

Deleterious Effects of the Agriculturally Important White Muscardine Fungus, Beauveria Bassiana on Sericulture: A Short Review

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ABSTRACT

Beauveria bassiana (Bals. -Criv.) Vuill. is widely valued as an entomopathogenic biocontrol agent in agriculture and forestry, yet it is also the causal agent of white muscardine, one of the most contagious and destructive diseases of the domesticated silkworm, *Bombyx mori* L. This short review integrates classical experimental evidence with recent molecular, microbiome and epidemiological studies to summarise how the fungus harms sericulture. Infection proceeds through cuticular penetration and gut invasion, and kills larvae within days; all developmental stages are susceptible, but late-instar larvae are the most vulnerable, with experimental larval mortality approaching 100% and median lethal concentrations falling from about 1,600 spores mL⁻¹ in first-instar larvae to about 435 spores mL⁻¹ in fifth-instar larvae. Cool (15–18 °C), humid (90–100% relative humidity) and poorly ventilated rearing conditions favour outbreaks, and infected moths produce far fewer eggs. Silkworm strains differ markedly in susceptibility, and host defences (cuticular proteins, serine protease inhibitors, antimicrobial peptides and mulberry-derived cuticular bacteria)

offer routes to resistance. Population-genetic tracing in China found no link between silkworm outbreaks and released biopesticide strains, so the risk from agricultural use of *B. bassiana* near sericultural areas remains plausible but poorly quantified. We outline diagnostic and management options and identify priorities for research and policy.

INTRODUCTION:

Sericulture, the rearing of the domesticated silkworm *Bombyx mori* L. for silk, supports rural livelihoods across Asia and parts of Europe, but it is vulnerable to infectious disease at every stage of the life cycle. Fungal, bacterial, viral and microsporidian diseases together are estimated to cost Indian sericulture 30–40% of potential silk yield, with white muscardine alone accounting for 10–40% (Parasuramudu *et al.*, 2025). White muscardine is caused by *Beauveria bassiana*, and it was among the first diseases of any animal to be traced to a microbial agent (Bassi, 1835, as cited in Venkataramana Reddy, 1978). The disease takes its name from the white, chalky mycelium that covers the cadavers; other muscardines are named for the colour of the fungus involved (green, yellow and black or brown), but the white form is the most widespread (Savu & Şapcaliu, 2024).

The same fungus has a second identity. *B. bassiana* has a very broad host range and is a mainstay of biological pest control, applied as a commercial mycoinsecticide against agricultural and forest pests (Cappa *et al.*, 2024; Chen *et al.*, 2015). Because the silkworm is highly susceptible, the expansion of fungal biopesticides into landscapes that also contain mulberry gardens and rearing houses raises an obvious concern. This short review therefore asks three questions. How does *B. bassiana* harm silkworms, and how large are the effects? What is known about the link between agricultural use of the fungus and silkworm outbreaks? And which detection and management tools are available? We draw on a classical experimental thesis on white

muscardine (Venkataramana Reddy, 1978), a recent review of mycotic diseases of *B. mori* (Savu & Şapcaliu, 2024), and molecular and applied studies published up to 2025.

The pathogen and the infection process

B. bassiana is a facultative parasite that passes through three phases: conidia, vegetative mycelium and aerial mycelium (Savu & Şapcaliu, 2024). Its conidia are globose and small, measuring about 1.5–2.9 µm in diameter, and conidia deposited in the rearing environment can remain infective for months to a year (Savu & Şapcaliu, 2024; Venkataramana Reddy, 1978). In the natural environment, infection can be divided into surface adhesion, cuticle penetration, and replication with host killing (Xing *et al.*, 2017). On the silkworm integument, conidia germinate about 8 h after contact, form an appressorium within 24 h, and penetrate the cuticle; the fungus then multiplies in the haemocoel, kills the larva in roughly five to six days, and completes its life cycle in seven to eight days (Kumar *et al.*, 1999). Penetration depends on cuticle-degrading enzymes, including chitinases that act synergistically with proteases (Liu *et al.*, 2025; Ortiz-Urquiza & Keyhani, 2016). Experimental work also shows that the digestive tract is an entry route in addition to the cuticle (Venkataramana Reddy, 1978), and the respiratory route and skin pores have been proposed as further portals (Savu & Şapcaliu, 2024). Cyclic peptide toxins secreted by the fungus are thought to contribute to host death (Xing *et al.*, 2017). The main steps of the cycle, from

contact to spread from the cadaver, are summarised in Fig. 1.

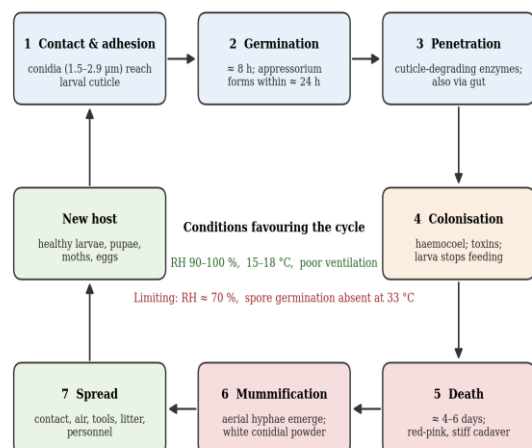


Fig. 1 Infection and transmission cycle of *Beauveria bassiana* on the silkworm *Bombyx mori*.

Timings and environmental thresholds are from Kumar *et al.* (1999), Savu and Şapcaliu (2024) and Venkataramana Reddy (1978). RH, relative humidity.

The symptoms are stage-specific. Infected larvae become sluggish, lose appetite, elasticity and irritability, and develop black or brown spots that may ooze an oily fluid; after death the cadaver turns pink-red and then becomes covered with white efflorescence, drying into a mummy that resembles a lime-covered stick (Venkataramana Reddy, 1978). Infected eggs are pale or pinkish and partially collapsed. Infected pupae soften and die and become covered with the same efflorescence, while infected moths are sluggish, less inclined to copulate and eventually die (Venkataramana Reddy, 1978). Reported times from infection to death range from two to six days depending on instar (Savu & Şapcaliu, 2024) to four to eleven days in experimental inoculations (Venkataramana Reddy, 1978), which reflects differences in dose, temperature and silkworm strain.

Epidemiology and environmental drivers

The main source of infection is the diseased cadaver, which produces enormous numbers of conidia; the fifth-instar larval cadaver alone

can carry several million (Savu & Şapcaliu, 2024). Spores also spread on contaminated surfaces, equipment and clothing, and in rearing rooms that have not been disinfected (Savu & Şapcaliu, 2024). Outbreaks are most common in the rainy and winter seasons, when humid, cool conditions inside rearing trays favour transmission (Parasuramudu *et al.*, 2025). Relative humidity of 90–100% combined with 15–18 °C is highly favourable to the fungus, whereas about 70% relative humidity prevents disease development, and spores do not germinate at 33 °C (Savu & Şapcaliu, 2024); temperatures of 24–28 °C are still reported as reasonably permissive, so temperature alone is not a reliable safeguard. Consistent with this, the fungal life cycle in fifth-instar larvae varied from 3.9 to 11.6 days across rearing seasons in one year of experiments, being longest in November–December and shortest in May–June (Venkataramana Reddy, 1978). Effects of temperature and humidity on rearing performance and disease incidence have been documented for silkworm hybrids (Kumari *et al.*, 2001), and poor ventilation and proximity to standing water further raise the risk (Savu & Şapcaliu, 2024).

Deleterious effects on silkworm survival, silk production and seed

Stage and instar susceptibility. In inoculation experiments on eleven silkworm races, all developmental stages were susceptible: mortality was about 5.7% for eggs, 99.9% for larvae, 40.9% for pupae and roughly three-quarters for moths (Venkataramana Reddy, 1978). Freshly laid eggs were more susceptible than older eggs, and younger pupae more than older ones. Among larvae, susceptibility rose with age (Table 1). Mortality after inoculation was 99.55% in fifth instar compared with 36–46% in the first two instars, and the concentration needed to kill half of the larvae fell from about 1,600 spores mL⁻¹ in first instar to 435 spores mL⁻¹ in fifth instar

(Venkataramana Reddy, 1978). Larvae were most susceptible during the first two days of the fifth instar. The practical consequence is that the period of greatest economic exposure, the last instar during which the crop is made, coincides with the greatest physiological vulnerability. Savu and Şapcaliu (2024) additionally note that larvae are particularly sensitive around the exit from moulting sleep.

Table 1 Susceptibility of *Bombyx mori* larvae to *Beauveria bassiana* by instar

Larval instar at inoculation	Larval mortality (%)	LC ₅₀ (spores mL ⁻¹)	Healthy cocoons (% of inoculated larvae)
I	45.92	1598	12.88
II	36.25	1410	8.63
III	60.20	1171	2.25
IV	88.67	602	0.75
V	99.55	435	0.00

Data from Venkataramana Reddy (1978). Mortality is from a fixed-dose experiment (four replicates of 300 larvae per instar); LC₅₀ is for newly moulted larvae; healthy cocoons are from a separate experiment on 800 larvae per instar in which larvae were inoculated in the instar indicated.

Loss of cocoons. Even larvae that survive inoculation rarely yield a sound cocoon. In the same experiments the proportion of healthy cocoons from inoculated larvae fell from 12.9% when inoculation occurred in the first instar to 0.75% in the fourth and none in the fifth (Table 1), so disease during the last instars translates almost directly into crop failure (Venkataramana Reddy, 1978). Field and laboratory studies in India continue to report reduced growth and yield of bivoltine breeds after white muscardine infection (Manjunatha et al., 2023), and the disease also affects non-mulberry silkworms such as the

muga silkworm *Antheraea assamensis* (Saharia et al., 2024).

Effects on seed production. White muscardine also threatens the seed supply. Females inoculated after mating laid on average about 256 eggs, and females mated after inoculation only about 101, compared with about 514 for healthy moths (Venkataramana Reddy, 1978). Because adult and egg stages are also susceptible, contamination of grainages can carry the fungus into the next rearing cycle.

Breed and strain variation. No race screened in the classical study was immune, but the Pure Mysore race showed lower larval mortality and gave more healthy cocoons, and isolates of the fungus differed in virulence; one isolate caused 100% larval mortality (Venkataramana Reddy, 1978). Modern work confirms wide variation on both sides of the interaction. In a comparison of two silkworm strains, almost all larvae of the sensitive strain Haoyue were dead 168 h after immersion in 10⁵ conidia mL⁻¹, whereas nearly 70% of the resistant strain Kang 8 remained alive and could spin cocoons (Xing et al., 2017). The resistant strain up-regulated cuticle-protein, serine-protease-inhibitor and antimicrobial-peptide genes and detoxification genes, while several of these were down-regulated in the sensitive strain (Xing et al., 2017). Isolate-level characterisation of *B. bassiana* from diseased silkworms is now reported from several regions (Park et al., 2024; Sahana et al., 2023), which will help to relate virulence to strain identity.

Economic scale. Historical estimates cited in the classical thesis put white muscardine losses at 5–35% in northern Italy and as high as 100% in individual outbreaks in Karnataka, India (Venkataramana Reddy, 1978). These figures are old and derive from unpublished or regional reports, and contemporary loss estimates are patchy (Parasuramudu et al., 2025), which is itself a gap for policy.

The agricultural interface: biopesticide use near sericultural areas

That *B. bassiana* is agriculturally important is precisely why it is a concern for sericulture. Formulations of the fungus are promoted as eco-friendly and generally regarded as safe for non-target organisms, but a growing literature documents adverse effects on some non-target insects. For example, exposure of the paper wasp *Polistes dominula* to a field-realistic concentration of a commercial strain (ATCC 74040) reduced individual survival, altered behaviour and impaired colony reproduction (Cappa *et al.*, 2024). A domesticated lepidopteran whose larvae are killed at a few hundred to about 1,600 spores per millilitre in the laboratory (Table 1) is clearly at high intrinsic risk, and drift of conidia to mulberry foliage or into rearing rooms via leaves, air or personnel is biologically plausible.

Direct evidence for this route is, however, limited and, where it exists, does not uniformly support it. In a molecular tracing study that compared 488 isolates from silkworm cadavers in 13 Chinese provinces with 327 isolates from pine caterpillars, where the fungus had been released as an insecticide, the authors concluded that white muscardine in southern Chinese silkworm populations was not caused by artificial release or by natural epizootics of the fungus (Chen *et al.*, 2015). This suggests that indigenous fungal populations around rearing houses are an important source of infection independent of biopesticide use. The two observations are compatible: silkworm rearing already faces a constant background risk from indigenous *B. bassiana*, and the incremental risk from large-scale releases has not been quantified. Recommendations to treat mulberry plantations with low-persistence antifungal substances (Savu & Şapcaliu, 2024) raise the reverse question of how such treatments affect fungal biocontrol agents used on neighbouring crops. A further complication is that the

fungus is used deliberately to produce the traditional Chinese medicine *Bombyx batryticatus* by infecting fourth- and fifth-instar silkworm larvae (Liu *et al.*, 2025); premises for this purpose need to be strictly separated from rearing houses.

Detection and management

Detection. Diagnosis has traditionally relied on clinical signs and microscopy: elastic cadavers that develop chalky conidial growth under humid conditions confirm the disease, and in asymptomatic cases laboratory examination can reveal vegetative hyphae (Savu & Şapcaliu, 2024). For isolation, Sabouraud maltose agar with 1% yeast extract supported the best growth and sporulation of *B. bassiana* among seven media, with sporulation within about three days (Venkataramana Reddy, 1978). Molecular identification is now used to confirm the fungus isolated from suspected white muscardine cases (Park *et al.*, 2024), and PCR-based and other molecular diagnostics for silkworm pathogens are increasingly available (Bebitha *et al.*, 2016; Deepika *et al.*, 2024). Image-based deep-learning models for detecting diseased silkworms are also being developed (Shi *et al.*, 2024), although their application to muscardine specifically requires validation.

Prevention and control. No treatment reliably cures infected larvae, so control is preventive. Recommended measures include thorough cleaning and disinfection of rearing houses and equipment before every rearing, daily removal of diseased and suspect larvae and cadavers followed by burning, disinfection of the larval body and litter with antifungal preparations after each moult, and the use of chlorinated lime spread under beds and on walkways to reduce moisture (Savu & Şapcaliu, 2024). Microclimate management is central: temperature of 22–26 °C, relative humidity below 75%, adequate ventilation and screened

doors and windows are advised (Savu & Şapcaliu, 2024). Diseased lots should be isolated at once to protect other lots (Savu & Şapcaliu, 2024).

Host resistance. Longer-term options include breeding and biotechnology. Transcriptomic comparisons of resistant and susceptible strains have identified candidate genes for molecular breeding (Xing *et al.*, 2017). A mulberry-derived bacterium, *Mammaliicoccus sciuri*, colonises the silkworm cuticle after moulting and feeding and secretes a chitinolytic lysozyme that completely inhibited *B. bassiana* spore germination in vitro and improved larval protection against topical infection (Zhao *et al.*, 2024). This opens the possibility of probiotic or feed-based interventions that support natural cuticular defences.

Knowledge gaps and conclusions

B. bassiana harms sericulture through a well-understood but hard-to-interrupt cycle: highly infective conidia, rapid cuticular and gut invasion, near-total late-instar mortality, cocoon failure and reduced egg output. Its impact depends strongly on humidity, temperature, silkworm strain and fungal isolate. Several gaps limit risk management. First, most quantitative data on stage and instar susceptibility come from a small, 1978 study of a few isolates and should be repeated with current hybrids and well-characterised biopesticide strains. Second, the extent to which commercial *B. bassiana* products contribute to silkworm outbreaks has not been measured directly outside the Chinese tracing studies, and comparable work in India, Europe and other producing regions is needed. Third, the effect of sublethal doses on growth, cocoon and silk quality is poorly defined. Fourth, there are no widely accepted buffer distances or timing rules for applying fungal biopesticides near mulberry and rearing houses. Research combining strain-level genotyping of field

isolates, dose–response tests with commercial formulations, and co-management guidelines for farmers and extension services would allow the benefits of biological control to be retained without endangering silk production.

Declarations

Conflict of interest- The authors have no relevant financial or non-financial interests to disclose. Data availability No new datasets were generated. All data discussed are from the cited publications.

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