GOVERNMENT DEGREE COLLEGE SHADNAGAR

DEPARTMENT OF SERICULTURE

A PROJECT WORK ON

DISEASES OF SILKWORMS

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DECLARATION

We the following students studying B.Sc II year at Govt Degree College ,Shadnagar during the academic year 2021-22 here by declared that is our original project work On DISEASES OF SILKWORMS submitted under the guidance of J.KARTHIK.

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Protozoan disease:

The protozoan disease of silkworm is called Pebrine because of characteristic pepper like spots appearing on the infected silkworm. The disease spreads very quickly and assumes epizootic importance, the entire silk industry of France and Italy would have been wiped out if Pasteur's discover was not come into results for the cause and control for this disease. The name' Pebrine was coined by Dequatrifage In India it was officially recorded in 1895, with introduction of mother moth examination and supply of disease free layings it is under reasonable control.

Causal organism:

The disease is caused by Nosema bombycis Naegeli belongong to family Nosematidae and Microsporidia.

Mode of transmisson:

Oral: Feeding of contamonated mulberry leaf

Contact: The infected larva in the rearing bed liberated spores enter through skin wounds of healthy worms.

Transovarian: By rearing infected silkworm eggs.

Life cycle of Pebrine:

The life cycle of the parasite is completed within a single host, the silkworm. There are two stages in the life cycle.

Spore stage: This is a resistant and infective stage.

Vegetative stage: It is a growing stage.

Spore stage:

The mature spore is oval or ovoaylondical it measures approximately 3.4 × 1.5 × 5.4 microns and refracts light. The spore stage is also called ~~spore~~ sporont. The spore has a protective thick tunic called spore capsule. Two large vacuoles present at the two poles restrict the protoplasm to a girdle like structure in the middle. It is called sporoplasm and binucleate. At one ~~of~~ end of the spore capsule is a bag-like structure called polar capsule. The bag extends to the other end through the interior of the sporoplasm. A long polar filament (4 times as long as the body) is kept coiled inside the polar capsule. The polar filament opens to exterior by means of a small opening called micropyle. The spore can survive in ordinary conditions of aerating house for more than 1 year.

Vegetative stage:

When live spores enter into the silkworm through mulberry leaf, they germinate in the gut due to high and potassium ions. As a result the polar filament is extruded and sporoplasm along with two nuclei creeps through it and injects into the mond gut tissues. Subsequently the polar filament gets digested in the alimentary track. The two nuclei of the sporoplasm unite to form a uninucleate 'planot'. The planot measure 0.5 - 1.5 microns and is formed.

in 1-2 days and multiplies by binary fission and penerates through the gut epithelium, enters the haemocoel and infects various organs like fat body, silk gland and reproductive organs. This is called autoinfection.

Planols are extracellular, motile and uninucleate and divide by binary fission and produce vegetive plemorphs.

Once the planonts the host cell, it transforms into a ~~secondary~~ sedentary from and becomes localized. This stage is known as Meront. meront is an intra cellular stage has a delicate cell wall immobile which absorbs nutrients from host cell and larger than planonts. They grow and under go multiplefission. The products are the spores when cytoplasm of the host cell is exhausted; meronts are arranged in parallel rows. Ultimately, the cells die and liberate the spores.

Symptoms:

The symptoms of this disease can be observed in all the stages of silkworms i,e. egg, larvae, pupa and adult.

Egg stage:

The eggs are over lapped, more of unfertilized and dead eggs indicating pebrone infection. Poor and irregular hatching or immidiately after hatching they may also die. The infected eggs have insufficient or irregularly ~~dispite~~ deposited glue and hence get easily detached from the egg card.

Larval stage:

The larva show poor appetite, retarded growth and development leading to unigvals. Larva moult irregularly and show sluggishness. Trans ovarially infected larva die before third moult but those which are heavily infected die during first instar itself.

The larva body shows wrinkled with skin with rustic brown colour and in moribund stage they do not remain roberry. The affected gut becomes opaque and the ~~skin~~ silkglands shows white pustules in different places along its length. Sometimes black irregular pepper like spots is noticed on larval skin.

Pupal stage:

The infected pupae are flabby and swollen with lusterless and softened abdomen. Sometimes irregular black spots are noticed near the rudiments of the wing and abdominal area. Highly infected pupae fail to metamorphose into adults.

Moth stage:

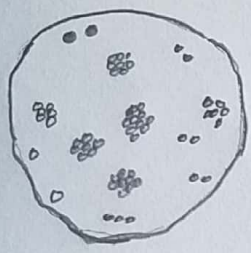
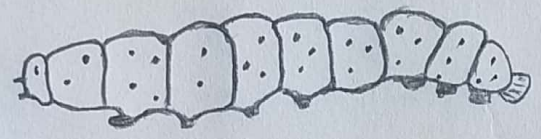
The moth emergence is delayed and improper. They have clubbed wings with ~~distorted~~ distorted antennae and do not mate properly. The scales from wings and abdominal area easily come off. In infected moths if ~~they~~ accessory glands detachment from the egg cords.

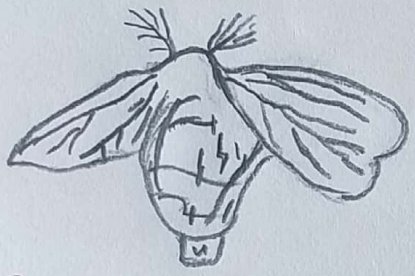
Prevention and control:

* Rearing disease free layings prepared in grainages.
* Effective disinfection and maintenance of hygenic

conditions during rearing helps in controlling the disease.

* Collect and burn the diseased eggs, larvae, pupae and moth, bed refuses faecal pellets, etc.
* Chemical like fumigillan, benomyl, bengard, bavistin, methylthiophanate controls the occurance of disease.



Bacterial Diseases:

Bacterial diseases affecting silkworms are collectively known as flacherie due to the flaccid nature of diseased larva.

(A) Septicaemia:

Causal organism: Bacillus, Streptococci and Staphylococcus

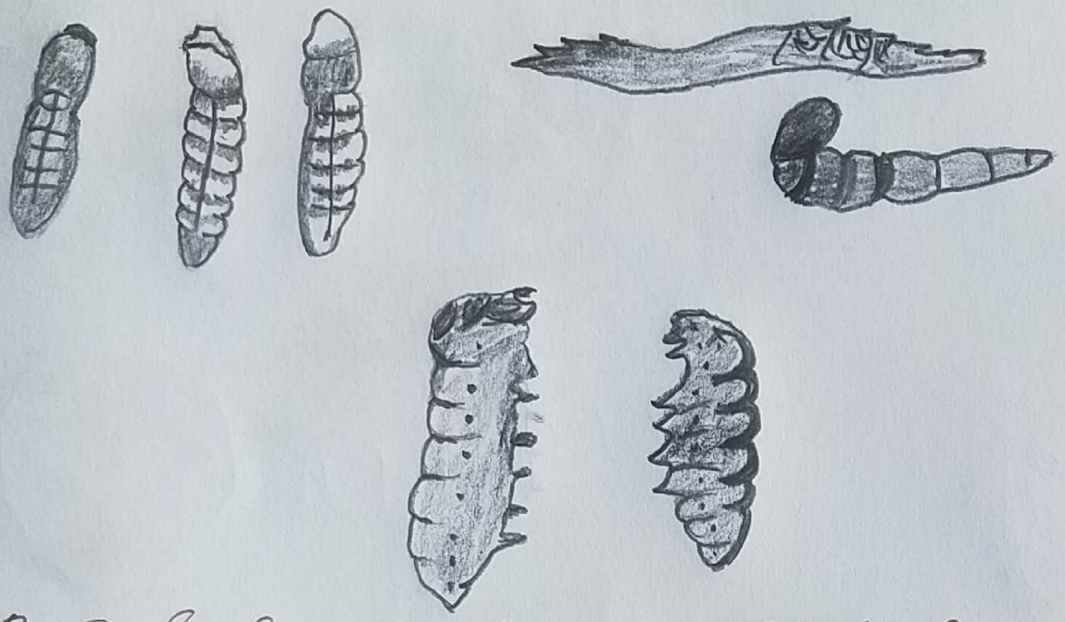
Site of infection: Haemolymph

Source of infection: Injury or rarely orally

Symptoms:

They have common symptoms like sluggish movement, decreased apetite, straightened body, swollen thorax, shrinkage of abdomonal segments. Vomiting and bead like faces and loss of clasping power of legs. Further, the body becomes soft and discoloured

Body wall easily raptures and emits foul smelling fluid.



B. Bacterial disease of the digestive track:

This disease is otherwise known as transparent head diseased due to the bacterial multiplication in the digestive track leading to the swelling and transparency of the head.

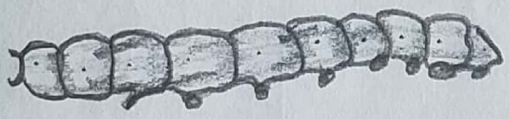
Causal organism: Streptococci; Colé aerogenous Bacillé and Proteus group bacillus.

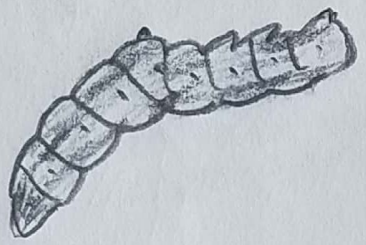
Site of infection: Digestive track

Source of infection: Oral and induction by bad rearing conditions.

Symptoms:

General symptoms are poor appetite, sluggish, transparent head, stunted growth body size and related growth. Sometimes with oral and anal discharges. The diseased worms remain in the spinning tray for a long period without spinning cocoons till they die.





C. Grasserie:

This disease otherwise known as bacterial toxicosis this happens when the silk worms come in contact with the toxin produced by bacilli.

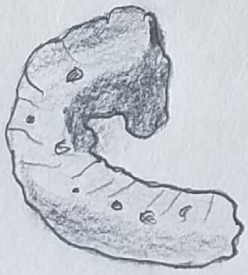
Causal organism: Bacillus thurengiensis.

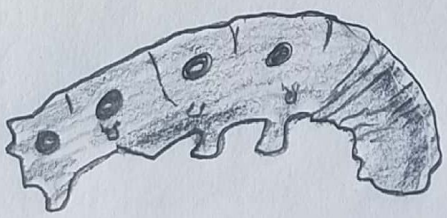
Site of infection: Gut and nervous system.

Source of infection: Usually per oral and wounds / injury.

Symptoms:

Infected larvae lose of appetite. Suddenly show the symptoms of convulsions, lifting of head, spasm, tremors, paralysis, distress, sudden collapse of body and turns to black after death.





Prevention and control measures:

* Disinfection of rearing room and appliances.
* Disinfection of worms, trays and discardong of sick worm.
* Maintanana of hygenic condition
* Feedong the larva with nutrients leaves.
* Avoid injury to the worms, over crowdong of trays and accumulation of ~~faeces~~ faeces in the rearing bed.
* Apply antibiotics like streptomycin / tetracyline / ampicillin on leaves.

Virul disease:

Nuclear polyhydrosis:

It is a major virul disease commonly known as Grasserie, Jundice, Milky disease, Fatty degeneration and having disease.

Causal organism:

Borellona bombycis belongong to the sub group A of the family Baculoviridae.

Site of infection:

Nuclei of tracheae, fatbodies, epidermis and haemolymph of silkworm body, Occasionally seen in middle and posterior portion of silk gland.

Shape of the polyhedra:

The virus is rod shaped and varies in size from 280-300 milli microns. The polyhedra is

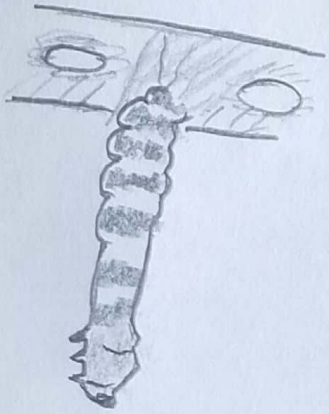
usually hexagonal rarely tetragonal

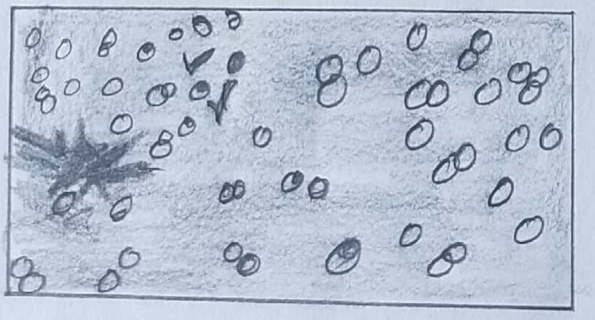
Source of infection:

Contaminated leaves, bad environment and through orally or wound infections.

Symptoms:

Incubation period of this virus varions from 5-7 days. Durong early part of the disease no external symptoms are noticed. But the larva becomes thin and fragile and body becomes milky white with inter segmental swellings. Th fragile skin gets easily ruptured liberating the liquified body contents containing innumerable number of polyhedra. This becomes the source of secondary contamination. The larva become restless and crawl aimlessly along the rims of rearing trays and falls down on the ground and die. Another important symptoms is that the diseased larva looses the holding power of legs except the last pair of legs with which they hang their heads downwards. If the infection is early larva fails to spin cocoon and die, whereas if the infection is late they are able to spin coorns but inside resulting melted cocoons.





Cytoplasmic polyhedrosis:

Causal agent:

Smithia virus belongong to subgroup type 1 of the family Reoviridae.

Site of infection:

Cytoplasam of cylondrical cells of midgut

Shape of the polyhedra:

The virus forms polyhedral in goblet and regenerative cells also showong the characteristic chalky white appearance of the whole midgut. The virus is spherical and varies in size from 60-70 millimicrons. The polyhedra are either hexagonal or tetragonal.

Source of infection:

Contaminated larves and environment. Peros & wound infection. Faeces enters peros is main source.

Symptoms:

Infected larva bag behond in their growth and development with stunted body and dull white in colour. More uneguals are seen resulting irregular Moulting and if infected larva is dissected the midgut of the healthy larva. The whitish and opaque nature of midgut stark from Posterior to anterior with the advana of disease fonally entire gut becomes chalky white.

Prevention and control:

The virus can persist in the form of polyhedra for more than one year inside the rearing house, hence disonfection with 2.5%. Formalon and 0.5%. slacked lime can control the disease.

Ensure proper environmental conditions. The infected larva, faecal matter and bed refuge are destroyed by burning or decomposing in a pit. Feeding quality leaves and mulberry leaf sprayed with 1% calcium hydroxide are fed to larvae to reduce the occurance of c.p.v.

Prevention and control:

* Disinfection of rearing house and equipments with 3% bleaching powder.
* Ensure proper ventilation air circulation.
* Collect and burn infected larvae, faecal matter and refuges.
* Fed the larva with nutricious mulberry leaves and during late age feeding of tender leaves should be avoided
* Proper bed drying is necessary before each feed to avoid accumulation of moisture in the bed.
* Dust bed disinfectant, vijetha or reshamkeetoushad on the larva, 1/2 hours later each moult before resumption of feeding

Infectious flacherie:

Causal agent:

Nonatar virus belonging to the family Picornviridae.

Site of infection:

Infects the goblet cells of the mid gut epithelium with advance of disease the virus is dispersed in the lumen of digestive tract and excreted with faeces.

have been named as.

White muscardine caused by Beauveria bassiana

Green Muscardine caused by Spicaria prasina.

Yellow Muscardine caused by Isaria farinosus.

Black Muscardine by Metarrhizium anisopliae.

White muscardine:

The characteristic feature of Muscardine is the mummification of larva after death by deposition of calcium oxalate salts. Hence this disease is also called Calcino disease.

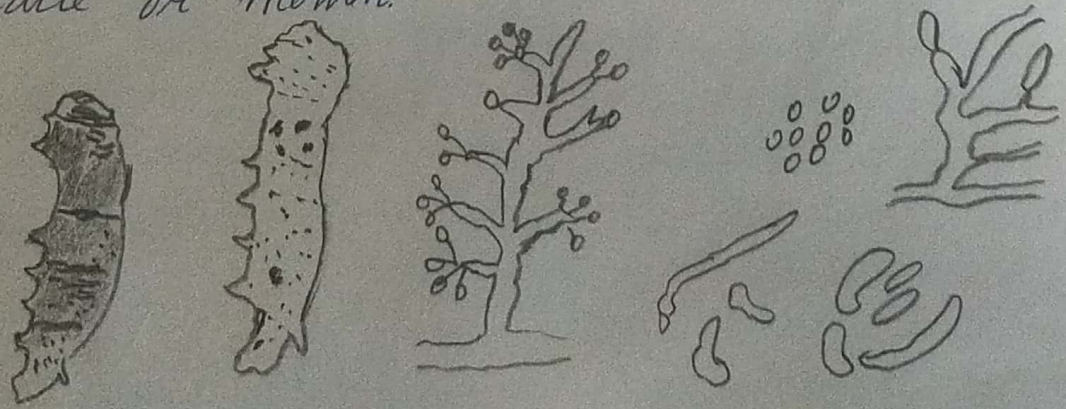
It is the most common fungal disease occurs during rainy and winter seasons under moderate to low temperature and high humidity conditions.

Causal agent:

This disease is caused by Beauveria bassiana belonging to the family Moniliaceae, class: Fungi imperfecti.

Mode of transmission:

Muscardine infection 90% occurs by penetration through the cuticle, 10% through the spiracle or mouth.



Development cycle Beauveria bassiana

a. Affected larva b. Conidia c. Germination of conidia d. Formation of cylindrical spores e. Cylendrical spores f. A Conidiosphore with conidia

Life cycle of muscardine:

The developmental cycle of white muscardine consists of three distinct stage namely conidium, vegetative mycelium and aerial mycelium. The conidium is colourless, globular or rarely oval in shape and porcelain white when gathered in a mass. Under favourable conditions of temperatures and humidity the conidium germinates within 8-10 hrs of contact with the body of silkworms. On germination the conidium not only sends out its germ tube but also secretes chitinase which facilitates the germ tube to penetrate the body wall for further multiplication. The germinating tube of conidium after invading the blood of the larva develops into vegetative hyphae.

Symptoms:

(Loss of appetite), inactiveness and lag in growth results 'unequal' in the rearing bed are the early symptoms. Moist / oily specks appear on the body mostly around the spiracles or the legs. The body of the larvae shrunks and skin becomes inelastic. Larva do not respond to external stimuli and loss spontaneous movement and finally they die. Before death, symptoms of diarhoea and vomiting appear. After death, larva becomes soft, within 6-8 hrs it becomes stiff and hard. Subsequently whole body is covered with conidia except the chitinised parts of the head Due to deposition of calcium oxalate

by the fungus the dead larva becomes mummified into a chalky white structure.

Incase of infected pupa, they do not respond to external stimuli. The thorax shrinks and abdomen is wrinkled. The conidia grow up to one third of its ordinary weight inside the cocoons such cocoons sound like dried coloms when shaken.

During moth stage the body is hardned and wings fall of easily.

Prevention and control:

If muscardine attack is noticed, control measures have to be taken both during rearing and in between rearing. The first and foremost is disinfection of rearing room and appliances and sorroundings thoroughly with 2% formaline or 5% bleaching powder solution. During rearing this disease favours with low temperature and humidity, it has to be regulated. The rearing bed should as much as possible be kept thin and dry in order to avoid the germination of conidia and spread of infection.

Diseased worms should be distroyed by burning with a disinfectant spray. The bed refuse of the infected source should be disposed of properly, if the disease is found during rearing, the trays, seat papers, cleaning nets, foam pads etc must be disinfected and replaced. Preservation of leaf inside rearing house during late age adds to humidity and should be avoided.

Keeping unslyebraked lime in the corners during rainy seasons absorbs excess moisture and reduce the humidity. During

moulting the bed should be kept thin and bed disinfectants should be used.

They are formalinized chalk chlorinated lime, dithane M45, Kesham Kulkarni.

Aspergillus:

Aspergillus attacks on young age larva particularly when high humidity is prevelant.

Causal agent:

This is caused by different species of Aspergillus and Stremomtocystis belonging to the family Moniliaceae of class Fungi imperfecti. Silkworms are infected about dozen species of which Aspergillus flavus Aspergillus oryzae are common.

Life cycle of Aspergillus:

The growth stages of the pathogen consist of the conidium, vegetative and aerial hyphae.

The conidium is spherical, 3-4 microns in size. They are resistant to environmental factors and formaline treatment. The favourable temperature for germination of conidia is 30-50c. Conidia after germination of short hyphae and they grow only at the site of invasion. The conidiosphore is thick and at distal end expands into a globular or oval structure bearing one to two rows of radiating sterigmata on which conidia are formed.

(a) Aspergillus

i. Conidiosphore (ii) Sterigmata (iii) Conidia (iv) Conidia

(b) Aspergillus oryzae:

(I) Conidiospore (II) ~~Phialidae~~ Phialidae (III) Sterigmata

Symptoms:

The disease pathogen attacks only on chawki worms as it is not strong enough to attack on late age larva. Infected larvae stops eating mulberry leaf, become lazy, show body tension, restlessness and then die. Just before death the head and thorax is extended outwards and vomiting occurs. One day after death aerial hyphae and later conidia cover the body. The colour depends on the type of pathogen. The hardening of carpose in dead larvae is limited to the site of fungus penetration and other parts become black and rotten.

Prevention and control:

They are basically similar to white muscardine, but main source is through appliances, special care should be taken to take of sundry. For disinfection purpose 4% Pentachlorophe-nol may be used instead of formalin.