B-LCL) and K562 cells as well as target-independent cytotoxicity.

$CD4^+$ cells exhibited a strong increase in proliferation after stimulation with HSP70, with rates of up to 29%. In the presence of target cells, a 35-fold up-regulation of granzyme B mRNA was observed after stimulation of $CD4^+$ T-helper cells with HSP70 in combination with IL-7, -12 and -15. The target cell-independent secretion of granzyme B by $CD4^+$ cells was greatly augmented after stimulation with HSP70 plus IL-2 or IL-7, -12 and -15.

In this study, we have shown that HSP70 is capable of inducing a cytotoxic response of T-helper cells in the absence of LPS or any other PAMPs. The granzyme B secretion and the cytolytic activity of $CD4^+$ T cells is induced in a target-independent way, whereas the cytotoxic activity of $CD3^+$ and $CD8^+$ T cells can be further enhanced in the presence of the target cells. Our data provide novel insights into the role of extracellular HSP70 on T-cell immune response concerning the induction of target-independent T-helper cell cytotoxicity.