The immune response to a transplanted organ

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The main immune cells involved in transplant immunology are APCs and T and B lymphocytes. These cell types interact by a series of specific surface molecules that are designated by CD (cluster of di ff erentiation) numbers. Cell surface receptors can only interact if they have the correct complementary three-dimensional structures. The immune response to an allograft is orchestrated by T lymphocytes. When T cells encounter foreign transplant HLA antigens they become activated and then proliferate into clones of cells that attack the allograft. Full T-cell activation requires a number of signals (Figure 88.6). Transplant antigen). -

CD4 A TCR MHC II CD3 H Signal 1: antigen APC T presentation CD28 CD80/86 Signal 2: co-stimulation Figure 88.6 Interaction between an antigen-presenting cell and a CD4+ T-helper cell. A, antigen; APC, antigen-presenting cell; CD, cluster of differentiation; MHC II, major histocompatibility complex

H class II; TCR, T-cell receptor; T , T-helper cell. (Adapted with permis

sion from Clatworthy M, Watson C, Allison M, Dark J. Transplantation at a glance . John Wiley and Sons Ltd, 2012.)

molecules by specialised APCs. The class II/peptide antigen complex is recognised by the T-cell receptor (signal 1). CD4+ T-helper cell activation also requires a co-stimulatory signal involving the interaction of pairs of molecules, one on the sur face of the T cell and the other on the surface of the APC (signal 2). An example of signal 2 is the interaction of T-cell CD28 with CD80 on the APC. When both signal 1 and signal 2 are received the CD4+ T-helper cell will upregulate expres sion of the interleukin-2 (IL-2) receptor (CD25) on its cell sur face. Binding of IL-2 to its receptor causes further activation of the T cell (signal 3). The T cell will then proliferate and release more IL-2, which, in turn, leads to activation and clonal proliferation of T-killer cells (CD8+). These cytotoxic killer cells infiltrate the allograft and cause cell death by the release of molecules called perforin and granzyme. Perforin punches holes in the target cell membrane and this allows passive dif fusion of granzyme into the cell, where it activates caspase enzymes and causes cell death by apoptosis.

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