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Non-Spinning Challenges in Silkworm Rearing and Effective Management

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Non-spinning syndrome is a problem affecting silkworm larvae (Bombyx mori), stopping them from forming cocoons even when they develop normally in the early stages. These larvae seem healthy until it's time to spin their cocoons, but then they fail to spin, often making little or no silk, becoming naked pupae, or dying within one to two weeks. The mulberry silkworm, Bombyx mori, is the main species used in commercial sericulture to produce natural silk. The success of sericulture depends largely on the health of larvae, the proper development of their silk glands, and the ability to spin cocoons at the final larval stage. Any disturbance during the last stage of larval development can harm cocoon formation and lower silk production. Among these problems, non-spinning syndrome has become a significant issue related to both biology and farming practices in modern silkworm raising. Non-spinning syndrome is when fully matured larvae fail to start or finish spinning despite appearing physically mature. Normally, mature larvae stop eating, empty their guts, climb onto support structures, and begin excreting continuous silk to make a tight cocoon. In affected cases, the larvae might delay climbing, stay inactive, have soft bodies, or die without spinning. This leads to a drop in the number of good cocoons and increased loss during rearing.The causes of non-spinning syndrome are complex and often come from multiple factors.Poor-quality mulberry leaves, lack of necessary nutrients, and low moisture levels can prevent the production of silk proteins. Unsuitable environmental conditions, such as wrong temperature, humidity, or poor air circulation during the spinning period, can also worsen the problem. Additionally, bacterial infections, pesticide residues on mulberry leaves, the genetic weakness of certain hybrid silkworms, and issues with silk glands can contribute to the disorder.Intensive rearing methods and poor management of mounting also affect spinning behavior. Overcrowding, limited space for mounting, and late transfer of mature larvae to mounting areas can disrupt normal spinning. Since cocoon formation is the most critical and valuable stage in the silkworm's life cycle, any decrease in spinning efficiency greatly impacts cocoon production and farm profitability.

Biology of cocoon spinning:

In the normal life cycle of the silkworm, cocoon spinning is an important process that protects the pupa and ensures successful transformation into a moth.These silkworms are different from their spinning counterparts in terms of genetics, silk gland function, behavior, and how silk proteins are expressed.

1. Genetic and Molecular Basis

The lack of spinning in silkworms is usually due to genetic mutations affecting the expression or function of silk-related genes. Normal silk production involves two main genes:

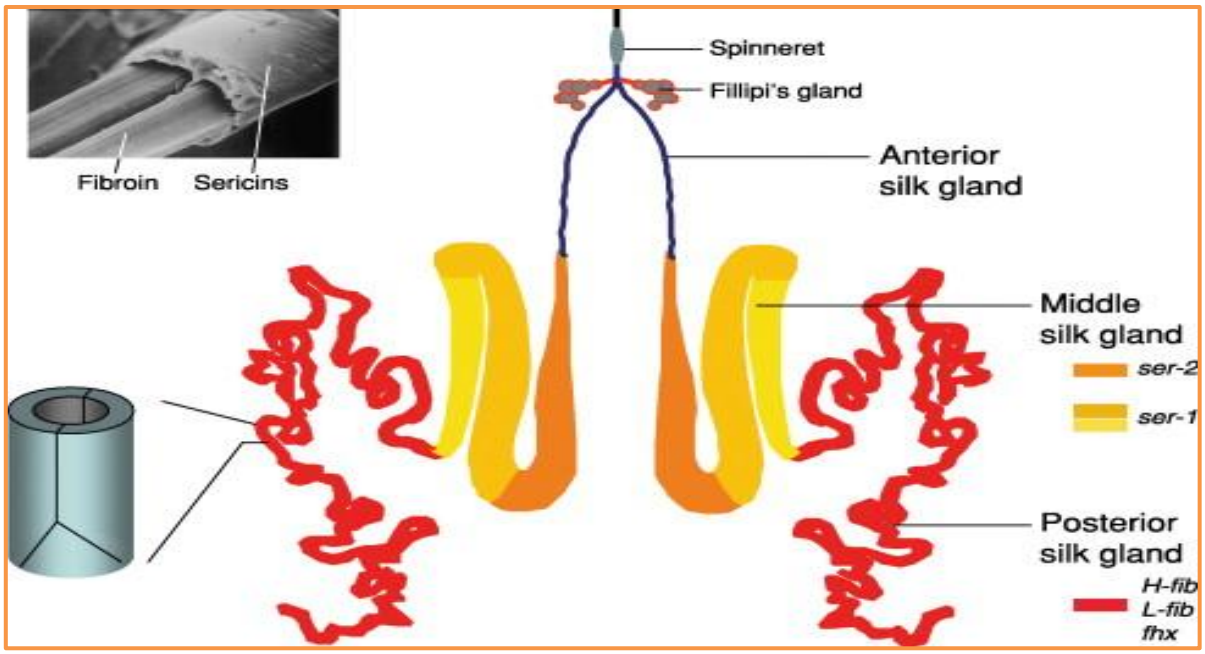
A.FibH and FibL, which code for the heavy and light chains of fibroin, the main structural protein of silk.

B. Ser1, which codes for sericin, a sticky protein that holds fibroin strands together.

In non-spinning silkworms, mutations or deletions in these genes cause the lack or improper function of silk protein synthesis.For example, mutations in the FibH gene prevent the production of enough fibroin, stopping cocoon formation. Also, upstream regulatory genes like SGF1 (Silk Gland Factor 1) and POUM2, which control the transcription of silk genes, may be disrupted. Silkworms with such mutations either don't produce silk proteins or can't organize them correctly in the silk glands.

2. Degeneration of Silk Glands

One of the most noticeable features of non-spinning silkworms is underdeveloped or damaged silk glands. In healthy silkworms, the posterior silk glands (PSGs) become highly enlarged by the end of the fifth instar, storing large amounts of fibroin. Studies have shown reduced expression of key silk genes and early programmed cell death (apoptosis) in silk gland cells.



3. Hormonal and Physiological Differences:

These hormones help move the silkworm from the feeding stage to the spinning stage. In non-spinning silkworms, hormone imbalances, often linked to genetic factors, can disrupt the normal start of spinning behavior. For instance, a drop in JH or a rapid rise in ecdysone can trigger pupation before the silk glands are ready, causing no cocoon to form.

4. Behavioural Abnormalities

Before spinning, normal silkworms show restless behavior, searching for a safe spot and beginning head movements to start making cocoons. Non-spinning silkworms either lack this behavior or do so incomplete. Even in ideal mounting conditions, they might fail to spin or only produce a little silk fluid that doesn't solidify into a cocoon. This indicates a defect in the neurological pattern for spinning, likely related to faulty silk gland output or disruptions in the central nervous system.

5. Evolutionary and Experimental Context

Non-spinning silkworms are not commonly found in nature but are developed and kept in laboratories for research on genetics, physiology, and silk proteins.These strains are important for studying silk gene regulation, transgenic expression, and silk production without sericin. They are also used in biomedical and recombinant protein studies, where cocoons are not needed. In some breeding programs, CRISPR-Cas9 technology has been used to create knockout lines with the FibH gene inactivated, resulting in non-spinning traits.

Impact of pesticide residues on silkworm spinning

Pesticide residues, especially from insect growth regulators (IGRs) like pyriproxyfen, fenoxycarb, and methoprene, severely disrupt silkworm (*Bombyx mori*) spinning by

mimicking juvenile hormones, preventing the shift to pupation. These contaminants reach larvae via mulberry leaves from nearby crop spraying or vector control, even at low doses.

Effects: Residues cause non-spinning syndrome, where healthy-looking larvae fail to form cocoons, produce minimal silk, or die soon after. They impair silk gland development, energy metabolism (e.g., mitochondrial and glycolytic disruption), and detoxification enzymes, leading to developmental delays and reduced cocoon yield.

Economic Losses: Cocooning rates drop linearly with higher doses; for example, methoprene reduces rates from 99% (untreated) to 75.5% at maximum tested levels. Survival at spinning falls, with thin cocoons or naked pupae, causing total loss of a month's rearing effort and threatening silk industries in producing countries.

Prevention methods for non-spinning disease

Prevention methods for non-spinning syndrome in silkworms focus on avoiding pesticide exposure, disease control, and optimal rearing practices. These steps minimize risks from residues, infections, and environmental stress that trigger the condition.

Pesticide Management: Source mulberry leaves from pesticide-free zones or wait 10-15 days post-spraying for residue degradation before feeding. Use botanical pesticides (e.g., neem extracts, essential oils) or green formulations on mulberry fields, as they leave fewer harmful residues and support silk gland function.

Disease Control: Pathogens such as viruses (Bombyx mori nucleopolyhedroviral, BmNPV), bacteria (Serratia marcescens), and fungi (Aspergillus, Streptococcus) directly compromise silk gland integrity, leading to reduced protein secretion and non-spinning behaviour. Their rapid transmission is often favoured by poor hygiene in rearing houses. Maintain strict hygiene in rearing environments. Disinfect rearing rooms, appliances, and mountages regularly using 2–4% formalin, bleaching powder, or slaked lime to eliminate microbial spores. Dispose of diseased larvae properly to prevent secondary infection. Encourage regular monitoring for early detection of subclinical infections.

Rearing Practices: Provide fresh, uncontaminated leaves; ensure proper humidity (70-80%), temperature (25-28°C), and frequent ventilation for clean air. Select disease-resistant hybrid breeds and avoid juvenile hormone misuse during late instars.

Leaf Sourcing: As the sole food source for silkworms, mulberry leaves play a decisive role in cocoon formation. They supply proteins (notably serine and glycine), carbohydrates, and minerals that form the building blocks of fibroin and sericin. Poor-quality, nutrient-deficient, or chemically contaminated leaves impair digestion and silk protein synthesis, resulting in weak or absent cocoon formation. Provide clean, pesticide-free, and disease-free leaves at all times. Apply balanced fertilizers (NPK) along with farmyard manure (FYM) to improve leaf nutrient content, especially nitrogen, which enhances protein accumulation in silk glands. Avoid irrigating mulberry fields with untreated wastewater or industrial effluents, as these may contain heavy metals and harmful microbes that damage silk gland physiology.

Hygiene and Disinfection: Disinfect rearing house, trays, and tools with 2% formalin or 0.2% bleaching powder solution before each crop; maintain personal hygiene and dispose of diseased larvae by burning. Seal cracks, use nylon nets on windows/doors, and avoid overcrowding with proper spacing.

Environmental Control: Silkworm growth and silk gland activity are strongly influenced by temperature and humidity. The optimal ranges are: Temperature: 24–28 °C Relative Humidity: 70–80% Deviation from these conditions can delay larval development, disturb hormonal balance (particularly ecdysone secretion), and induce stress-related degeneration of silk glands. Use fans, heaters, humidifiers, or dehumidifiers to maintain stable microclimatic conditions. Avoid overcrowding, which increases heat stress, reduces oxygen availability, and facilitates pathogen spread.

Breed and Monitoring: Certain mutant strains (e.g., “csr” (cocoonless silkworm races), “fl” (flaccid larvae), and “ns” (non-sericin)) are known to carry heritable defects that impair cocoon formation. If such traits persist within breeding lines, they reduce genetic fitness and lower cocoon productivity over successive generations.

Preventive measures

- Pesticide residue in soil can be degraded by integrated soil remelioration methods like soil solarization/ phytoremediation/ Bioremediation.
- FYM should be applied excessively to facilitate adsorption of pesticides.
- Plants can be irrigated by furrow method/drenching to facilitate leaching of pesticides from the mulberry root zone at the earliest.
- Problematic soil can be replaced from the mulberry root zone with FYM and fresh soil.
- Until soil reclamation, silkworms can be fed with the leaf from pesticide problem mulberry garden till IV moult and leaf from any other normal garden from V instar 1st day upto spinning and thus avoid nonspinning.

Conclusion

The occurrence of non-spinning larvae is not attributed to a single cause but arises from the interaction of multiple factors, including poor nutritional quality of mulberry leaves, environmental stress during the late larval stage, microbial infections, pesticide contamination, genetic susceptibility, and improper mountage practices. Disruption in silk gland development and impaired silk protein synthesis further aggravate the condition, ultimately preventing normal cocoon formation. Effective management of non-spinning syndrome requires an integrated approach that combines scientific rearing practices, strict hygiene, optimal temperature and humidity regulation, timely mounting of ripe worms, and the supply of fresh, nutritious mulberry leaves. Preventive measures are more practical and economical than corrective actions after symptom appearance. Furthermore, capacity building among farmers, timely government interventions and supportive sericultural extension services can enhance early detection and control of non spinning outbreaks. A coordinated, multidisciplinary effort combining traditional knowledge with cutting-edge science is imperative to ensure that the golden thread of silk continues to weave economic security and rural prosperity across generations.

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