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Cryptosporidium spp.

Prevalence

- Infection of contemporary mouse colonies seldom reported (occasionally observed in wild rodent populations)
- Typically, not observed in healthy, conventional mice and rats
- Patent infection appears to be dependent on host intestinal microflora which assist resistance - suckling mice particularly at risk of **Cryptosporidiosis**
- SCID and athymic nude mice – unable to clear *C. muris* and *C. parvum* infections

Significance

- Clinically ill animals are not suitable for research
- Zoonotic risk to humans and potential danger to immunocompromised mice
- Suckling and immunosuppressed mice – useful in vivo model for anticryptosporidial compound testing
- Work done in immunocompromised models – elucidate immune mechanisms responsible for infection clearance

Disease

- *C. parvum* includes related species - *C. tyzzeri* (mouse genotype I) and mouse genotype II
- Clinical signs:
  - Infection may ascend biliary tract in nude and SCID mice – chronic cholangiohepatitis with focal hepatic coagulative necrosis
  - SCID mice infected with *C. parvum* and treated with dexamethasone – develop neoplasia in stomach, duodenum, and ileocecal region
  - Immunosuppressed – persistent infection with weight loss and sticky stools
  - Suckling mice infected – grow more slowly and are less active
  - Immunocompetent adult mice – develop transient infections
  - Heavy infection – mucosal hyperplasia and foci of dysplasia

Transmission

- Once consumed, sporozoites invade gastrointestinal epithelium and develop into merozoites
- Merozoites develop into micro- or macrogametes and eventually into sporulated oocysts
- Oocysts shed in faeces as fully infective parasites

Isolation and Diagnosis

- Histological examination of GIT tissue:
  - *C. muris* - infects glands of the gastric mucosa (relatively nonpathogenic):
    - gastric gland dilation and lymphoid infiltrate

    - *C. parvum* (mice also susceptible to bovine genotype) – infects small intestinal epithelium (marginally pathogenic), heavy infections cause:
        - blunting and fusion of intestinal villi
        - crypt hyperplasia
        - lymphocytic-plasmacytic infiltration of underlying lamina propria
    - *C. parvum* in athymic nude mice – hepatobiliary tree and pancreatic duct infection
    - Routine hemoxylin-eosin staining of histological sections of the gastrointestinal mucosa
    - Differences in size and tissue location – species differentiation
- Faecal floatation
- PCR

Prevention and Control

- Infection in immunocompetent animals and humans is self-limiting
- Not susceptible to chemotherapeutics
- Oocysts remain infective in chlorinated water and are resistant to common disinfectants
- Steam sterilization, pasteurization, and ethylene oxide exposure for 24hrs – inactivate oocysts
- Rederivation (through caesarean section or embryo transfer into pathogen free dams) and barrier maintenance

Reading

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