VANYA
SILKWORM PATHOLOGY

Dr. Mahesha H. B.
Professor and Head
Department of Sericulture
Yuvaraja's College,
University of Mysore, Mysore, India

Pathology - The scientific study of the nature of disease and its causes, processes, development, and consequences.

Also called *pathobiology*.

OVERVIEW OF THE NON MULBERRY SILKWORMS DISEASES

DISEASE	TASAR	MUGA	ERI
POLYHEDROSIS	**25%**	**OCCATIONAL**	**IMMUNE**
BACTERIOSIS	**15%** **SAL** **CTE** **RP**	**30%**	**14-30%**
MYCOSIS	**NEGLIGEBLE**	**NEGLIGEBLE**	**NEGLIGEBLE**
MICROSPORIDIOSIS	√	**20-30%**	√

Pathology of Tasar Silkworm

35 to 40% of the tasar crop is lost from disease.

Four types of diseases:
Polyhedrosis (virosis),
Bacteriosis,
Mycosis,
Microsporidiosis.

Polyhedrosis (virosis)

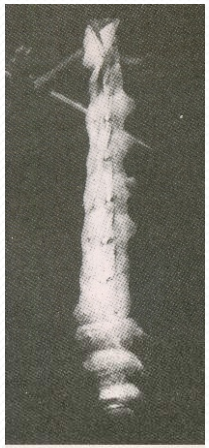
Causative agent: The virus is CPV.

Transmission: Oral.

Incidence: The estimated loss is 25%.

Factors: High humidity and temperature, higher density populations, Feeding on tender, juicy leaves.

Symptoms: The larva > soft and sluggish, body loses its shape and swollen, The integument turns opaque and brownish, internal tissues disintegrate. The larva dies after 24 hours. After death it hangs head down.



A. mylitta larva infected with polyhedrosis

Bacteriosis

Causative agent: Gram-negative micrococci and gram-positive bacilli

Incidence: Causes 15% of larval mortality.

Transmission: Transmitted orally.

Predisposing factors: Over-mature leaves. Infection, is accelerated by lower temperature and R H

Three types

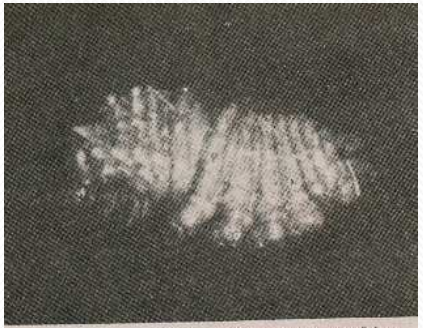
Sealing of the anal lips (SAL)

Chain Type Excreta (CTE)

Rectal Protrusion (RP)

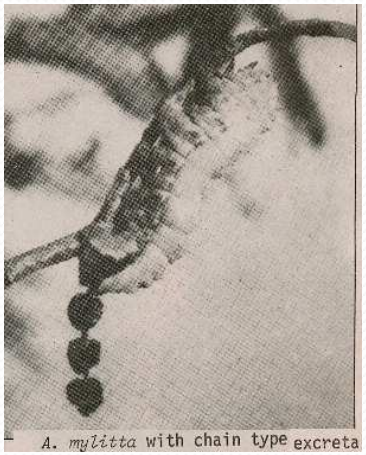
Symptoms: Sealing of the anal lips (SAL) the larva becomes restless, loses appetite and becomes sluggish, it starts defecating a soil-coloured, sticky, semisolid excreta which solidifies on contact with air, sealing the anal lips completely.

At this stage the larva stops feeding, immobile and shrinks, loses its grip on the twig and topples. The larva dies in another 24 hours.



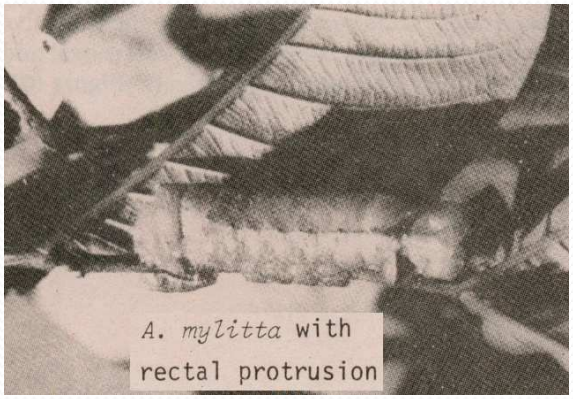
A. mylitta larva with sealing of the anal lips

Chain Type Excreta (CTE): The larva is slightly thin, soft and sluggish, limited feeding. In about 24 hours it starts to excrete a slimy substance along with the faeces, as a result of which chain of faecal beads hangs from the anal opening. The claspers lose hold of the twig, and the posterior half of the larva hangs obliquely. It remains in this position for about 12 hours before toppling and dying.



A. mylitta with chain type excreta

Rectal Protrusion (RP): The infected larva becomes restless, sluggish. In 18 to 20 hours the rectum is protruded in the form of a transparent bag filled with green haemolymph. The anal lips dilate, feeding stops and the body contracts lengthwise. The larva remains attached to the twig for 6 to 8 hours before falling. It dies in another 24 hours.



Mycosis

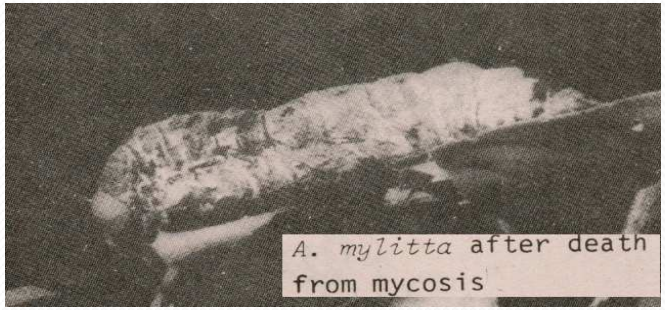
Causative agent: *Penicillium citrinum* & *Paecilomyces varioti*.

Transmission: Integumental injuries and the spiracles.

Incidence: late August to early September. The total loss is negligible.

Predisposing factors: Wounds, high R H, high temperature.

Symptoms: The larva becomes hard, pale and inactive, followed by dorsal bending of the body. In 6 to 8 hours it dies, and within 48 hours the corpse is covered with green, powdery spores. The dead specimens become mummified.

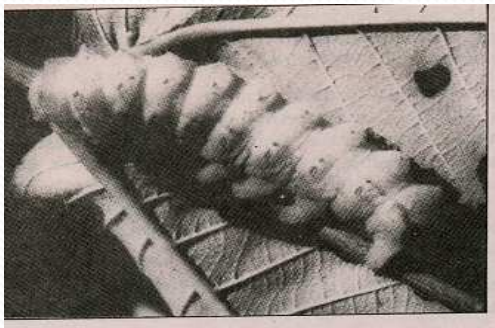


Microsporidiosis

Causative agent: Caused by Microsporidian
Family Nosematidae
Genus Nosema.

Transmission: Oral and Transovarial

Symptoms: Reduced feeding, sluggishness, a thin and darker body, black spots over the integument and stunted growth



Microsporidiosis in an *A. mylitta* larva

Microscopic field of *Nosema* spores causing microsporidiosis in *A. mylitta*

Controls

- **The infection can be reduced by Mother Moth Examination *i.e.*, Examination of Mother moth, disinfection of eggs and maintenance of aseptic conditions during grainage and rearing.**

- **ii. Heat treatment, at the pupal (40°C for 8 hours) and egg (46°C for 5 minutes) stages reduces infection by 80 to 85%.**

Drugs, such as fumagillin and benomyl, are quite effective.

.

Precautions in an epidemic:

i) Seed cocoons should be procured only from uninfected areas (seed zones), and dfls should be selected on the basis of microscopic examination.

ii) The transfer of cocoons from infected to uninfected areas should be prohibited.

iii) Smears of infected moths and larvae of infected lots, along with leaves plucked from that particular bush, should be burned and buried.

iv) Periodic surveys should be conducted at both the larval and the pupal stage to forecast the possibility of epidemic

Pathology of Muga Silkworm

The muga silkworms are vulnerable for: bacteriosis, microsporidiosis and occasionaly Virosis. Negligible amount of Mycosis is reported.

Bacteriosis: CTE & RP have been reported. Symptoms are similar to *A. mylitta*. The disease accounts for a nearly 30% crop loss.

The incidence is highest in the summer crop, and late-instar worms are more susceptible.

Unsuitable leaves and overcrowding induce the disease.

Microsporidiosis:

The disease is caused by a microsporidian- Nosema.

It is transmitted both orally and transovarially.

The incidence of the disease is of sporadic nature being less frequent during spring and autumn but causing a 20-30% crop loss in the rainy season.

Appearance of black spots on the body integument. Spots are of two types: smaller & bigger.

Worms with the latter symptom, if they survive, moult five times instead of the usual four.

Apart from microscopic examination, the pebrine disease can also be checked by exposing the cocoons to 33.8°C and 55-65% R. H. for 15 hours in the pre pupal stage.

The treatment of eggs with hot water (40°C) for 30 minutes for 36-40 hours.

Virosis:

The symptoms are identical to in *A. mylitta*.

A virus characterized by inclusion bodies.

Transmission Oral & the loss is almost negligible.

Control of Diseases: The rearing of disease-free layings, disinfection of equipment and the maintenance of optimum population density.

Pathology of Eri Silkworm

Flacherie: Is the major disease

Flacherie is caused by an ultravirus infection followed by a bacterial attack.

It is transmitted orally through contaminated food.

The disease accounts for the loss of 14 to 30%.

Minimum loss in Jan-Mar & Maximum in April-June.

The first symptom is loss of appetite. The larvae becomes soft and the body colour changes. It vomits and excretes a semisolid excrement. After death the worm putrefies and emits a repellent odor.

***P. ricini* is immune to Grasserie.**

Muscardine:

***Botrytis densa* has been identified for muscardine. The route of infection is the integument.**

The crop loss due to this disease is negligible.

Symptoms The infected worm loses flexibility and becomes brittle and compressed. The corpose of the worm is mummified and covered with a white encrustation, which later turns green.

Controls: As flacherie is due to starvation and malnutrition, the worms should be fed with leaves of suitable quality.

Pebrine:

The symptoms are similar to those in *A. mylitta*.

The *Nosema bombycis* is the causative agent.

Transmission is both transovarial and oral.

The incidence of microsporidiosis is sporadic.

- ***Prevention and control:***
- ***1. Disease Free Layings***
- **2. Strict Disinfection**

Besides,

PREVENTION IS BETTER THAN TREATIMENT/CURE

THANK YOU