# INTRODUCTION TO VETERINARY PATHOGENESIS

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# HOST-PATHOGEN-ENVIRONMENT INTERACTIONS

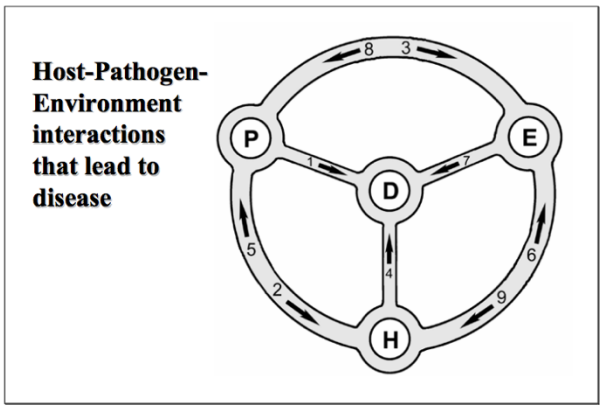
Infection: presence of organisms within the host with or without manifestation of disease

Colonisation: presence of organisms within the host which do not cause disease

Commensals: parasites that establish a non-offensive relationship with the host

Sub-clinical disease: disease in which manifestations are unnoticeable through clinical exam

Clinical disease: disease that produces clinical signs




## DENSITY VS FREQUENCY DEPENDENT TRANSMISSION

- Density-dependent:
  - By random contact
  - Less-dense host → less transmission → extinction of pathogen
- Non-density-dependent:
  - Transmission depends on the frequency of infected individuals in population
  - E.g. sexually transmitted diseases

## PERSISTENCE

- Organisms that optimise transmission rate are most likely to persist
- Persistence can be in the host (keep host alive), or in the population (kill host and disperse in population)

## MHC DIVERSITY

- The higher the MHC diversity (in the host) the more diverse range of antigens it can respond to

## CONCLUSION

- HPEI gravitates to a dynamic equilibrium → large scale ecology
- We only see a snapshot of it
- Understanding this concept is essential for identifying management points and assessing disease risks

# CELL INJURY

## DEVELOPMENT OF CELL INJURY

1. Membrane Damage
    - Damaged membrane = leaky membrane
    - Membrane damage often followed by influx of Na+, Ca2+, water into cell
    - Results in swelling → spatial arrangement changes → substrates diffused
    - Free radicals (often charged O2) released faster than cell can clear them
    - In animals chronically exposed to toxins, sER increases enzyme activity, increasing detoxification ability
    - Sudden increase in toxins leads to massive cell injury (sER doesn't have time to fight)
    - Actual membrane damage is peroxidation of membrane lipids

2. Energy (ATP) Depletion
    - Cells need energy to drive ion pumps, synthesise molecules/enzymes etc.
    - Na-K-ATPase pump fails, Na rushes in → cellular swelling
    - Mitochondria and rER first affected by swelling
    - Cell turns to anaerobic glycolysis, increases lactic acid, decreases pH, not ideal for enzymes
    - Most common cause is hypoxia (oxygen depletion)
    - No oxygen, no ETC, TCA cycle slows as well

### CAUSES OF HYPOXIA

a. Oxygen not reaching blood
b. Oxygenated blood not reaching tissues
c. "respiratory poisons" stopping cells from using oxygen

### MORPHOLOGICAL CHANGES

- Cellular swelling (most obvious) (AKA oncosis) → membrane's damaged
    → Vacuolar degeneration or hydropic degeneration
- Sublethal injury: cloudy swelling, hydropic change, fatty change
- Lethal cell injury: combination of membrane damage and Calcium influx
- Apoptotic cells: cell shrinkage, rapid condensation of chromatin + cytoplasm, intact membrane
- Necrotic cells: lighter than surrounding tissue, swollen, surrounding inflammation