Parasites

Niche

1. **Ecological niche** = multidimensional summary of tolerances and requirements of a species
2. **Fundamental niche** = species occupies this in absence of any interspecific competition
3. **Realised niche** = species occupies this in response to interspecific competition
4. There are two outcomes of interspecific competition:
   - **Competitive exclusion**—niches of 2 species are so similar that one is locally extinct
   - **Resource partitioning**—niches are different enough for potential competitors to co-exist
5. **Parasitism** = symbiotic relationship where parasite benefits in terms of energy & nutrients at the expense of the host
6. Unlike predator-prey relationship, reproductive potential of parasite is greater than host
7. Most parasites are **host specific** so have a **narrow (specialised) niche**
8. Many parasites are **degenerate** = lack structures/organs found in other organisms
9. **Ectoparasite** lives on the surface of the host, **Endoparasite** lives within the host

Parasite life cycle

1. Some parasites require one host to complete their life cycle, many require more than one
2. **Definitive host** = organism where parasite reaches sexual maturity
3. **Intermediate host** = may also be required for parasite to complete life cycle
4. **Vector** = active role in transmitting parasite (could also be a host)
5. Malaria is caused by Plasmodium:
   - Infected mosquito (vector) bites a human and Plasmodium enters blood stream
   - Asexual reproduction occurs in liver and then red blood cells
   - Red blood cells burst and release gametocytes in blood stream
   - Another mosquito bites infected human, gain gametocytes which mature into gametes
   - Gametes are able to carry out sexual reproduction & mosquito can infect another human
6. Schistosomiasis is caused by schistosomes:
   - Schistosomes reproduce sexually in small intestine & pass out fertilised eggs in faeces
   - Fertilised eggs reach water where they develop into water and infect water snails
   - Asexual reproduction occurs in snail which produces larvae which escape snail & penetrate skin of human to get to the blood stream
7. Viruses are parasites that can only replicate in a host
8. Viruses contain genetic material (**DNA or RNA**) packaged in a protective protein coat
9. Some viruses are surrounded by phospholipid membrane derived from host cell materials
10. Outer surface of a virus contains **antigens** that a host may or may not detect as foreign

Parasite life cycle

11. **Life cycle**:
    - Infect host cell with genetic material
    - Host cell enzymes replicate viral genome
    - Transcription of viral genes and translation of viral proteins
    - Virus assembles and new viral particles are released

12 **Retroviruses** (RNA is the genetic material) use enzyme **reverse transcriptase** to convert RNA to DNA, which is then inserted into the genome of the host cell

Transmission and Virulence

1. **Transmission** = spread of parasite to a host
2. **Virulence** = harm caused to a host species by a parasite
3. **Ectoparasites** are generally transmitted by direct contact or consuming intermediate hosts
4. **Endoparasites** of body tissues are often transmitted by vectors
5. Transmission rates are increased by overcrowding of hosts at high density or mechanisms (such as vectors and waterborne dispersal) that allow the parasite to spread even when hosts are incapacitated
6. **Host behaviour can be exploited and modified by parasites** to maximise transmission (alteration of host foraging, movement, sexual behaviour, habitat choice or anti-predator behaviour)
7. Host behaviour becomes part of the **extended phenotype** of the parasite
8. Parasites can suppress host immune system and modify host size and reproductive rate in ways that benefit parasite growth, reproduction or transmission

Parasites

Defence against parasitic attack

1. Non specific defences:
   - **Physical barriers** (epithelial tissue blocks entry of parasites)
   - **Chemical secretions** (hydrolytic enzymes in mucus/saliva/tears destroy bacterial cell walls and low pH in stomach/sweat glands denatures cellular proteins of pathogens)
   - **Inflammatory response**—injured cells release signalling molecules which enhances blood flow to site, bringing antimicrobial proteins and phagocytes)
   - **Phagocytes** engulf pathogens and store them in a vacuole during phagocytosis. Lysosomes fuse with vacuole and enzymes digest pathogen/parasite
   - **Natural killer cells** identify and attach to cells infected with a virus, releasing chemicals that induce apoptosis

2. **Specific cellular defences**
   - If tissues are damaged/invaded, cells release **cytokines**
   - Cytokines increase blood flow, which causes non-specific and specific white blood cells to accumulate at the infection site
   - **Lymphocytes** possess different receptors on their surface which can recognise antigens
   - Antigen binds to receptor to cause lymphocyte to divide and produce a **clonal population** of the lymphocyte
   - Some lymphocytes produce **antibodies** & some induce **apoptosis** in parasite-infected cells
   - Antibodies possess regions where amino acid sequence varies greatly between antibodies —this gives each antibody specificity for a different antigen
   - When antigen binds to antibody binding site, the **antigen-antibody complex** can inactivate the parasite
   - This makes it susceptible to a phagocyte or stimulates a response that results in **cell lysis**
3. **Memory lymphocytes** are also formed from initial antigen exposure. Memory cells are specific to that antigen and can produce a **secondary response** if the antigen enters the body again
4. Secondary response **enhances antibody production in terms of speed of production, concentration in blood and duration**
5. A range of white blood cells constantly circulates to monitor tissues (**immune surveillance**)

Immune evasion

1. Endoparasites **mimic host antigens** to evade detection and modify host immune response to reduce chance of destruction
2. **Antigenic variation** allows parasites to change between different antigens when invading a host. This can also allow re-infection of the same host with the new variant
3. Some viruses escape immune surveillance by **integrating their genome into host genomes**, existing in an inactive state of latency. Virus is active again in favourable conditions.

Challenges in infection & control

1. **Epidemiology** = study of outbreak and spread of infection disease
2. **Herd immunity threshold** = density of resistant hosts in population required to prevent an epidemic
3. **Vaccines** have antigens that will elicit an immune response
4. Similarities between host and parasite metabolism makes it difficult to find drugs that only target parasite
5. **Antigenic variation** has to be reflected in design of vaccines
6. Parasites are difficult to culture in the lab which can make designing a vaccine difficult
7. **Challenges** arise where parasites spread most rapidly—tropical climates & overcrowding. This makes co-ordinated treatment and control programs difficult to achieve
8. **Civil engineering projects** to improve sanitation combined with vector control nay often be the only practical control strategy
9. **Improvements in parasite control** reduce child mortality and result in population-wide improvements in child development and intelligence as individuals have more resources for growth and development