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T cell recognition of an immunodominant peptide in a genetic disease (HEL) where host HEL is the immunogen. The interaction of T cells with antigen-presenting cells (APC) in vivo or in vitro is influenced by factors such as the density of APC and the concentration of antigen. We have studied this interaction in the disease model of P3HR-I cells expressing the HEL protein, a major sub-nucleus Ag in the Prt genes. Under normal conditions, the activation of HEL-specific T cells is weak and limited, but is greatly enhanced by increasing the number of APC or by increasing the peptide concentration on the surface of APC. When control P3HR-I and HEL-transfected P3HR-I (HLA-DR3) cells were co-cultured in the presence of antigen for 18 h, large numbers of T cells, however, were found in the APC, indicating that this very low level of T cell activation could be converted by increasing the dose of antigen on the APC surface. Activation of HEL-specific T cells was also high when the antigen was present in high local concentration. Normal P3HR-I or HL-A3.3 T cell blasts were cultured with target cells for 18 h in the presence of antigen and the products of the lytic reaction were fixed with glutaraldehyde and then examined by scanning electron microscopy. In this system we observed large numbers of T cells bound to target cells at ratios of 1:1 and 3:1. Hereafter, we shall refer to such T cells as "high-avidity" (HA) cells. We have also shown that the generation of the HA phenotype is dependent on the antigen/MHC density ratio and is disrupted by the presence of anti-Leu-11 or Leu-3 monoclonal antibodies. The conditions that we have described define a new HA phenotype for T cells, a phenotype where T cells are selectively drawn by the presence of antigen to the surface of target cells in a way that depends upon the ability of the target cells to express the appropriate HLA Class II products.
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