Diseases	
SSPE	sub acute sclerosing panencephalitis brain disease that is a late consequence of measles
NEMO Deficiency	X-linked hypohydrotic ectodermal dysplasia and immunodeficiency lack one subunit of IKK --> impaired activation of NFκB inefficient macrophage activation through TLR4 signaling on x-chromosome --> more common in boys
SCID	severe combined immunodeficiency disease one of the RAG genes doesn't work both B and T cells are absent children die in early infancy from common infections (unless they have bone marrow transplants)
Omenn Syndrome	RAG proteins have partial enzymatic activity differs from SCID in symptoms --> red rash on face and shoulders
Bare-Lymphocyte Syndrome	TAP is non-functional no peptides in ER --> MHC I doesn't reach surface patients have less than 1% of normal MHC class I lack of MHC II expression (CD4 T cells fail to develop); lack of MHC I (loss of CD8 T cells) patients have poor CD8 T-cell responses and suffer from chronic respiratory infections
Burkitt's Lymphoma	parts of chromosome 8 and 14 have been exchanged MYC (proto-oncogene) on chromosome 8 and heavy (14)/light (2 or 22) chain genes switch abnormal expression as a result of translocation causes increased growth
DiGeorge's Syndrome	deletion in chromosome 22 thymus fails to develop T-cells are absent susceptibility to wide range of opportunistic infections (resembles SCID)
ALPS	(Lymphadenopathy in autoimmune lymphoproliferation syndrome) patients lack functional Fas molecules cannot control size of lymphocyte population nor remove autoimmune cells secondary lymphoid organs become swollen in absence of infection
Hyper-IgM syndrome	patients that lack CD40 ligand (on T cell) abnormally high levels of IgM in blood serum almost no IgG and IgA B cells are unable to switch isotype cannot make Ab responses to TD antigens

	no germinal centers X-linked; prominent in males
Hemolytic Disease of the Newborn	Mom is Rh negative but dad isn't 1st child is fine but when first baby is born, fetal RBCs bleed into mom --> adaptive immune response second pregnancy --> if fetus is also Rh positive, mom can make IgG antibodies that can cross the placenta and tag RBCs for destruction Anti-Rh IgG (RhoGAM) is given to mom at 28 weeks and within 72 hrs after birth coats baby's RBCs that enter mom tricks immune system into thinking it's a secondary immune response
Influenza	RNA virus Pattern of infection = epidemics Virus spreads fast and subsides quickly genome: 8 RNA molecules RNA replication is error-prone; generates many point-mutations new viral strains lack hemagglutinin or neuraminidase epitopes that induced protective immunity in previous epidemis new generation of virus evades protective immunity --> ensures long-term survival of virus antigenic drift: changes are small (point-mutation), so some cross-reaction occurs; epidemics occur every other winter pandemics are caused by recombinant viruses (part avian and part human), so hemagglutinin/neuraminidase epitopes are very different from humans Antigenic shift: pandemic, severe, avian origin creates very different strain every 10-50 years most often occurs in farmers (near to livestock) viruses arise from pigs that become infected with both avian and human simultaneously
African sleeping sickness	caused by trypanosomes (a protozoan) trypanosomes live in mammals and insects (cyclic) trypanosomes use gene rearrangement to change surface antigens they usually express one dominant type of VSG --> antibodies produced against it but not against those that have switched VSG genome contains more than 1000 genes encoding VSGs this mechanism produces a dramatic cycling in # of parasites chronic cycling of Ab production and antigen clearance --> heavy deposition of immune complexes and inflammation --> inflammation causes neurological damage and coma
Herpes	persists in host by 'hiding from immune response' in sensory neurons

	infects epithelial cells; spreads to sensory neurons serving infected area Latency: dormant state (no replication nor generation of enough peptides to signal presence) virus is unrecognized stress reactivates the virus --> reinfects epithelial tissues --> causes sores nerve cells have little MHC --> reduced CD8 T cell participation
Herpes: Epstein Barr	childhood infection (mild, cold-like diseases) infects B cells by binding to CR-2 component of B-cell receptor complex infected B cells proliferate and produce virus --> stimulates EBV-specific T cells to react small number of B cells become latently infected but replication is controlled by CD8 cells
Herpes: Varicella-Zosters	chicken pox dormancy in dorsal root ganglia reactivation causes shingles
Tuberculosis	caused by mycobacterium tuberculosis prevents fusion of phagosome with lysosome
Syphillis	caused by treponema pallidum coats itself with human protein
Toxic shock syndrome	toxins bind to sites shared by many different TCRs stimulate excessive polyclonal response (involved 2-20% of total # of CD4 T cells) after proliferation T-cells with bound antigen die by apoptosis and remove memory T cells massive cytokine release: IL-1, IL-2, TNF-alpha antigen bridges MHC class II and TCRs
X-linked Agammaglobulinemia	Antibody deficiency B cell growth and differentiation blocked at pre-B cell stage due to defect in gene coding for tyrosine kinase (Btk) intracellular signaling is lost increase in bacterial infections that require antibodies for clearance (capsulated) -complement activation increase in viral infections that enter in gut (normally neautralized by antibodies)
X-linked hyper IgM	diminished antibody production see Hyper IgM syndrome above
C3 complement deficiencies	defects in complement components C3 deficiencies: increased bacterial infections phagocytosis is decreased