Bio. Res. Today 2026, 8(4):62-65

Biotica Research Today

e-ISSN: 2582-6654

April, 2026

Article ID: RT1884 Popular Article

Non-Spinning Behaviour in *Bombyx mori:* Unraveling the Mystery of the Naked Silkworm

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Conflict of interests: The author has declared that no conflict of interest exists.

How to cite this article?

Micheal, A.S, Kumar, A.M.D., 2026. Non-Spinning Behaviour in *Bombyx mori*: Unraveling the Mystery of the Naked Silkworm. *Biotica Research Today* 8(4), 62-65.

Abstract

Non-spinning syndrome (NSS) in the domesticated silkworm, *Bombyx mori* L., is a rare but economically important disorder characterized by failure to initiate or complete cocoon spinning at the end of larval development. Its significance lies in the paradox that affected larvae may develop apparently normally until the critical cocoon-spinning stage and subsequently fail to pupate. Recent research indicates that NSS is multifactorial, with pesticide exposure, insect growth regulators (IGRs), endocrine disturbance, metabolic imbalance, silk-gland dysfunction and viral infection implicated in its occurrence. BmDNV and BmNPV have been detected in non-spinning larvae, together with differential haemolymph protein expression. Pesticide studies further demonstrate that sub-lethal exposure can induce non-spinning before mortality occurs. This article synthesizes the recent evidence to explain NSS as a disturbance of the coordinated developmental, physiological and pathological processes required for successful cocoon formation.

Keywords: *Bombyx mori,* Endocrine disruption, Insect growth regulators, Non-spinning syndrome

Introduction

The domesticated silkworm, *Bombyx mori* L., is an economically important insect whose life cycle is closely linked to silk production. At the end of larval development, a healthy silkworm undergoes a precisely timed transition from feeding and growth to wandering, silk secretion and cocoon construction. The cocoon provides protection during the subsequent pupal stage and is the principal source of raw silk for sericulture. Non-spinning syndrome (NSS) represents a striking failure of this terminal developmental process. Affected larvae may appear healthy and develop normally during earlier instars but become unable to construct a cocoon when they reach the spinning stage. Gürel (2025) described this characteristic pattern as larvae that develop apparently normally until cocoon formation and then fail to spin, and may survive for another one or two weeks before eventually dying.

The biological mystery of NSS is therefore not simply why a silkworm fails to produce silk. Rather, the important question is why a larva that has successfully completed most of its development suddenly fails at the precise stage when endocrine signalling, metabolism, silk-gland activity and behaviour must operate together. The available literature suggests that no single explanation adequately accounts for all cases. Pesticide residues, particularly juvenile-hormone-like insect growth regulators, have received considerable attention, while recent molecular studies have associated BmDNV and BmNPV infections with non-spinning phenotypes (Sindhu *et al.*, 2024; Gürel, 2025). This article therefore examines NSS as a multifactorial disturbance of the larva-to-pupa transition.

Cocoon Spinning: The Critical Endpoint of Larval Development

Silkworm development proceeds through the characteristic stages of egg, larva, pupa and adult. During the final larval stage, the insect prepares for metamorphosis by shifting from intensive feeding and growth toward cocoon construction. The cocoon is not an incidental product; it is

Article History

RECEIVED on 21st March 2026 RECEIVED in revised form 15th April 2026 ACCEPTED in final form 17th April 2026

the protective structure within which the insect completes pupal development (Gürel, 2025).

The unusual feature of NSS is its timing. The disorder generally becomes apparent at the end of larval development rather than immediately after exposure to a damaging factor. Gowda *et al.* (2026) emphasize that affected larvae may appear healthy during the first four instars and that the crisis emerges during the fifth instar, when successful cocoon formation depends upon coordinated endocrine, metabolic and silk-gland activity.

Consequently, failure to spin can be regarded as an endpoint manifestation of the disorder. It may represent the final manifestation of a disturbance that has already affected internal physiology. The larva may continue to live, but it cannot complete the developmental programme that normally culminates in cocoon formation and pupation.

The Silk Gland and the Machinery of Spinning

A proper understanding of NSS requires consideration of the silk gland. In *B. mori*, the paired silk glands originate from salivary glands and discharge through a pore near the lower lip. They consist of three principal regions: the posterior silk gland (PSG), middle silk gland (MSG) and anterior silk gland (ASG).

The PSG is principally associated with fibroin production, whereas the MSG contributes sericin, which forms the coating around the fibroin filament. Silk proteins are stored in soluble form at high concentration before being transported through the anterior gland. During spinning, the silk filament is drawn out through the spinning apparatus by the movement of the larva's head (Andersson *et al.*, 2016). The organization and functional sequence of these glandular regions are summarized in figure 1.

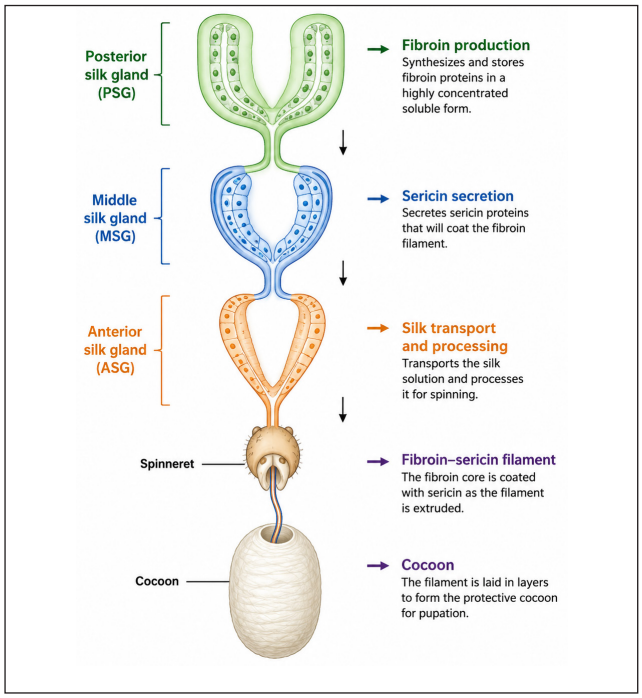

Figure 1: Simplified organization of the B. *mori* silk-spinning system

The process is consequently more complicated than the simple production of a fibre. Silk proteins must be synthesized, stored, transported and converted into a suitable fibre, while the larva must simultaneously perform the coordinated movements required for spinning. Andersson *et al.* (2016) also describe the importance of the glandular chemical environment and pH-related processes in silk processing.

A disturbance of any of these components could therefore contribute to spinning failure. However, the available evidence does not support the conclusion that every non-spinning larva has a primary structural defect of the silk gland. Rather, gland dysfunction may occur as one component of a broader physiological disturbance.

The Endocrine Clock behind the Spinning Behaviour

The final larval transition is under strong endocrine control. Amarnatha *et al.* (2024) describe the endocrine system of *B. mori* as involving neurosecretory cells in the brain, corpora cardiaca, corpora allata, prothoracic gland and sub-oesophageal gland. These systems participate in the regulation of activation, juvenile hormone (JH), moulting hormone/ecdysone and diapause-related processes.

PTTH is particularly important in regulating ecdysone production. When the larva reaches the appropriate developmental condition, PTTH released from the brain stimulates the prothoracic gland to produce ecdysone, thereby initiating developmental changes associated with moulting and metamorphosis. JH, produced by the corpora allata, is a major regulator of insect metamorphosis.

This hormonal relationship provides a useful framework for understanding the final developmental transition and explains why chemicals with juvenile-hormone-like activity are particularly important in NSS research, as summarized in figure 2. If an insect growth regulator interferes with the normal endocrine transition, the larva may fail to move appropriately from a larval physiological state toward metamorphosis and cocoon formation.

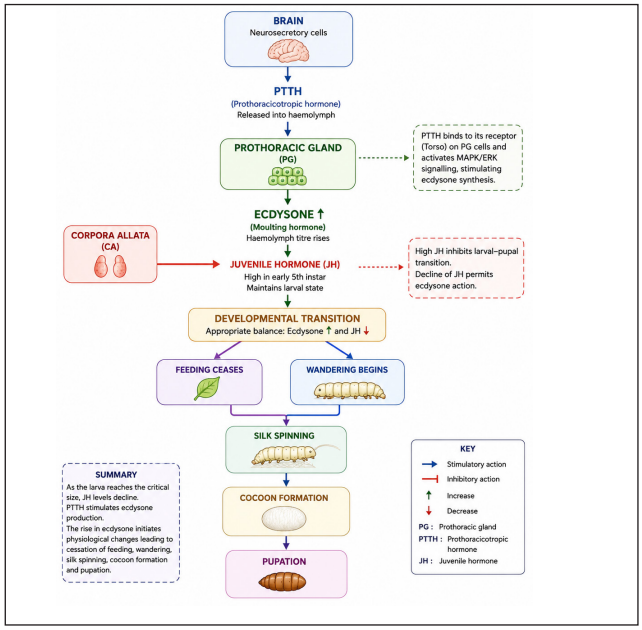

Figure 2: Endocrine framework of the final developmental transition

The endocrine model should, however, be interpreted cautiously. The available literature establishes the importance of JH, PTTH and ecdysone in developmental regulation, but does not demonstrate that endocrine disruption alone explains every instance of NSS. It is therefore more appropriate to consider endocrine disturbance as one important mechanistic route within a multifactorial syndrome.

Pesticides and Insect Growth Regulators: A Major Suspect

Among the possible causes of NSS, pesticide exposure has received particular attention. Gürel (2025) reported associations between low-dose exposure to certain newer broad-spectrum pesticides with insect-growth-regulating properties, particularly juvenile-hormone analogues such as pyriproxyfen, fenoxycarb and methoprene, and non-spinning behaviour in silkworms. Exposure occurs primarily through contaminated mulberry leaves, including contamination resulting from pesticide applications to neighbouring non-mulberry crops or vector-control activities.

This pathway is particularly important because mulberry is the direct food source of the silkworm. A chemical applied elsewhere can therefore become biologically relevant to sericulture if residues reach mulberry foliage.

The mechanism is especially intriguing in the case of IGRs because their biological purpose is to alter insect development. Previous research summarized by Sindhu *et al.* (2024) shows that pyriproxyfen can injure the silk gland, reduce silk output and cocooning speed, and decrease expression of genes involved in silk-protein production. The same source identifies the JH-Met2-Kr-h1 pathway as being involved in pyriproxyfen-induced defects in metamorphosis and silk-protein synthesis.

Thus, pesticide-associated NSS can potentially involve more than direct toxicity. A chemical may interfere with the developmental programme itself, impair silk-gland function, or disturb the physiological preparation necessary for cocoon formation.

Sub-Lethal Toxicity and the Failure to Spin

An important finding of Lokanath *et al.* (2025) is that pesticide toxicity does not necessarily result immediately in death. Their experiments documented vomiting, flaccidity, rectal protrusion, chain faeces, body shrinkage, partial spinning, non-spinning and mortality. Their probit analysis indicated distinct concentration ranges associated with spinning, non-spinning and mortality.

The study also examined acetylcholinesterase (AChE), an important target in insecticide toxicity. Imidacloprid produced the greatest inhibition of AChE extracted from the silkworm head among the compounds examined, while anthranilic diamides, pyrethroids and fungicides also inhibited AChE in time-dependent assays. A rapid paper-strip method was subsequently used to screen pesticide residues on mulberry leaves and soil; 35 of 62 field samples examined were positive (Lokanath *et al.*, 2025).

These findings demonstrate why non-spinning should be considered a serious toxicological endpoint in sericulture. A larva does not have to die immediately for an exposure to cause substantial economic loss. If its development, nervous function or spinning behaviour is sufficiently disturbed, the intended agricultural product (the cocoon) is lost.

Viral Infection: Another Route to the Same Phenotype

Pesticides, however, do not explain all NSS observations. Sindhu *et al.* (2024) investigated BmDNV and BmNPV infections in non-spinning silkworms using morphological, molecular and proteomic approaches. Their results associated both viral infections with abnormal silk-gland growth and the non-spinning phenotype. PCR analysis demonstrated amplification of both viral infections in the investigated samples.

The study also identified substantial differences in haemolymph protein expression between healthy and non-spinning larvae. SDS-PAGE revealed differential regulation of proteins involved in physiological and immunological processes, while MALDI-TOF/MS identified an up-regulated lepidopteran low-molecular-weight lipoprotein.

The importance of this finding is that NSS may involve direct disturbance of the tissues and physiological systems associated with silk production. Sindhu *et al.* (2024) further noted that feeding pesticide-contaminated mulberry leaves has been associated with non-spinning and the interactions among disease, pesticide exposure, hormonal regulation and biochemical pathways require further investigation.

The viral evidence should nevertheless be interpreted according to the scope of the study. Detection of BmDNV or BmNPV in affected larvae provides evidence of association and supports their importance as disease-related factors, but it does not establish that all cases of NSS have a viral origin.

NSS as a Multifactorial Syndrome

The combined evidence supports a model in which different initiating factors can converge upon the same developmental endpoint, as summarized in table 1.

Table 1: Major factors associated with non-spinning syndrome

Factor	Principal level affected	Potential consequence
Juvenile-hormone-like IGRs	Endocrine/ developmental	Disturbed metamorphic timing
Pesticide toxicity	Neural/physiological	Abnormal behaviour and impaired spinning
BmDNV	Disease/silk-gland system	Abnormal gland development and non-spinning
BmNPV	Disease/physiological system	Altered protein expression and abnormal development
Metabolic dysregulation	Whole-animal physiology	Potential disruption of developmental transition
Environmental exposure	Multiple systems	Increased risk of developmental failure

Gowda *et al.* (2026) similarly summarize NSS as being associated with pesticide residues, hormonal mimics, biochemical imbalance, viral protein alterations, metabolic dysregulation and oxidative-toxicity pathways.

This multifactorial interpretation is important because the external phenotype alone is insufficient to identify the cause. A silkworm that fails to spin may have experienced chemical exposure, viral infection or another physiological disturbance. Diagnosis therefore requires examination of the rearing environment as well as the insect.

Diagnosis and Prevention

Early diagnosis is essential because NSS becomes evident near the end of a larval cycle in which considerable time and resources have already been invested. Field observations should include larval behaviour, feeding pattern, body condition and the timing of spinning failure. Where pesticide exposure is suspected, both soil and mulberry leaves should be considered as potential sources.

Recent literature emphasizes environmental assessment in the management of NSS. Gowda *et al.* (2026) recommend reviewing the previous land-use history before establishing mulberry gardens, including previous crops and IGR use, and suggest pilot rearing as a practical preliminary assessment. Their proposed management measures for pesticide-suspected gardens include shifting to pesticide-free foliage during the fifth instar and approaches such as phytoremediation, soil solarization, organic amendment and controlled leaching.

Research Priorities

The literature reveals several areas requiring further investigation. First, the relationship between endocrine disruption and NSS deserves direct experimental study. Sindhu *et al.* (2024) explicitly identify the need to investigate hormonal regulation under disease and pesticide exposure and to determine biochemical changes associated with larval-pupal metamorphosis.

Second, molecular biomarkers could improve early diagnosis. The differential haemolymph protein expression reported in non-spinning larvae suggests that proteomic approaches may identify physiological signatures associated with the syndrome.

Third, the interaction between pesticide exposure and infectious agents requires greater attention. The available studies indicate that both chemical and viral factors can influence silkworm health, but their combined effects are not yet sufficiently resolved. Consequently, future NSS research should move beyond single-factor experiments toward integrated studies involving endocrine, metabolic, immunological and silk-gland parameters.

Conclusion

Non-spinning syndrome in *Bombyx mori* represents a complex failure at the most economically consequential point of silkworm development. The characteristic "naked silkworm" phenotype is the visible consequence of an inability to initiate or complete cocoon spinning, but the underlying disturbance may originate at several biological levels. Recent literature identifies pesticide exposure, particularly juvenile-hormone-like insect growth regulators, as an important concern because such compounds can interfere with development, silk production and physiological function. Experimental evidence further demonstrates that pesticide exposure can produce non-spinning at concentrations below those causing mortality. At the same time, BmDNV and BmNPV have been associated with abnormal silk-gland development and differential protein expression in non-spinning larvae. Endocrine regulation provides an additional framework for understanding why the disorder emerges specifically at the larval-to-pupal transition. Therefore, NSS should be approached as a multifactorial syndrome rather than a single disease with one universal cause. Effective prevention requires attention to pesticide history, mulberry-leaf quality, environmental conditions, disease surveillance and timely diagnosis. A clearer understanding of the interaction among endocrine, metabolic, infectious and silk-gland processes will ultimately be essential for protecting cocoon production and ultimately reducing the occurrence of this economically important phenotype.

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