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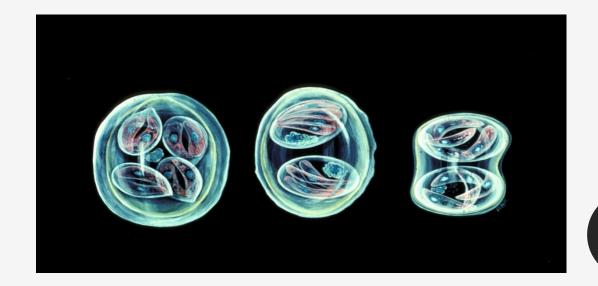
Coccidiosis

Smaro Sotiraki VRI HAO Demeter, Greece

Apicomplexan zoite

Polar ring Conoid -Pellicle-Micronemes Rhoptry-Micropore-Golgi body Nucleus-Endoplasmic reticulum Mitochondria Posterior ring

Phylum: Apicomplexa subclass Coccidia Families: Eimeriidae Cryptosporidiidae Toxoplasmatidae Sarcocystidae



protozoa/coccidia

Parasite species	Oocyst size	Host species	Site of infection	Pathogenicity
E. tenella	23 x 19 µm	chicken	caecum	high
E. necatrix	20 x 17 µm	chicken	small intestine, caecum	high
E. acervulina	18 x 14 µm	chicken	anterior small intestine	high
E. brunetti	26 x 22 µm	chicken	small and large intestines	high
E. maxima	30 x 20 µm	chicken	posterior small intestine	moderate
E. adenoides	25 x 16µm	turkey	small and large intestines	high
E. meleagrimitis	19 x 16 µm	turkey	anterior small intestine	high
E. meleagridis	24 x 18 µm	turkey	caecum	moderate
E. stiedae	35 x 20 µm	rabbit	liver	high
E. flavescens	30 x 20 µm	rabbit	intestines	moderate
E. intestinalis	26 x 18 µm	rabbit	intestines	moderate
E. truncata	20 x 14 µm	geese	kidney	moderate
E. zuernii	18 x 16 µm	cattle	small and large intestines	high
E. bovis	28 x 20 μm	cattle	small and large intestines	high
E. ovina	31 x 20 µm	sheep	small and large intestines	high
E. ahsata	36 x 24 µm	sheep	small intestine	moderate
E. ovinoidalis	24 x 20 µm	sheep	small and large intestines	moderate
E. arloingi	28 x 19 µm	goat	small and large intestines	high
E. ninakohlyakimovae	21 x 16 µm	goat	small and large intestines	moderate
E. debliecki	18 x 14 µm	pig	small intestine	moderate
E. leuckarti	55 x 38µm	horse	intestines	moderate
E. macropodis	25 x 13 μm	kangaroos	intestines	low
I. belli	35 x 10 µm	man	small intestine	moderate
I. canis	40 x 30 µm	dog	small intestine	moderate
l. ohioensis	25 x 16 µm	dog	small intestine	low
. burrowsi	12 x 10 µm	dog	small intestine	low
I. felis	40 x 30 µm	cat	small intestine	moderate
I. rivolta	25 x 16 µm	cat	small intestine	low
l. suis	20 x 18 µm	pig	small intestine	moderate

• Eimeria

Isospora

Important facts

hi specificity

- host
- tissue
- immunity

hi abundance

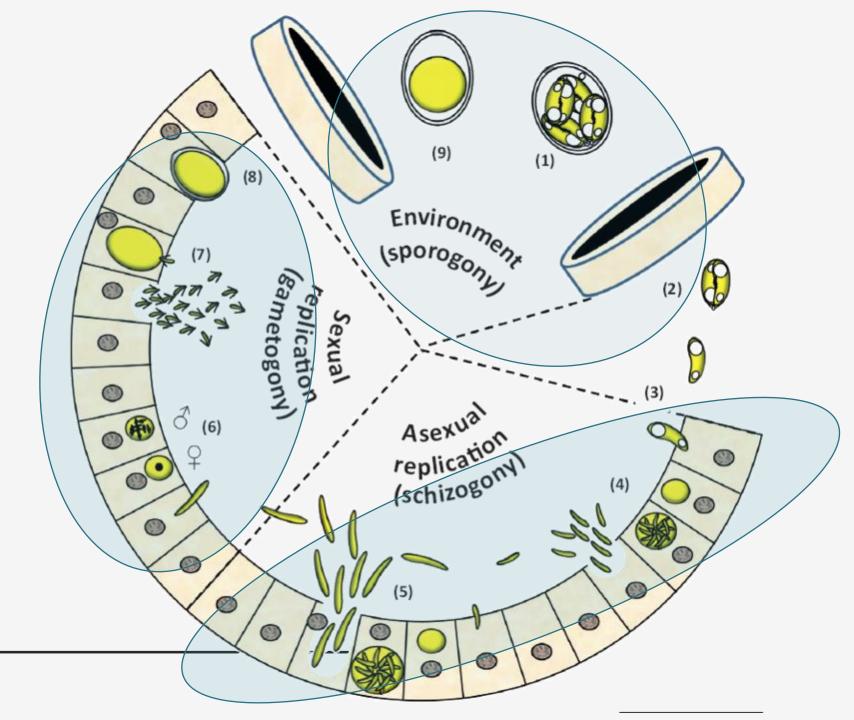
- direct life cycle
- rapid development
- resistance stages in the environment





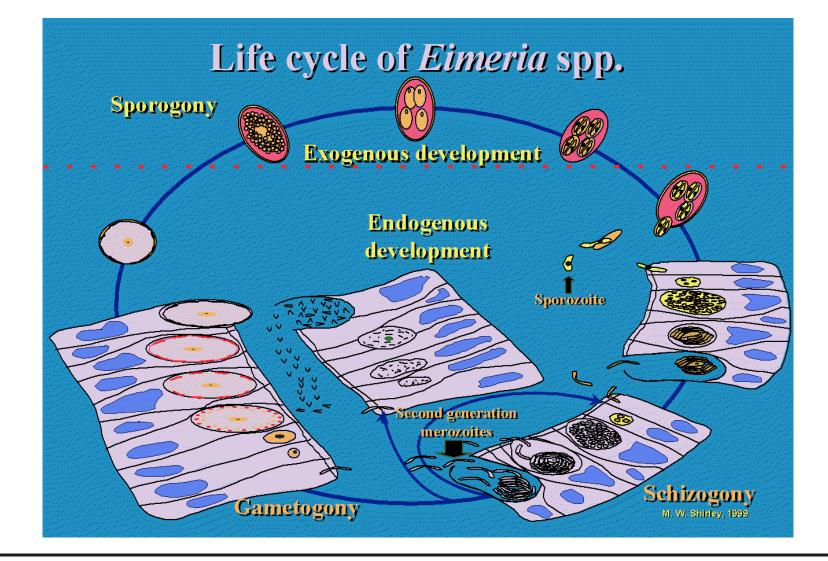
mainly production losses diarrhoea, dehydration, BW losses

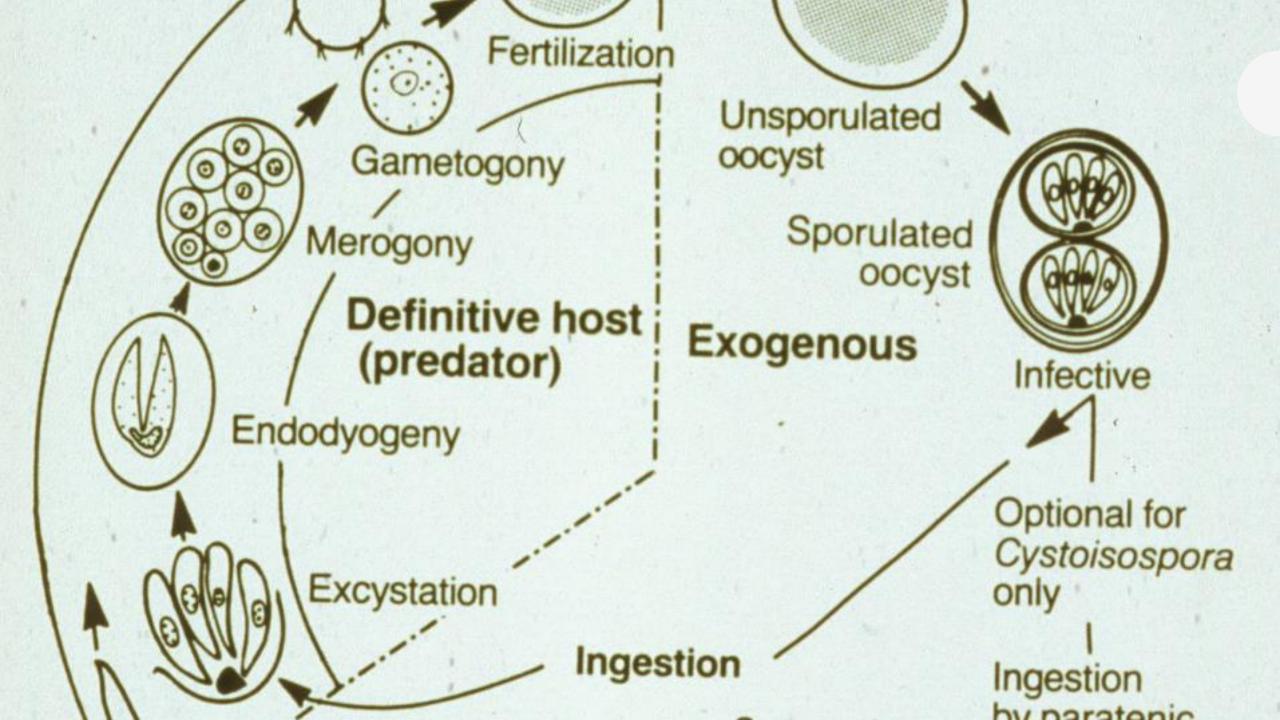
mortality depends on the species intention of infection

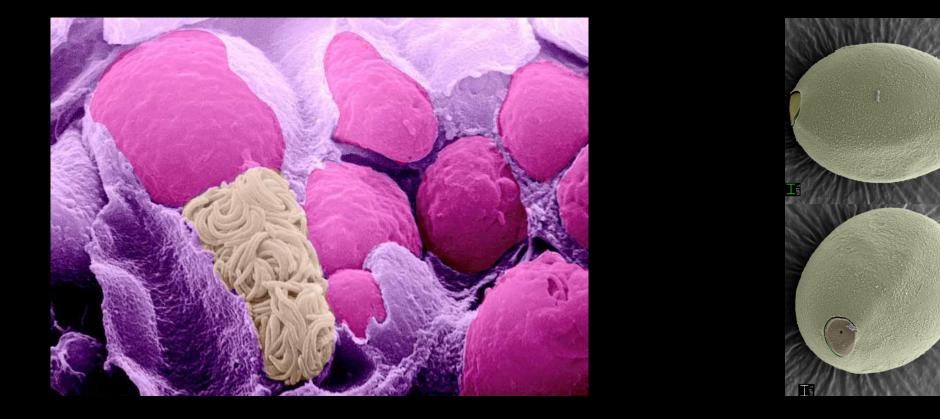


How much the epithelium is affected depends on the number of oocysts injected

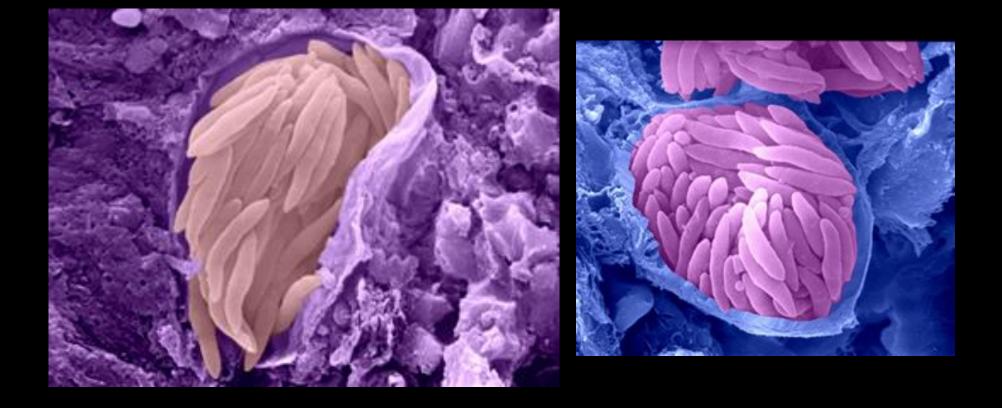
> Sporogony depends on temperature, humidify and oxygen which are offered in any farm conditions







MICRO/MACRO GAMETES - OOCYST

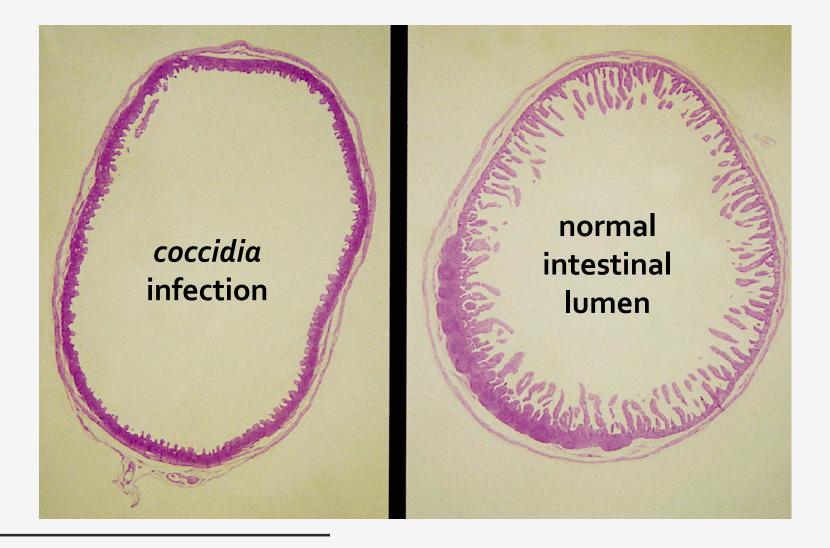


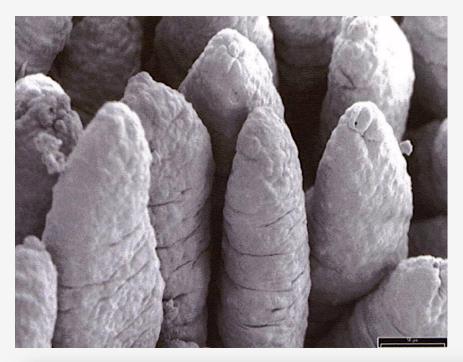
2ND GENERATION MEROZOITES

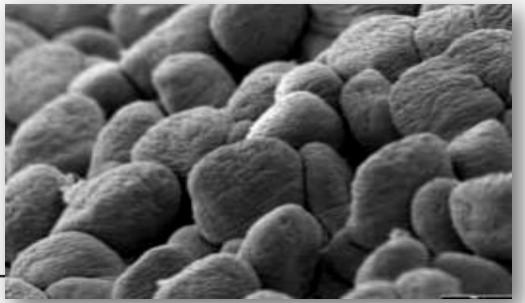
15 **Polar Rings** Micronemes < Rhoptry - Golgi Apparatus Nucleus н Mitochondrion

TROPHOZOITES

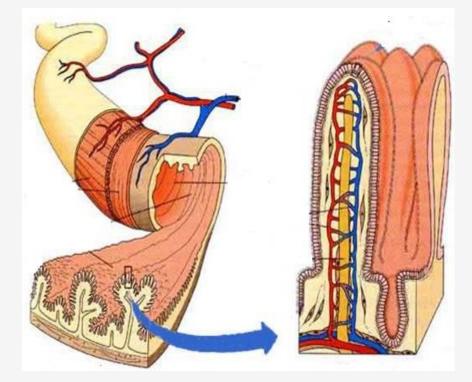
What do they do to the intestines ?

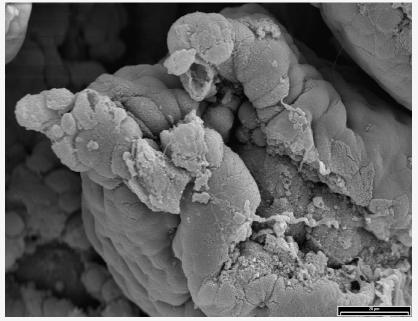






MUNDT, H.-C., S. MUNDT-WÜSTENBERG, A. DAUGSCHIES, A. JOACHIM (2005 & 2007):

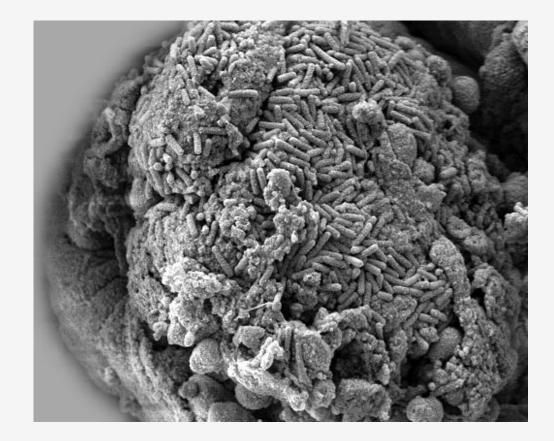


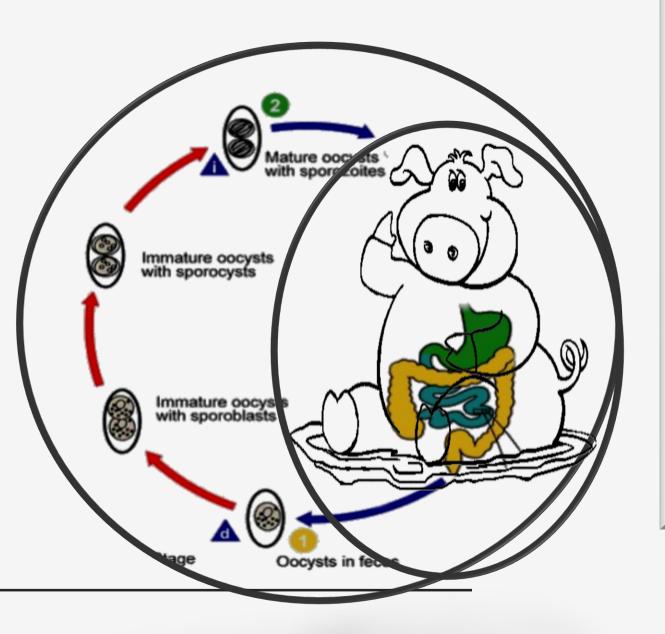


Re-epithelisation is not enough to get the mature physiological status

Secondary infections







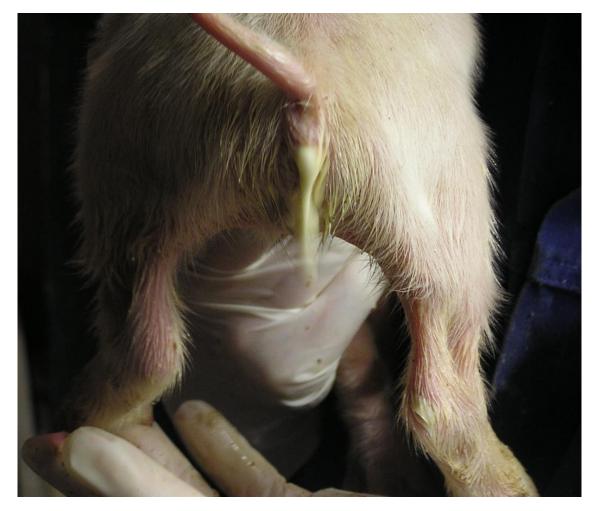
(Cystoisospora) Isospora suis is an important cause of diarrhoea in nursing piglets and an increasing problem throughout the world.

the infected piglets develop characteristic non-hemorrhagic diarrhoea at 7 to 14 days of age accompanied by depression and dehydration. The diarrhoea is yellowish to grey and initially pasty but becomes fluid after 2 to 3 days Epidemiology of the piglet coccidiosis in intensive farming systems

- *Isospora suis* causes diarrhoea in neonatal piglets
 - Clinical outbreaks of diarrhoea in the second week (8-15 days) of life
 - Seldom earlier and after weaning
- Excretion of oocysts takes place in waves two peaks at day 5-9 and 11-14 post infection
- Piglets which are infected soon after birth have increased and prolonged oocysts excretion and severe diarrhoea as compared to older piglets

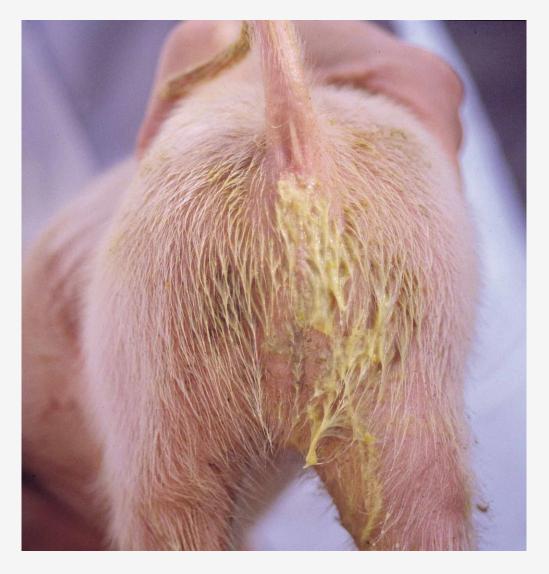
Epidemiology of the piglet coccidiosis in intensive farming systems

- The piglets develop an age related resistance to infection which results in lower excretion rates and less pronounced diarrhoea in older animals,
- In a farrowing facility, morbidity can be high, but mortality is usually low except for cases with secondary bacterial infections
- Not all litters or piglets within a litter in a farrowing facility are affected equally
- Sow does not play an important role in transmission of *Isospora suis* in the farrowing pen









High morbidity - Low mortality



Uneven growth / no mortality







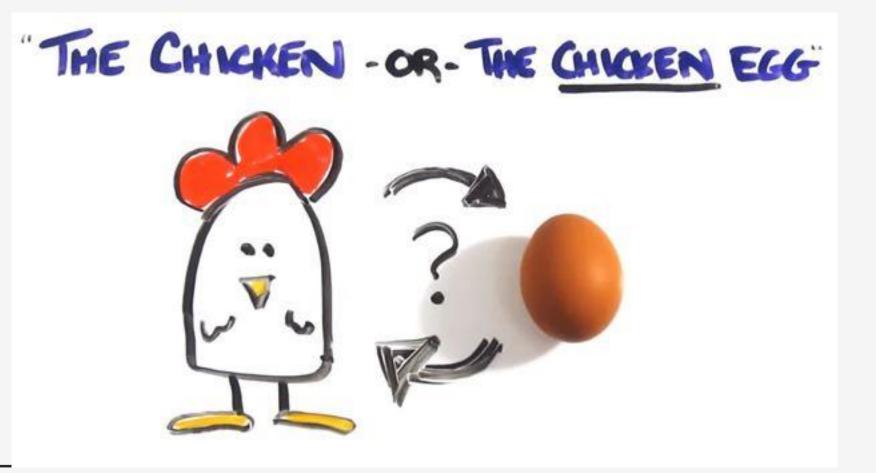
Country	Prevalence Information	Reference
Belgium	80% infected farms 33% infected litters	Leten (2002)
Netherlands	72% Infected farms 36% Infected litters	Hollanders (1993)
Spain	85% infected farms	Enric (2000)
Germany	53% infected litters	Meyer (1999)
Italy	68% infected farms	Vezzoli (2002)
Denmark	80% infected farms	Larsen (2002)
Mexico	70% infected farms	Iglesias (2000)
Brazil	45% infected farms	Ristow (2002)

PIGLET COCCIDIOSIS IS GLOBAL IN INTENSIVE FARMING

the spread of *I. suis* infection is strongly related to environment contamination

once *I. suis* has established itself on a farm, the infection is probably maintained through piglet-to-piglet transmission via contaminated farrowing pens

^-



sow plays a role in introducing the parasite in to the farrowing crate either by an undetected active parasitism or passive transportation of parasites'

oocysts





Coccidios increase mortality in mixted infections

Complication and mixed infection can cause hemorrhagic diarrhoea and mortality

Necrotic enteritis + coccidiosis = high mortality (Exp. 30 %)



Haemorrhagic gut content

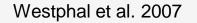
Mucus

Gut membrane

Necrotic mucosa

Blood, mucus and epithelium

Mundt et al. 2009



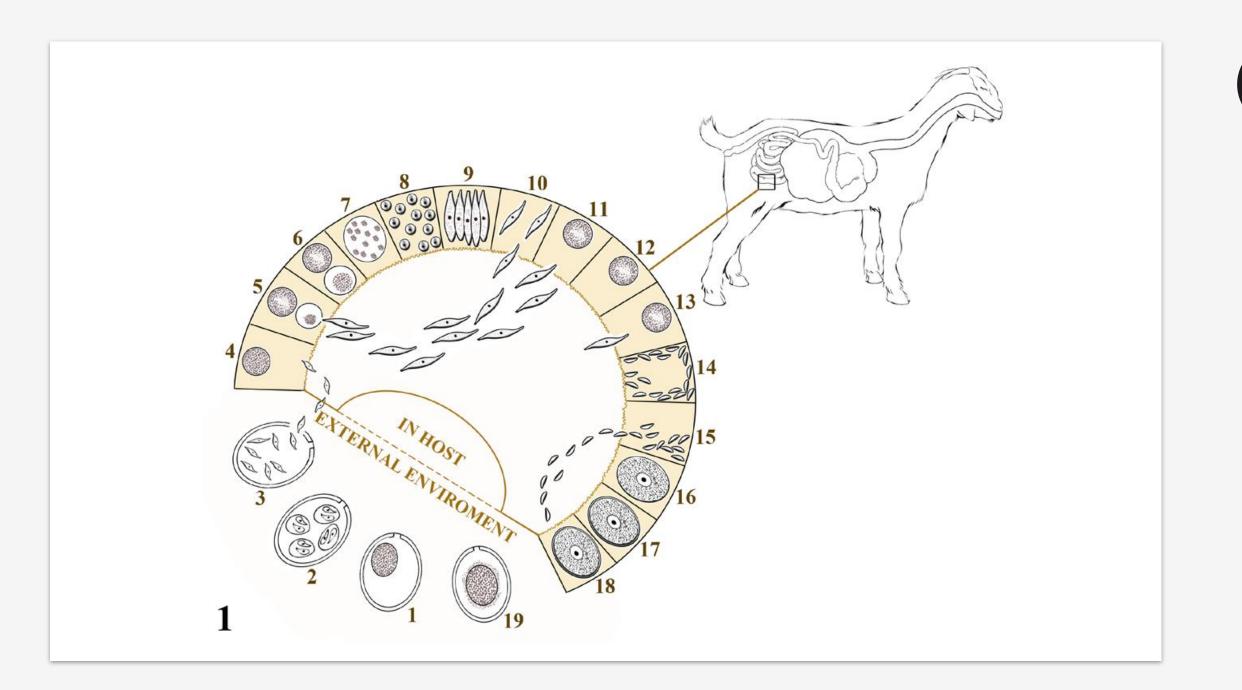
interactions

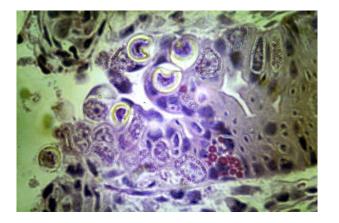
Nursing piglets

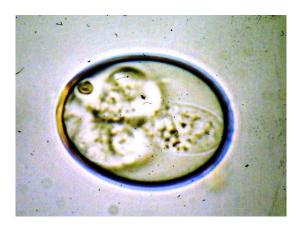
- Cryptosporidium/Giardia
- Isospora suis
- Rotavirus
- E. coli

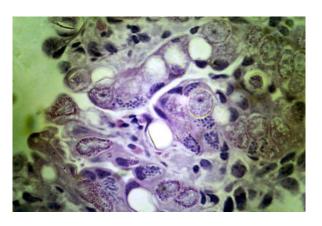
Fattening pigs

- Lawsonia intracellularis
- Brachyspira hyodysenteriae (swine dysentery)
- Salmonella
- PCV



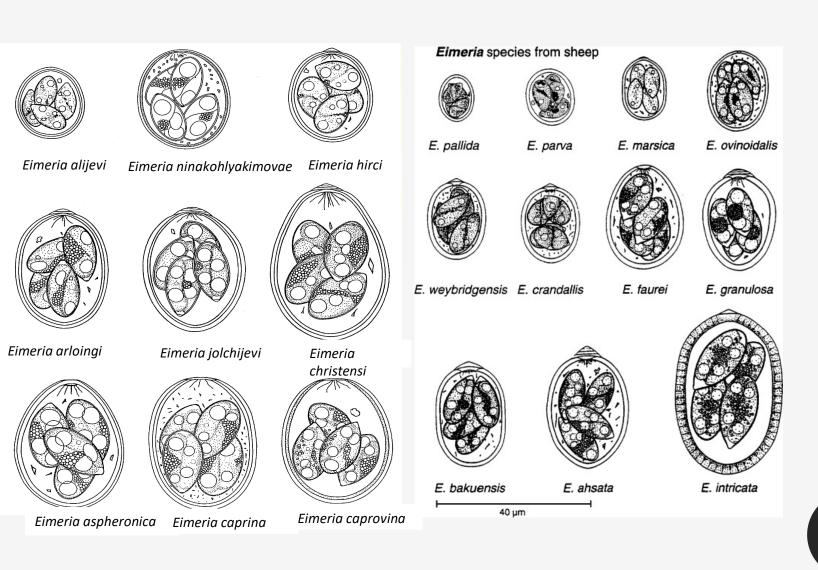




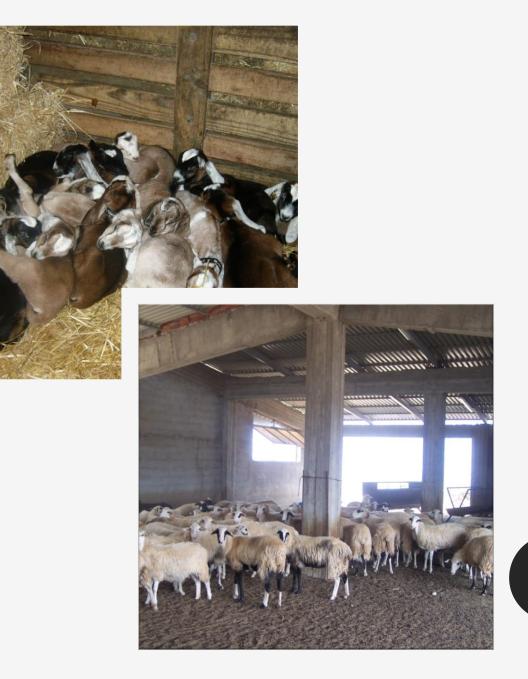


- Coccidiosis is caused by *Eimeria* spp. (as with other ruminants)
- Eimeria spp. are host-specific
 - Sheep species do not infect goat or vice versa
- 15 *Eimeria* spp. have been described in sheep
- 9 species of *Eimeria* are commonly found in goats
- Not all species are pathogenic
- Oocysts of sheep coccidia are morphologically identical to the equivalent species found in goats

Goat Coccidia Species	Equivalent Sheep Species
E. ninakohlyakimovae	E. ovinoidalis
E. hirci	E. crandallis
E. christenseni	E. ahsata
E. arloingi	E. bakuensis
E. jolchijevi	E. granulosa
E. aleijevi	E. parva
E. apsheronica	E. faurei
E. caprina	
E. caprovina	E. caprovina
E. pallida	E. pallida
	E. marsica
	E. weybridgensis
E. kocharli	E. intricata
E. tunisiensis**	E. ahsata
E. punctata **	
E. capralis	
E.masseyensis	
E. charlestoni	



- All ages of σηεεπ/goats are susceptible to infection
- Adult goats are usually resistant to disease but continue to excrete small numbers of coccidia
- Kids are particularly susceptible
- Colostrum provides passive protection during the first few weeks of life
- ✓ E. ovinoidalis and E. crandallis **★** most pathogenic
- The main pathogenic species found in the large intestine are:
 - E. ninakohlyakimovae
 - E. caprina
- ✓ Disease occurs when large numbers of oocysts from <u>pathogenic</u> species are ingested by parasite-naive or nonimmune kids
- Clinical signs / Pathology: diarrhoea, dehydration, epithelial hyperplasia, inflammation, villus atrophy, mucosal collapse



IMMUNITY

- Low levels of infection stimulate protective immunity
- Developed immunity is strong but <u>species-specific</u>
- Immunity in young kids thus depends on:

- Nutritional status
- Colostral intake
- Body condition and age (but NO age-related immunity)
- Low level exposure to all species of coccidia



Epidemiology

- Heavily contaminated environments predispose to disease
- Low levels of infection stimulate protective immunity
- Management factors that may precipitate disease in young kids include:
 - Poor hygiene
 - Overcrowding
 - Weaning age and condition
 - Mixing of different age groups
 - Changes in diet
 - Inclement weather
 - Transport



What are the main risk factors for coccidiosis?

High oocyst challenge

Faeces in water or food supply

Dirty conditions

Heavily stocked

Older lambs previously infield

Susceptible lambs

4-12 weeks old

No previous exposure to that species of coccidia

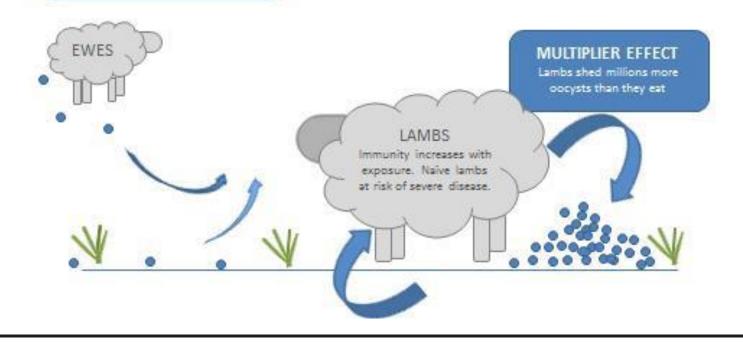
Stress due to cold wet, weather

Stress due to poor nutrition

Which animals are the target?



- Ewes pass low numbers
- Some overwinter on pasture

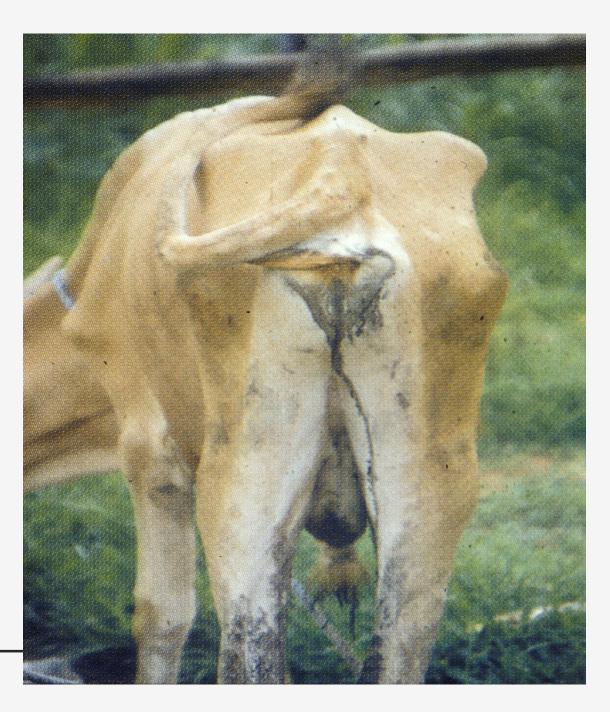


SHEEP MANAGEMENT SYSTEMS

- **1.** Spring lambing, production at grass
 - Lambing February/March/April
 - Weaning in June-August at 3-5 months of age
 - Intensive or extensive grazing
 - Lowland or Upland
- 2. Winter/Spring Lambing, early weaning, finishing indoors
 - I. Weaning at birth
 - II. Weaning at 6-8 weeks of age, raised and finished indoors
- 3. Summer/Autumn lambing, production at grass, weaning, finishing indoors



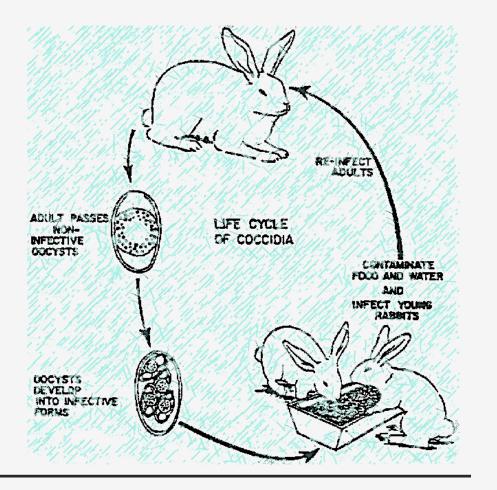


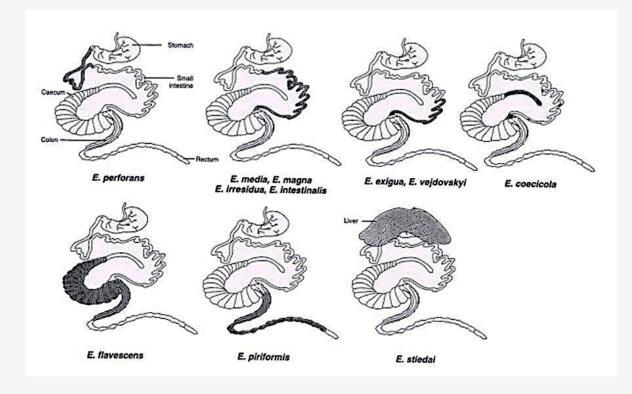






Coccidiosis in rabbits





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<u>Eimeria stiedai</u>

hi research activity in poultry



Basic research

- -omics approaches
- mainly targeting the apical complex and organels such as paired rhoptries, micronemes and dense bodies which are involved in the cell invasion and protein production
- Biochemical pathways
- Studies on CD4/CD8 lymphocytes and cellular immunity pathoways

Applied research

• Test of antococcidial

Immunity







Efforts in piglets



No so much information in sheep, goats, rabbits Necropsy (at large)

Oocyst presence in faeces (and further species identification)

Farm history

Diagnosis

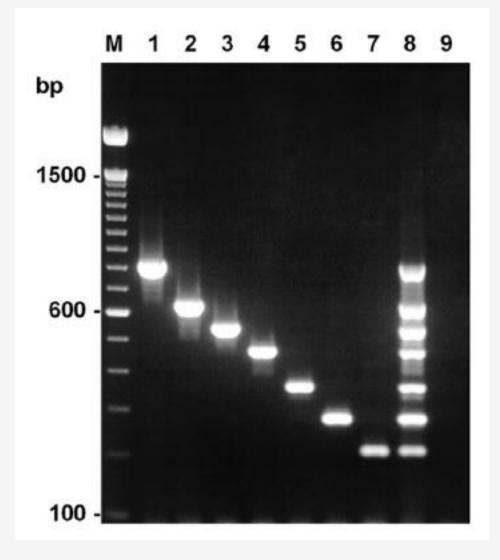


Figure 2.5 Agarose gel electrophoresis of multiplex PCR products using DNA samples of *E. acervulina* (lane 1), *E. brunetti* (lane 2), *E. tenella* (lane 3), *E. mitis* (lane 4), *E. praecox* (lane 5), *E. maxima* (lane 6), *E. necatrix* (lane 7), a mixture of the seven *Eimeria* species (lane 8) and a control with no starting DNA (lane 9). Molecular size markers (lane M) in base pairs are indicated on the left *Reproduced from Fernandez et al.* (2003b) with permission from Cambridge University Press.

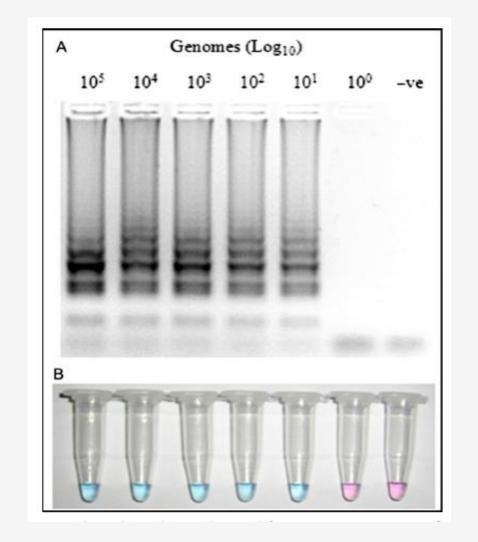
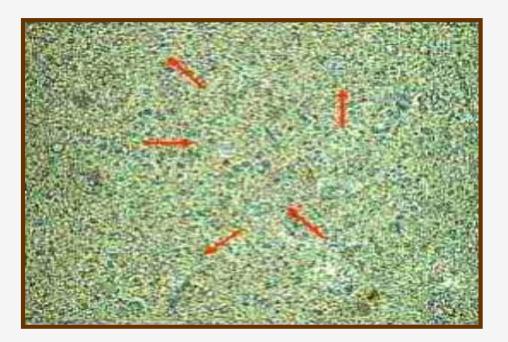
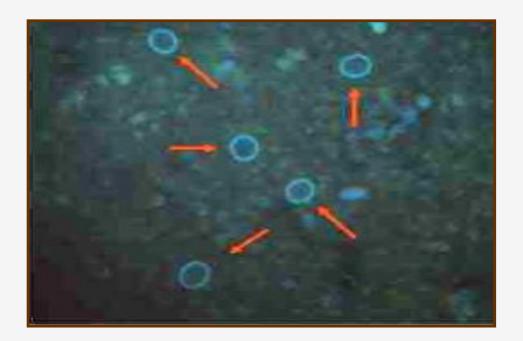


Figure 2.7 Loop-mediated isothermal amplification (LAMP) specific for *E. tenella*. Application to a purified genomic DNA dilution series revealed a limit of detection of between 1 and 10 *E. tenella* genomes using agarose gel electrophoresis (A) or hydroxynaphthol blue as a visual indicator (B, blue: positive, pink: negative). –ve, no template negative control.

Difficult to diagnose





- biosecurity (difficult out doors, source of infection)
- environmental measures
- disinfenctants (Ammonium hydroxide, ammonium salt and sodium hydroxide (Oocide, Antec International Ltd, UK), cresol (Neopredisan, Menno Chemie, Norderstedt, Germany) formol 37% and sodium dodecylbenzene sulphonate 12%)

Management

anticoccidial

toltrazuril /diclazuril – resistance ??

Heavy use for years

not licensed in all species

new?

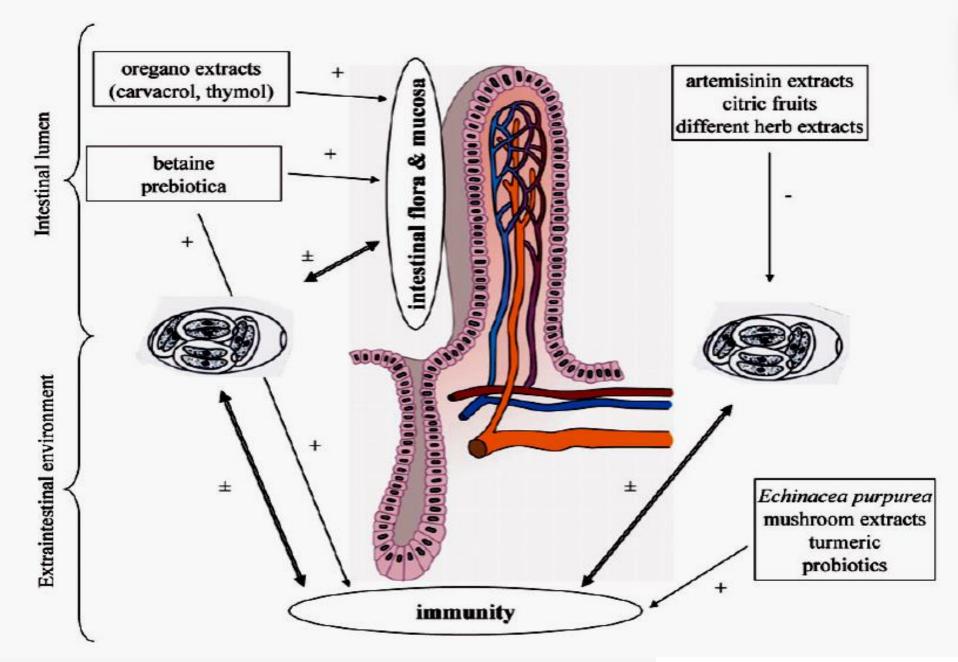
residues in meat?

Nutraceuticals

Probiotics ?

(ensuring `gut health' boosting immunity)





H.W. Peek & W.J.M. Landman (2011)



vaccines

- Hi activity in the poultry sector
- Effort in pigs ??

✓ several gaps still✓ coordinated effort

CONCLUSIONS

✓ *Reduce chemical drug dependencies*

 \checkmark Incorporate new diagnostic tools – new therapeutic/prevention schemes





thank you for your attention

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