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CD30 在調節型 T 細胞所扮演之角色

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CD30 Is Involved in Inhibition of T-cell Proliferation by Hodgkin's Reed-Sternberg Cells[1]

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ABSTRACT

CD30 is expressed on Hodgkin's Reed-Sternberg (H-RS) cells, the tumor cells in Hodgkin's disease (HD). Increased levels of serum CD30 are observed in HD patients and are a good marker for predicting a poor prognosis and response to therapy. In this study, we addressed the effect of CD30 on T cells. We showed that CD30, either as a membranous protein on H-RS cells and Chinese Hamster Ovary (CHO) cells, or as a plate-bound chimeric protein, inhibited T-cell proliferation. Anti-CD3-stimulated T cells in the presence of CD30 failed to increase tritium uptake, and failed to express CD25 and CD26 and produce IL-2. The inhibition of T-cell proliferation was,

however, reversed with addition of exogenous IL-2 or soluble CD153. Pretreatment of H-RS cells with anti-CD30 also reversed the inhibition. Inability of T cells to express CD25 and CD26 in cocultures with H-RS cells or a plate-bound CD30 chimeric protein is in accordance with the results of immunohistochemistry on disease-involved tissues. We also found increased level of mRNA for P21, a cell cycle inhibitor, in anti-CD3-treated T cells in the presence of plate-bound CD30-Fc fusion protein. This may explain the profound inhibitory effect of CD30 on T cells. We conclude that H-RS cells are able to inhibit the proliferation and activation of T cells through CD30-related interaction.