1. _____ are able to bind to and kill infected cells and tumor cells that suppress MHC-I production.

A. CTLs
B. NK cells
C. Macrophages
D. Effector T4-cells
E. Dendritic cells

2. NK cells use the _____ on their surface to bind to _____ on infected cells and tumor cells.

A. killer-inhibitory receptors; stress glycoproteins
B. TCRs and CD8 molecules; MHC-I molecules with bound peptide epitopes from endogenous antigens
C. killer-activating receptors; stress glycoproteins
D. MHC-II molecules with bound peptide epitopes from exogenous antigens; TCRs and CD4 molecules

3. NK cells are able to bind to _____ that have reacted with epitopes of infected cells and tumor cells and kill them by apoptosis.

A. IgG
B. MHC-I molecules with bound endogenous antigen
C. IgM
D. stress glycoproteins
E. caspase enzymes

4. Activation of macrophages requires:

A. The binding of TCRs and CD8 molecules on CTLs to MHC-II molecules with bound peptide from exogenous antigen on the macrophage.
B. The binding of TCRs and CD4 molecules on naïve T4-lymphocytes to MHC-II molecules with bound peptide from exogenous antigen on the macrophage.
C. The binding of TCRs and CD4 molecules on T_h1 cells to MHC-II molecules with bound peptide from exogenous antigen on the macrophage.
D. The binding of TCRs and CD4 molecules on T_h2 cells to MHC-I molecules with bound peptide from endogenous antigen on the macrophage.

5. Increasing the production of toxic oxygen radicals, nitric oxide, and hydrolytic lysosomal enzymes; producing inflammatory cytokines and cytokines that enable naive T4-lymphocytes to differentiate into T_h1 cells. These are all benefits from the activation of _____ by T_h1 cells.

A. macrophages
B. NK cells
C. CTLs
D. dendritic cells
E. T_h2 cells