

INTRODUCTION TO VETERINARY PATHOGENESIS

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HOST-PATHOGEN-ENVIRONMENT INTERACTIONS

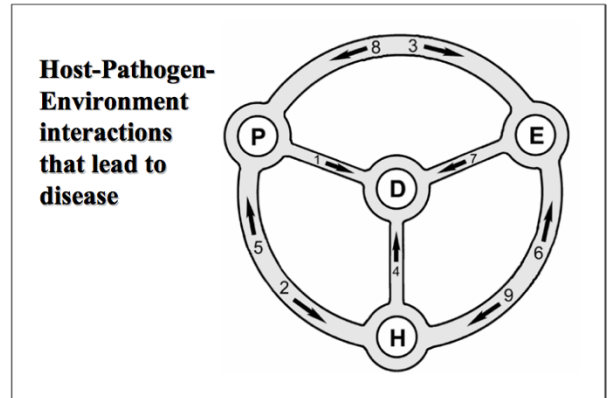
Infection: presence of organisms within the host with or without manifestation of disease

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^+ , Ca^{2+} , water into cell
- Results in swelling → spatial arrangement changes → substrates diffused
- Free radicals (often charged O_2) 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