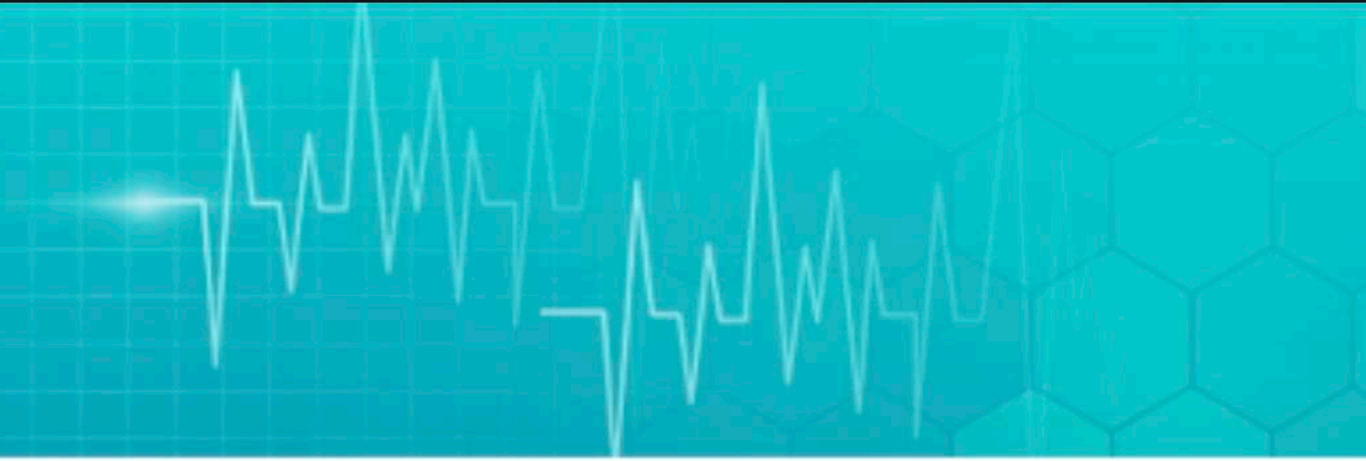




# Epidemiology and pathogenesis of GI parasitism

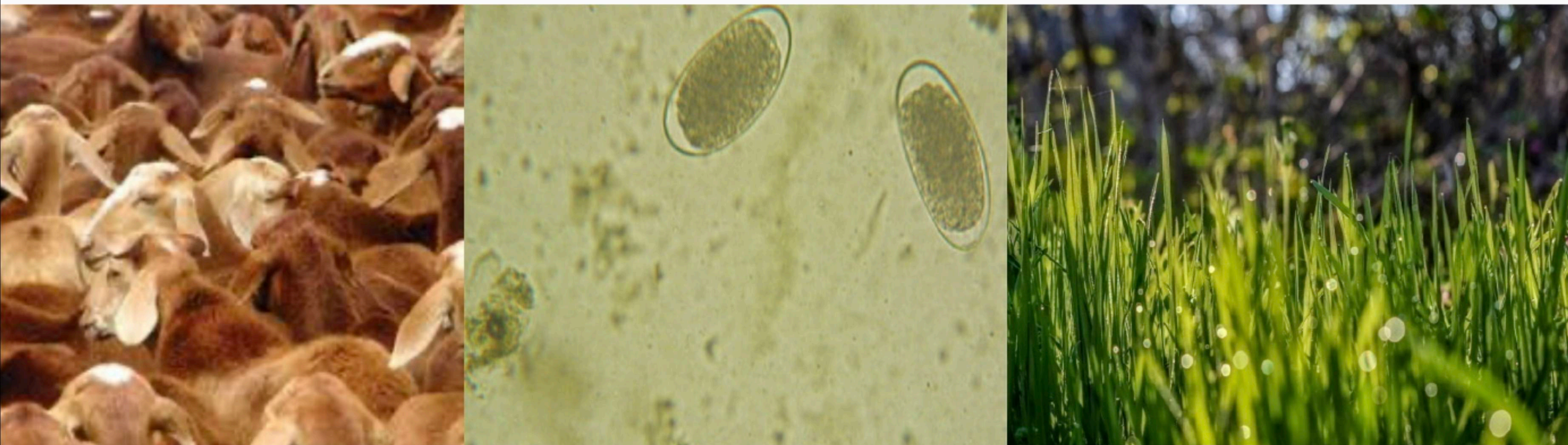
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## Epidemiology and pathogenesis of GI parasitism

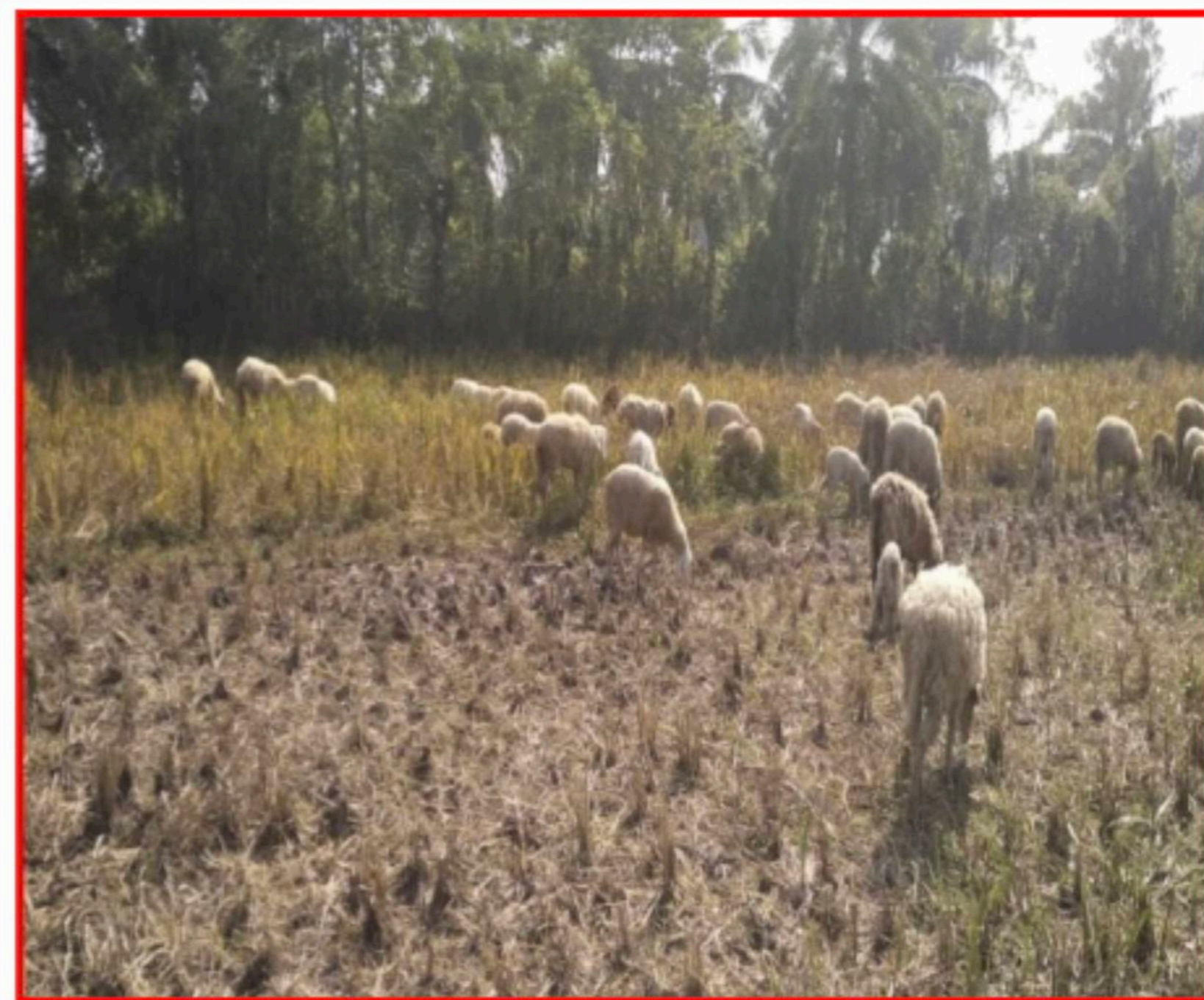
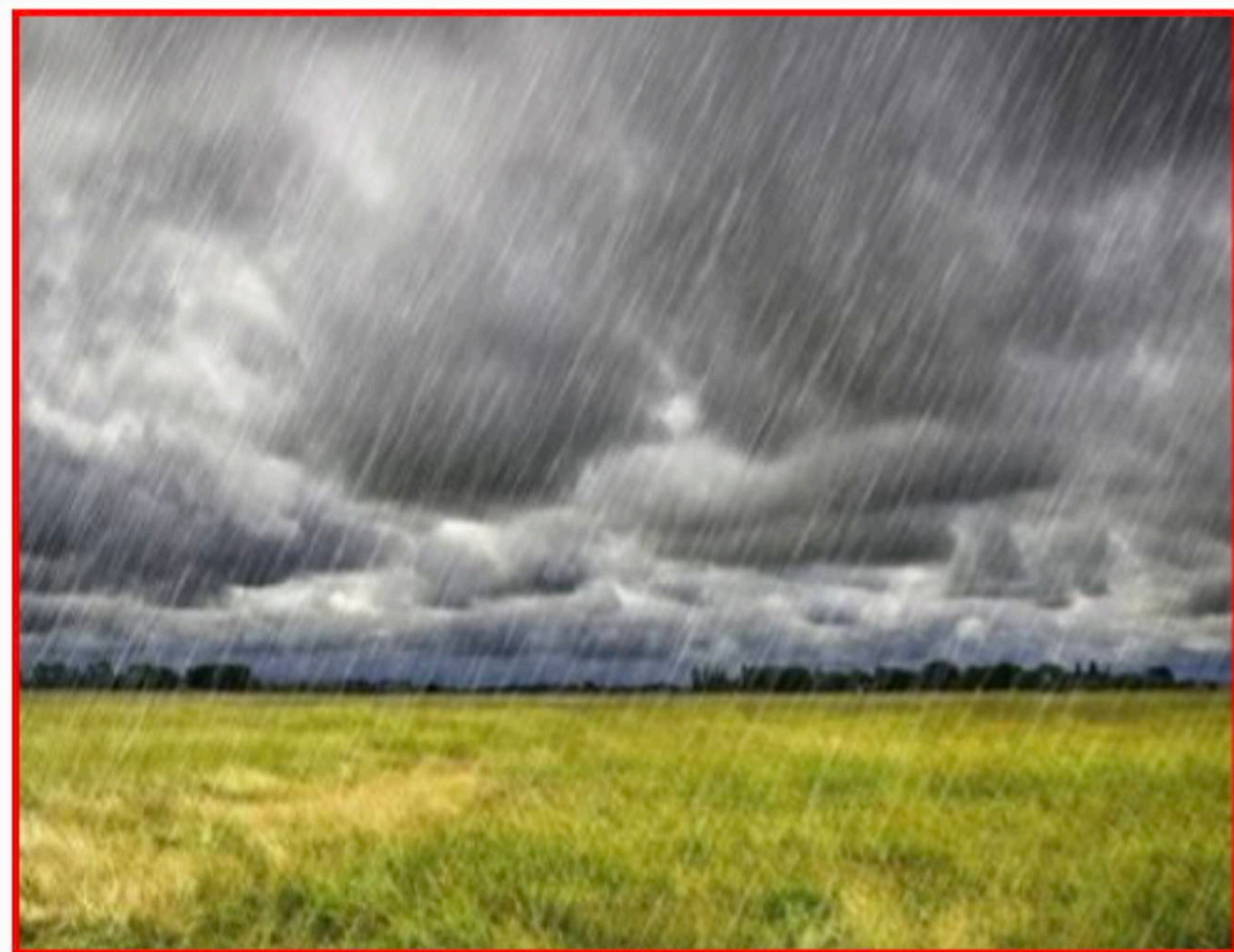
- ▶ 1. Factors governing PGE epidemiology
- ▶ 2. Pathogenesis of GI nematodes
- ▶ 3. Clinical signs in GI parasitism





## Epidemiological intelligence on PGE

- ▶ Study of parasitic diseases and disease - causing agents at the population level.
- ▶ It predict to characterize the pattern of distribution, occurrence of disease and the factors responsible for these patterns.
- ▶ A good understanding on factors that drives parasite biology is essential to adopt effective control strategy





## Factors governing PGE epidemiology

### Host & Parasite



Worm species  
Host factors/Age  
Immune status  
Genetics  
Nutrition  
PPR  
Hypobiosis

### Environment



Rainfall  
Climate/Temperature  
Moisture  
Sunlight  
Microclimate

### Management



Type of management  
Grazing pattern  
Drug dosing

## Host factors

- ▶ **Goats do not develop age-related immunity**
- ▶ Young animals more susceptible to GIN (< 6 months)
- ▶ Young adults (1-2 Y) develop varying degree of immunity
- ▶ Adults are generally immune to parasites, but stress can reduce immunity
- ▶ **Lactating animals are more vulnerable** & source of infection to young ones



# Parasite factors

## Worm species

- ▶ Knowledge on type of parasite species prevalent
- ▶ Mode of transmission and their economic
- ▶ importance is essential
- ▶ India - common GIN includes

*Haemonchus contortus*,  
*Trichostrongylus colubriformis*,  
*Oesophagostomum columbianum*  
*Strongyloides papillosus*





# Host defense mechanisms

## Types of immunity

### ▶ Innate

inherited (rare)

### ▶ Acquired

Acquired during life results from exposure to infection



## Self – cure phenomenon

### ▶ Self defense mechanism

▶ Adult worms are expelled spontaneously when there is continuous re infection over a short period

▶ IgE mediated hypersensitivity reaction

▶ noticed in Barder's boile worm infection



## Nutritional status

- ▶ Lack of grazing/ poor nutrition –more susceptible to GIN
- ▶ Deficiency in dietary protein – lowers the immune response to GIN
- ▶ Animals in good plan of nutrition showed
  - Decreased FEC
  - Good antibody level &
  - Increased goblet cells in small intestine
- ▶ Thin animals are more susceptible







# Hypobiosis

- ▶ Considered as an evolutionary adoption
- ▶ It has importance in GIN epidemiology because
  - ◆ Worms survive and maintain its population during harsh climatic extremes
  - ◆ Resume development in favourable conditions responsible for clinical disease
  - ◆ Major source of pasture contamination during monsoon





## Peri Parturient Egg Rise (PPER)

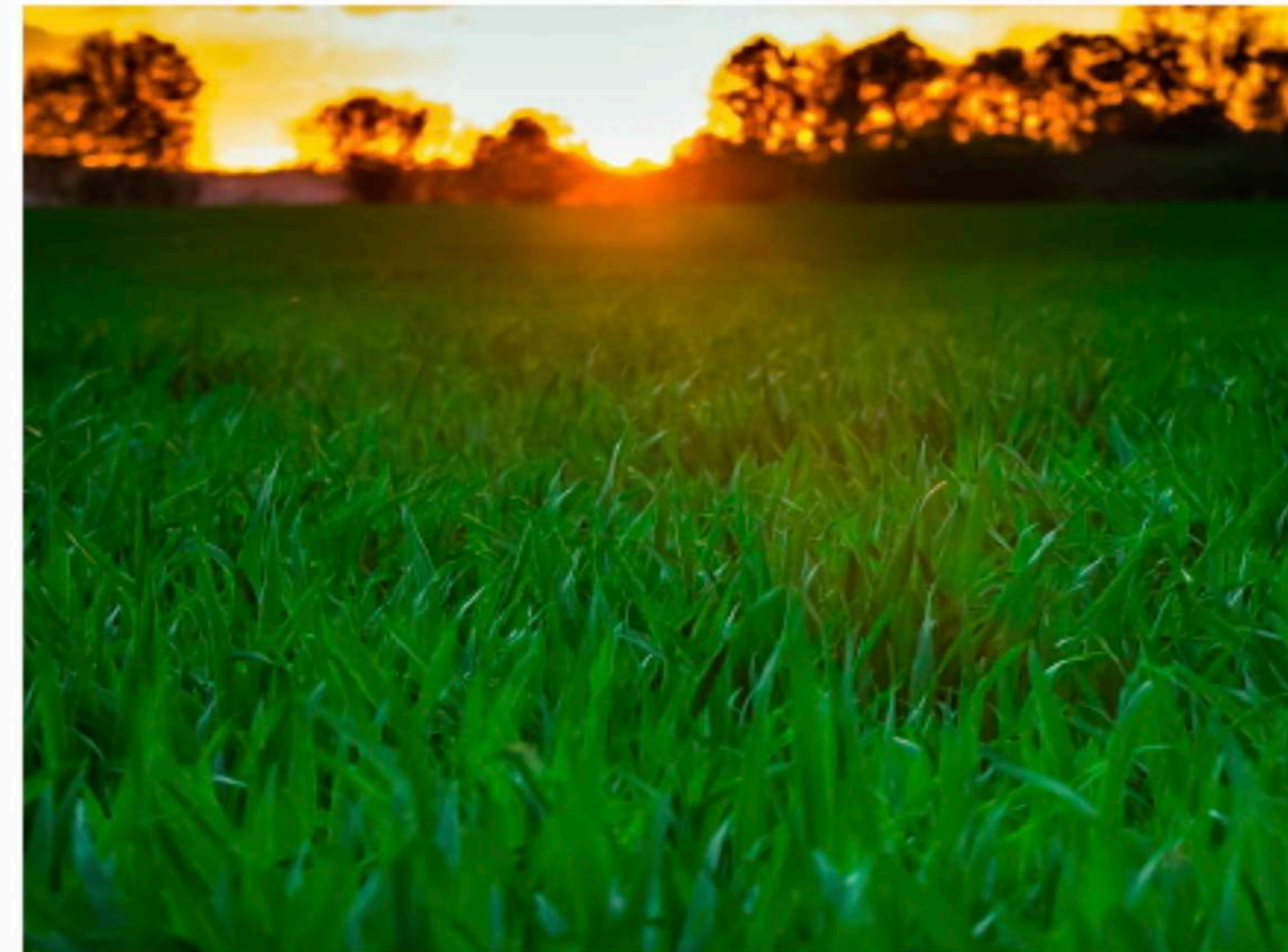
- ▶ PPR is important in parasite survival
- ▶ Large numbers of eggs passed onto the pasture at the same time as the numbers of new **susceptible** hosts also increases
- ▶ Periparturient relaxation of immunity due to prolactin
- ▶ **Exacerbation of PPER** in
  - Inadequate/poor nutrition in animals in late pregnancy
  - Lack of vegetation in pasture
  - Deficiency of minerals





## B. Environmental factors and Nematode survival

- ▶ **Factors – Climate, seasons, temperature, humidity, rainfall etc.,**
- ▶ **Influences development and survival of larva on the pasture and migratory behaviour**
- ▶ **Bio climatograph – helps to predict the occurrence of disease**

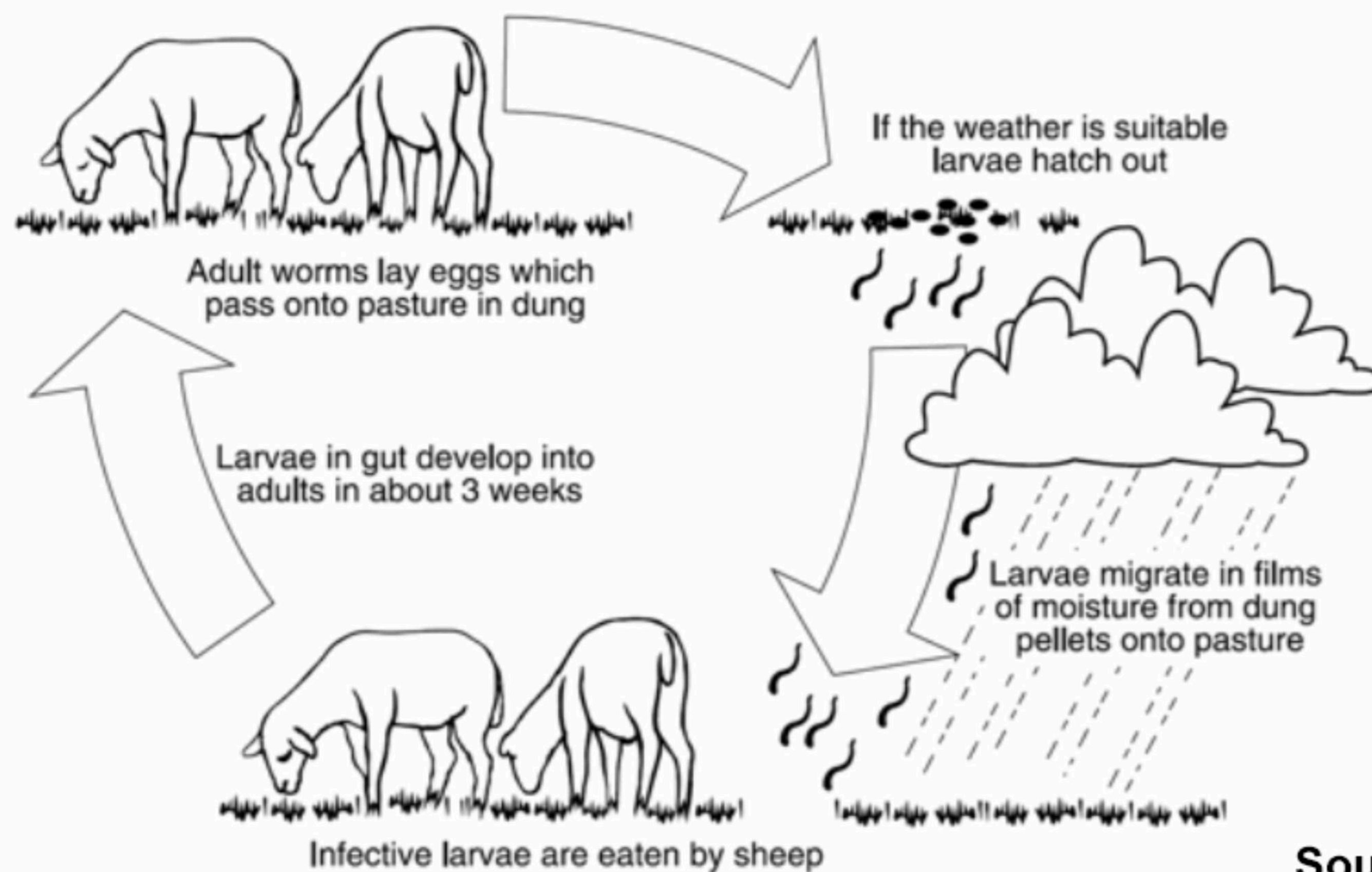




# Free living stages are of 2 stages, largely dictated by climate

- ▶ The first stage is egg development to infective L3
- ▶ The second stage is L3 survival

Life cycle of sheep worms



Source : Jennifer Cotter, Agriculture and Food



## Bionomics of infective larvae

Under Indian tropical conditions,

- ▶ Optimum temperature for egg to infective larvae

*Haemonchus contortus* - 25- 37°C, highly susceptible to *cold and desiccation*

*Trichostrongylus colubriformis* - 20-33°C, susceptible to sub freezing winter

- ▶ Survival of infective larvae

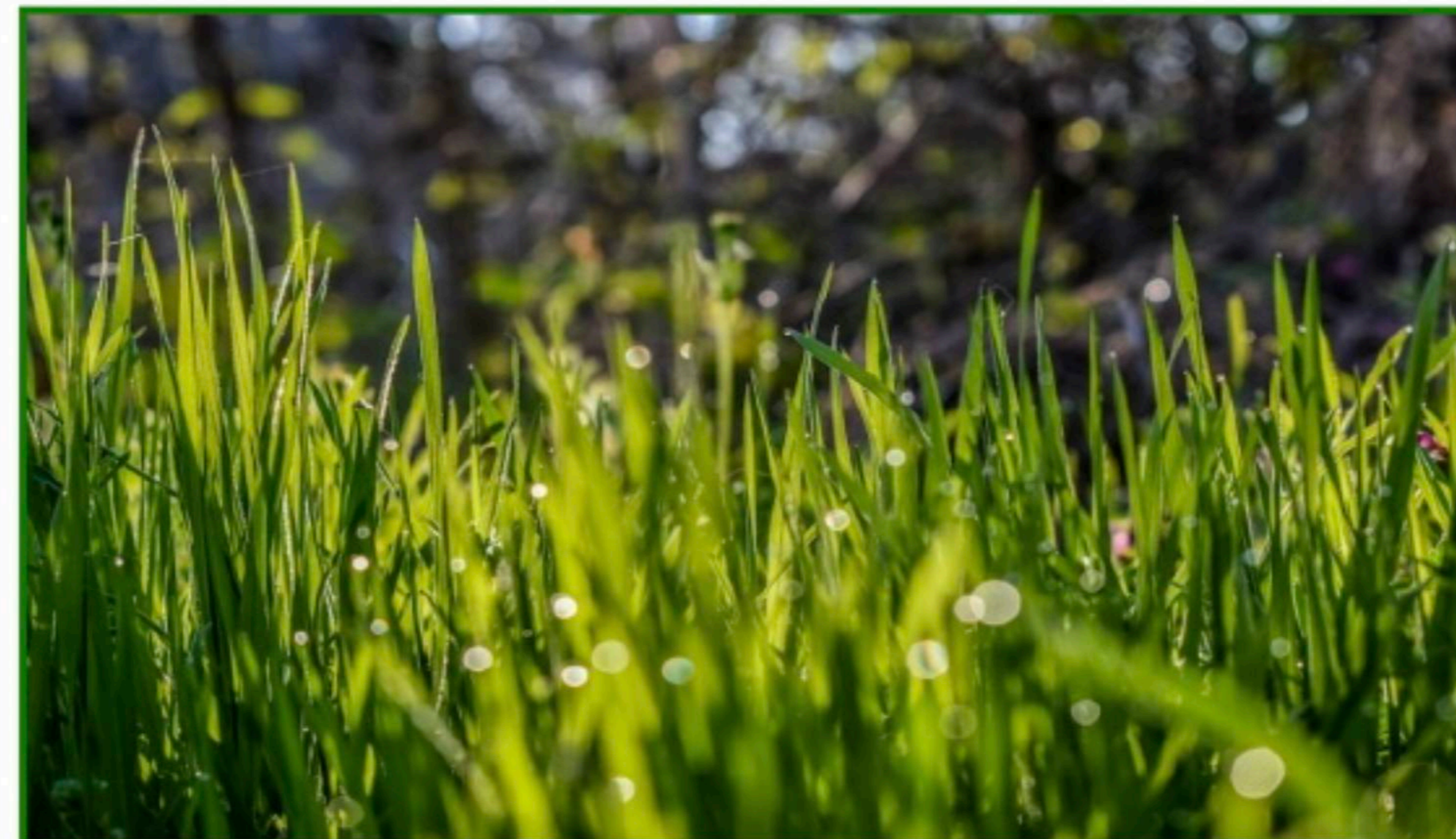
*H. contortus* - warm, moist condition with adequate rainfall

*T. colubriformis* - warm/cool conditions with moist environment

- ▶ Survival period- 8 to 9 weeks in monsoon Up to 4 weeks in summer

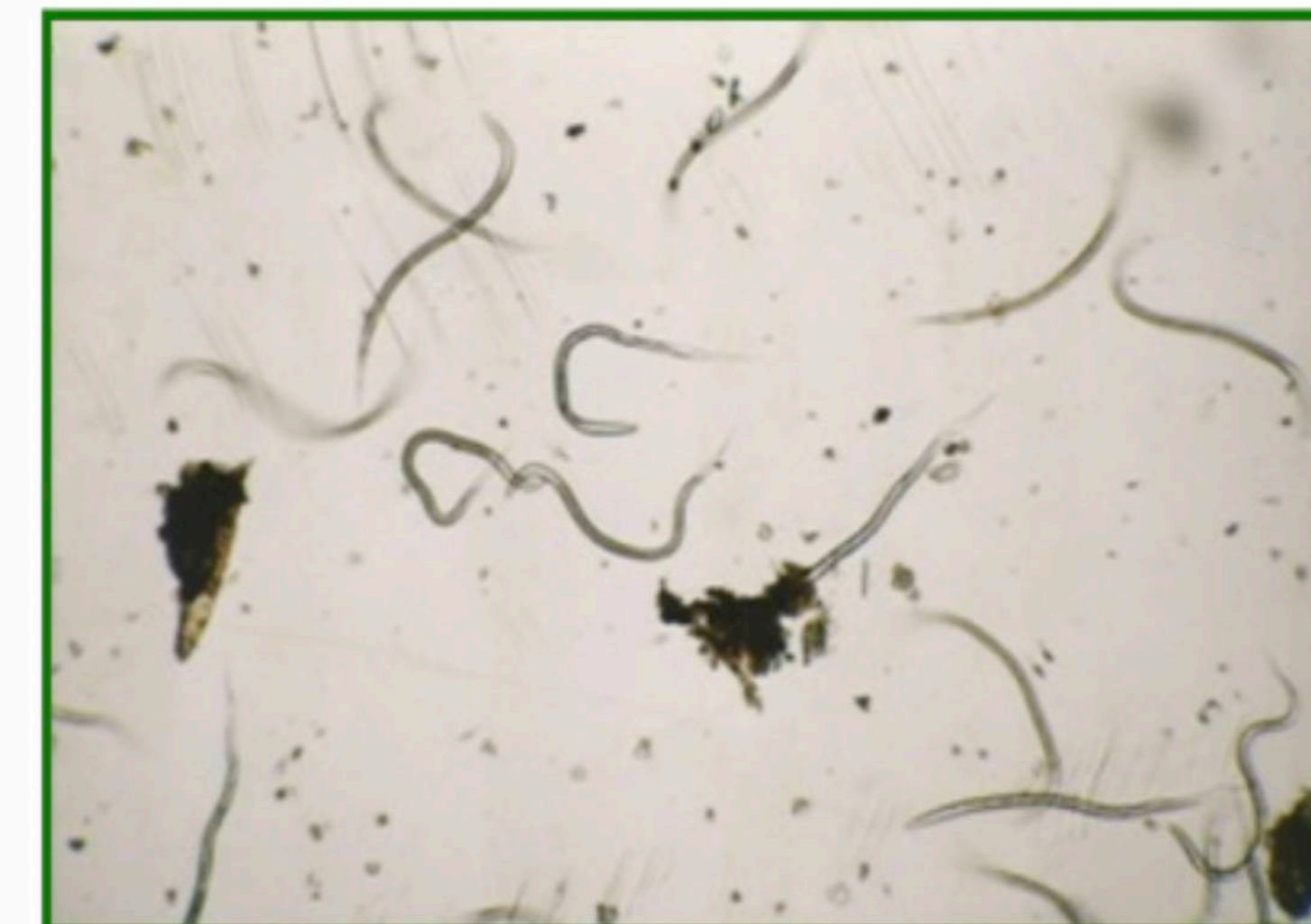
Migration - horizontal - up to 50 cm

vertical - up to 10 cm





- ▶ Heavy **morning dews** or moisture will allow migration L3 up on the grass blades, enhancing infectivity of the pasture
- ▶ Hot, sunny days will drive the **L<sub>3</sub> down** to soil level, thus reducing infectivity
- ▶ Pasture larval burden(PLB) - **higher in monsoon** (July to November) period
- ▶ Over dispersion in FEC - **>1000 EPG** (August to November)
- ▶ Construction of **bioclimatographs** in a geographical area will explain pattern of GIN epidemiology
- ▶ In India, **Rainy and Autumn seasons** (June to December) are best suited for survival and migration of exogenous stages and higher GIN in small ruminants





## C. Managemental practices vs GIN

Grazing system and pasture management

**Factors enhances the risk of infections are :**

- ▶ Lack of quality pasture (high risk pasture)
- ▶ Resting period –not adequate
- ▶ Non practice of rotational grazing
- ▶ Community grazing (pasture sharing by many animals)
- ▶ Grazing close to the ground and manure
- ▶ Early morning grazing in winter/rainy season
- ▶ Overstocking causes more egg deposits





## System of management and deworming

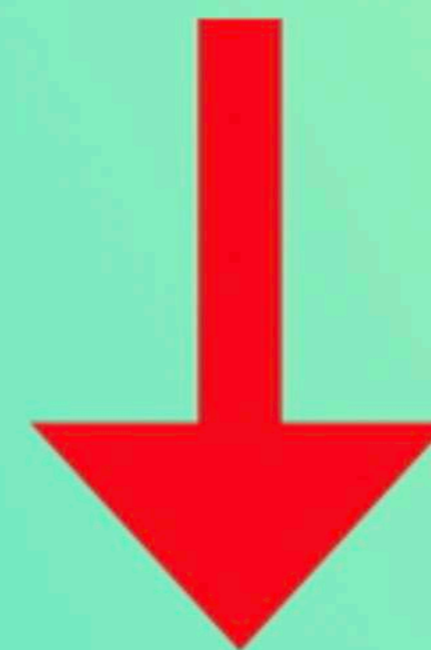
### Type of rearing –

- ▶ Extensive & semi intensive system - increases the risk of infection
- ▶ Intensive (stall fed) system – No source of infection



### Anthelmintic dosing

- ▶ Periodical deworming
- ▶ Improper deworming
- ▶ Blanket treatment



**Drench failure**

**Anthelmintic resistance**



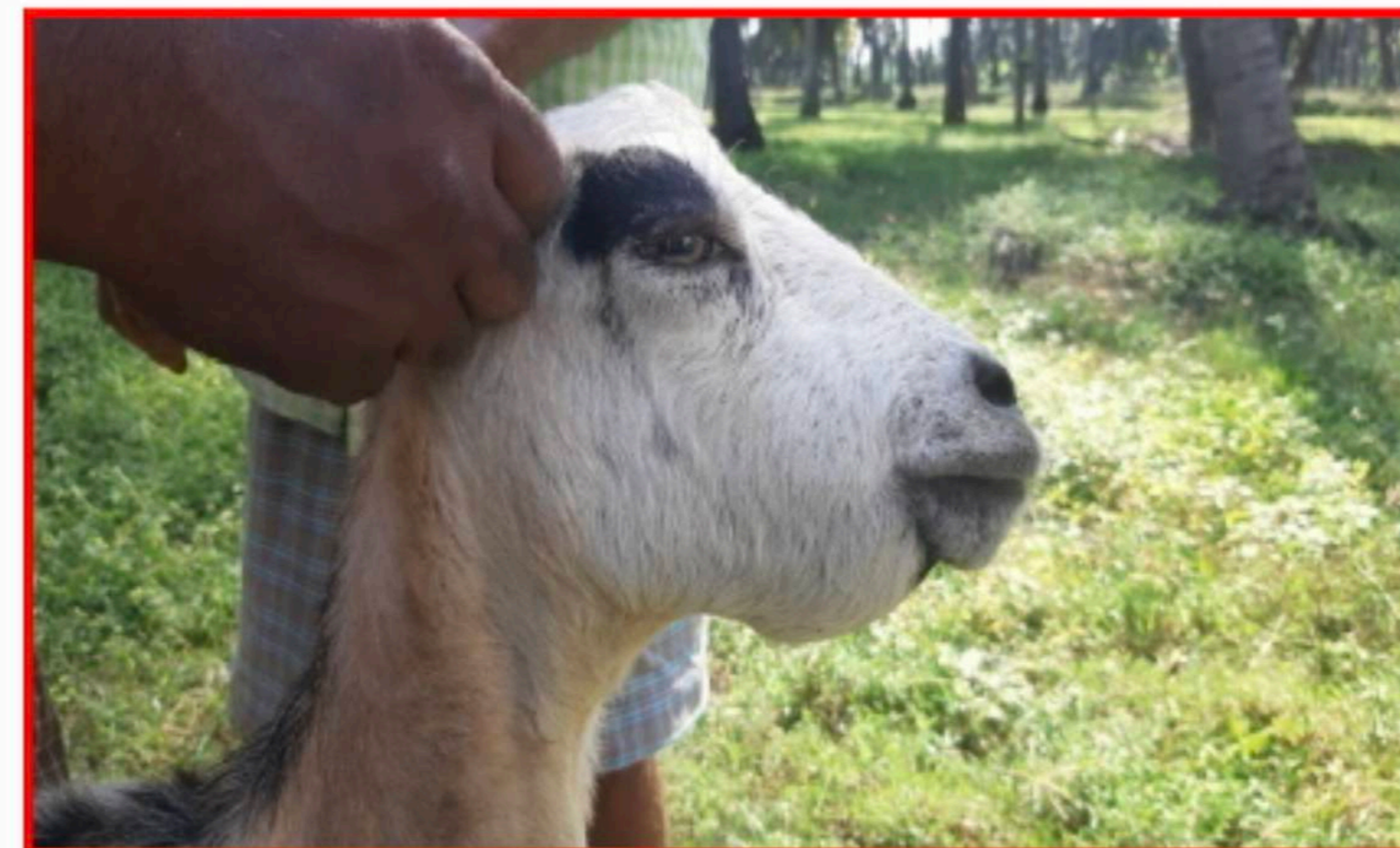


# Pathogenesis of Gastro Intestinal Nematodes



## ***Haemonchus contortus* (Haemonchosis)**

- ▶ **Abomasal nematode**
- ▶ **L<sub>4</sub> and adults - Blood feeders → Anaemia**
- ▶ **Apparent after 2 weeks of infection**
- ▶ **Diarrhoea is not a sign** of pure infection
- ▶ **Responsible for more production losses in tropics**





- ▶ **Type of infection depends on intensity of infection**

- ▶ **Hyper acute-  
Death within 1 wk of heavy infection  
without signs**

- ▶ **Acute –  
haemorrhagic anaemia, dark coloured  
faeces, bottle jaw, weakness, death**

- ▶ **Decreased PCV level, from 33 to 22 %**

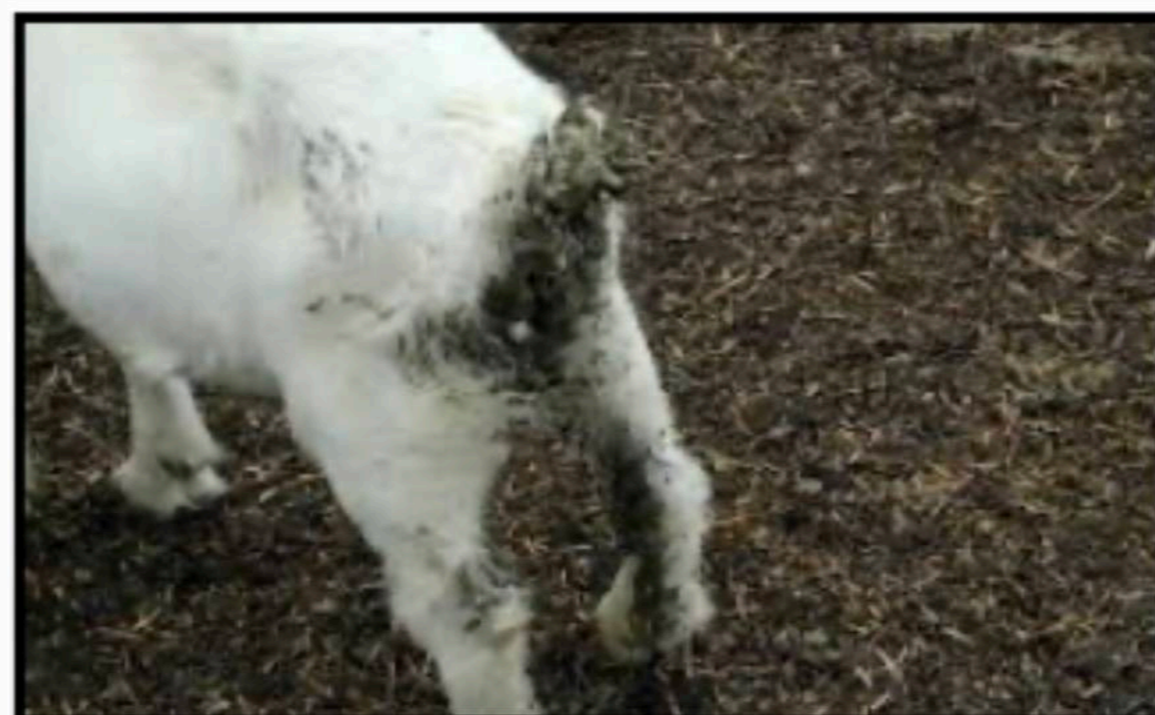
- ▶ **Chronic-  
Anaemia, weight loss & inappetance**





## Intestinal Trichostrongylosis (Black scour/Bankrupt worms)

- ▶ Major effects in lambs and weaners
- ▶ Migration of young adults and immature worms
- ▶ Extensive damage to intestinal villi and duodenal mucosa
- ▶ Generalised enteritis including haemorrhage, oedema
- ▶ Plasma protein loss- hypoproteinaemia, hypoalbuminaemia
- ▶ Heavy infection cause watery diarrhoea, stains the fleece of hind quarters (Black scours)



source : Tafere et al., (2022)



## Ostertagiosis

- ▶ Less fecund worm- 200 eggs/worm/day
- ▶ Main pathogenic effects by its larval stages development
- ▶ Cause **extensive damage to parietal cells and gastric glands of abomaum**
- ▶ Decrease in HCL production - increase in abomasal  $pH$
- ▶ Failure of **pepsinogen to convert pepsin** (active form)
- ▶ Elevated plasma pepsinogen level & reduced protein digestion





- ▶ Moderate infections cause diarrhoea, hypoproteinaemia, poor weight gain and weight loss
- ▶ Type I & Type II type
- ▶ Nodular gastritis covered by mucus
- ▶ **Abomasitis with 'ostrich leather' appearance and ulceration of the mucosa**
- ▶ The brown stomach worms are so small that they are hardly visibly with the naked eye.



Courtesy: Dr. M.Hobson



## Other GIN infections...

### ▶ *Oesophagostomum* sp.

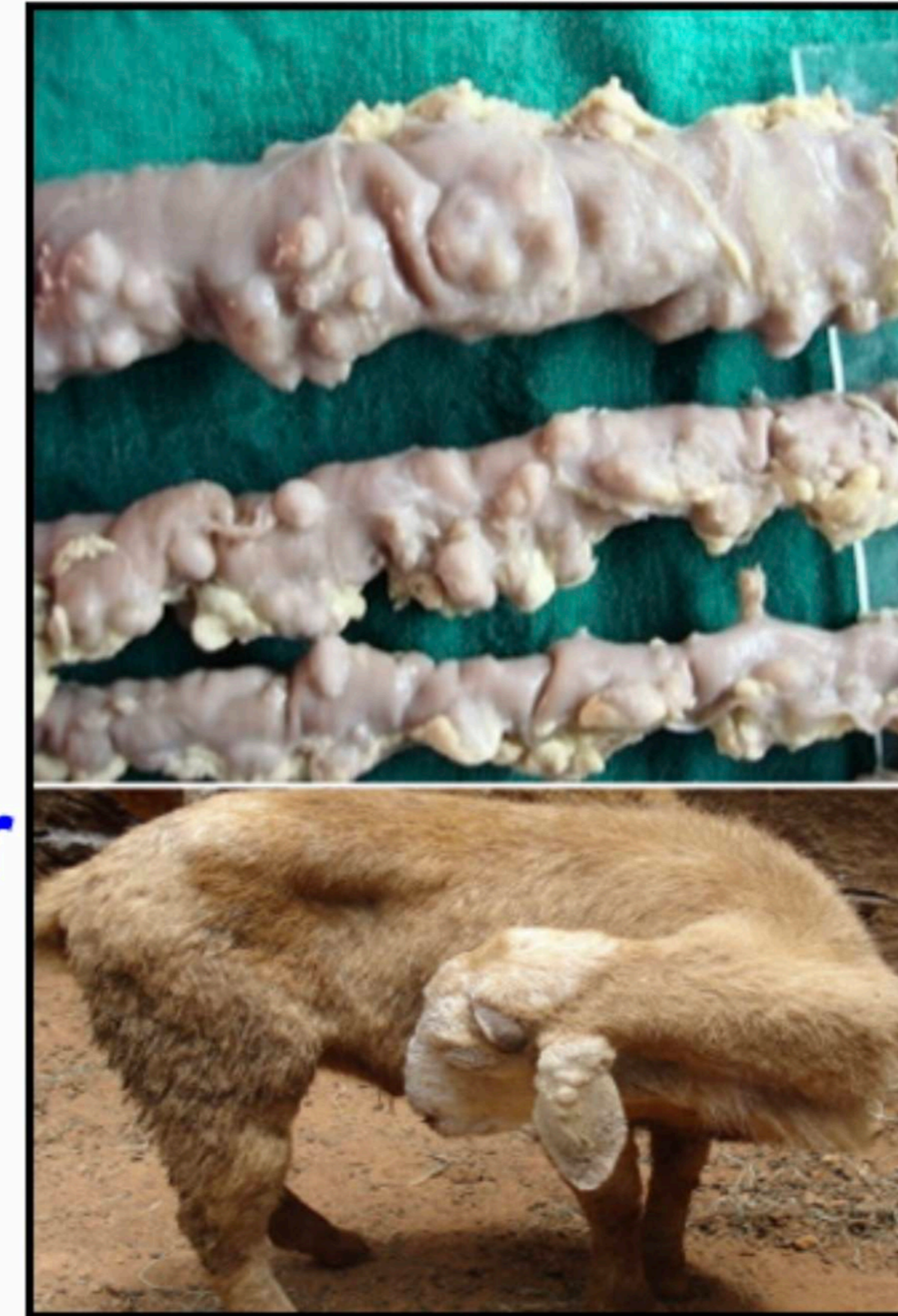
Persistent, foetid diarrhoea, weight loss  
**Nodule formation (pimply gut)**

### ▶ Hook worms (*Bunostomum* & *Gaigeria* sp.)

*Gaigeria pachyscelis* – **voracious blood sucker**  
 Anaemia, diarrhoea and hypoproteinemc edema

### ▶ *Strongyloides* sp

Intermittent diarrhoea with mucus,  
 loss of appetite and weight  
**Foot rot – skin penetration**



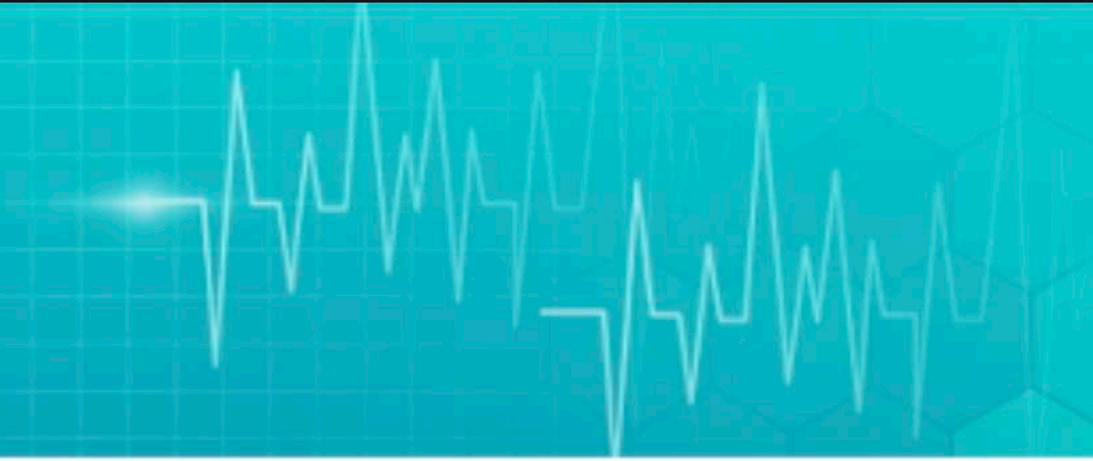


## How can you identify PGE affected animals?

- ▶ Clinical signs, seasonal incidence and history are indicative
- ▶ History –
  - Age, grazing history,  
Anthelmintic usage
- ▶ Clinical PGE – Young animals
- ▶ Mixed GI nematodes are common







## How can you identify PGE affected animals?

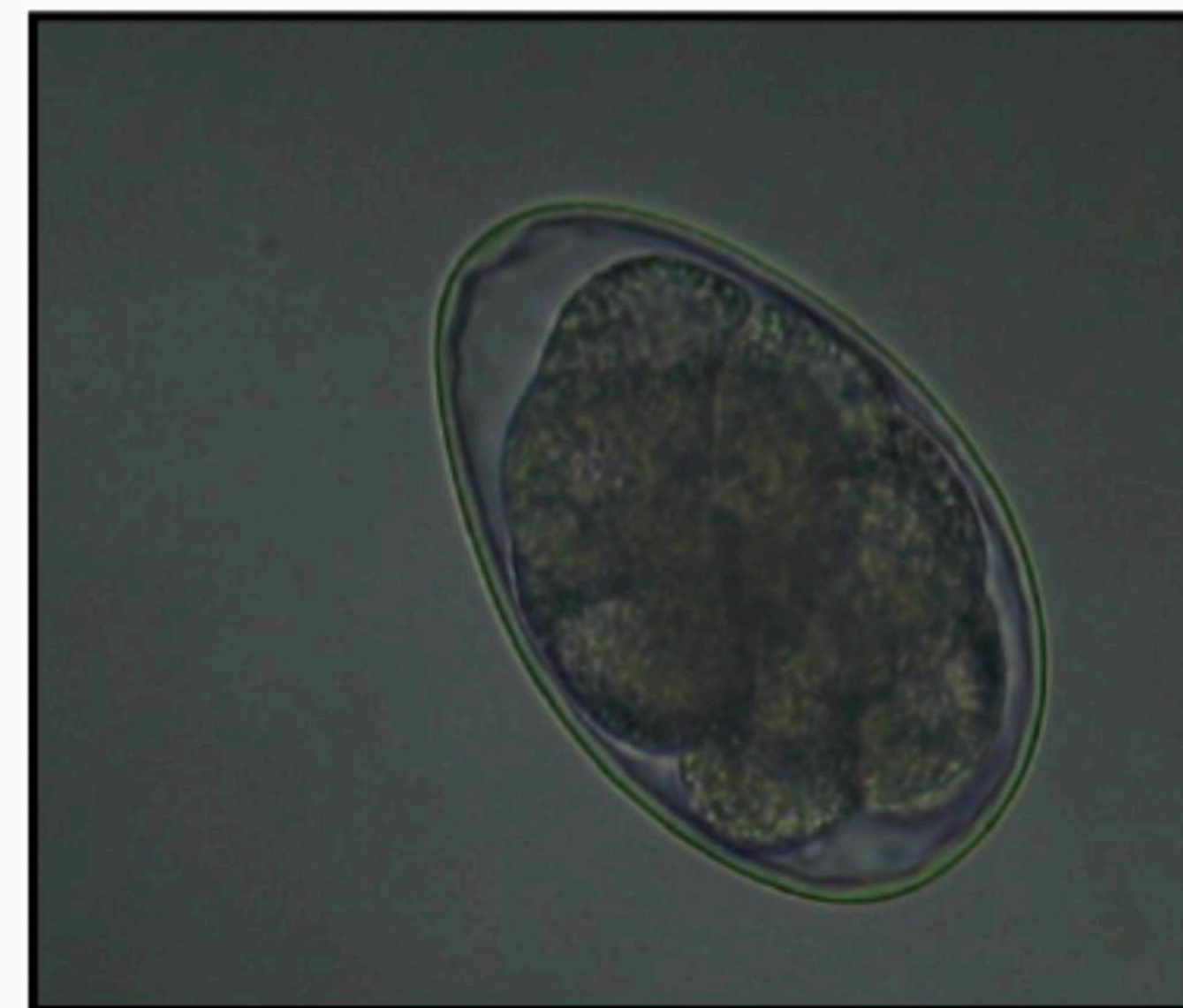
- ▶ **Diarrhoea**  
Profuse and characteristic (Dark / greenish)
- ▶ **Poor body condition/weight loss**
- ▶ **Rough hair coat**
- ▶ **Anorexia/inappetance**





## Signs of GI parasitism

- ▶ **Haemonchosis**  
Anaemia,  
Hypoproteinaemia  
(submandibular oedema- bottle jaw)
- ▶ **Ostertagiosis, Trichostrongylosis & others-**  
Diarrhoea is the common feature
- ▶ **Death in severe cases**
- ▶ **Confirmation by Faecal examination**





## Conclusion

- ▶ **Epidemiology of GIN is mainly governed by host factors, weather conditions and adopted managemental practices.**
- ▶ **Haemonchosis causes more worm related fatalities in small ruminants in India**
- ▶ **Common signs of GI parasitism includes diarrhoea, hypoproteinaemia, decreased weight gain, anaemia and inappetance**



# Epidemiology and pathogenesis of GI parasitism





*Thank you*