The effect of cytotoxic T cells and NK cells

- Regression transplanted tumours due to CTLs
- Tumour variant with low MHCI, less sensitive to CTLs but more sensitive to NK cells
- *nude* mice = no T cells, more NK cells than normal
- Tumour rejection antigens = peptides of tumour cell proteins presented on MHC
- Target of tumour-specific response even though present on normal tissues e.g. strategy to induce response to melanoma antigen → vitiligo

1) Normal mice will mount an immune response using mostly CTL, while *nude* mice will not be so successful at dealing with the tumour.

2) Tumour which is not displaying MHCI normal mice will not be successful at dealing with the tumour but *nude* mice which have increased numbers of NK cell dealt with the tumour faster and better.

3) Tumour lost MHCI, is supplied with a MHCI gene we are back to original scenario.

There are certain types of proteins which are more liable to be tumour rejection antigens. It is often random and some of these proteins are not even cell surface antigens.

**Tumour rejection antigens**
- Point mutations or gene rearrangements occurring during oncogenesis.
- Proteins normally only expressed in male germ cells, male germ cells do not express MHC therefore these peptides not normally presented to T cells.
- Differentiation antigens, usually very tissuespecific, e.g. tyrosinase – normally only found in melanocytes.
- Tumour immunotherapy tries to harness and augment these responses.