Slow Bee Paralysis Virus

Comprehensive Findings Review

Synthesised from the abstracts and full texts of the most-cited and representative papers within a harvested corpus of 177 records (164 open access). A curated review of landmark findings, not an exhaustive catalogue. SBPV is the least-studied of the core honey bee viruses. Generated 2026-06-12.

Executive Summary

Slow bee paralysis virus (SBPV) is a positive-sense, single-stranded RNA virus of the family **Iflaviridae**. Despite the name, it is unrelated to the (dicistrovirus) acute and chronic paralysis viruses. SBPV causes a paralysis that classically begins with the **anterior two pairs of legs** and kills affected adult bees roughly ten days after infection. Its most distinctive feature is its **rarity and restricted distribution**: in the largest survey to date its natural prevalence was under 2%, with positives found only in England and Switzerland. Like DWV, SBPV is transmitted by the mite *Varroa destructor*, which is thought to underlie what localised outbreaks do occur. This document synthesises the findings by theme and catalogues the landmark studies.

Family	Iflaviridae; positive-sense ssRNA, ~9.5 kb, single ORF, polyadenylated (de Miranda 2010)
Strains	Two strains 'Rothamsted' and 'Harpenden', 83% nt / 94% aa identity (de Miranda 2010)
Disease	Paralysis starting in the front two pairs of legs; death ~10 days post-infection
Prevalence	<2% across 847 colonies in 162 apiaries / 5 EU countries — very rare (de Miranda 2010)
Distribution	Positives found only in England and Switzerland in that survey
Vector	Varroa destructor — associated with mite-infested colonies
Note	Naming is historical; SBPV is NOT related to ABPV/KBV/IAPV or to CBPV

Taxonomy and the Misleading Name

Slow bee paralysis virus is a member of the family **Iflaviridae** — the same family as deformed wing virus and sacbrood virus — and is therefore **not** related to the acute bee paralysis / Kashmir / Israeli acute paralysis dicistroviruses, nor to chronic bee paralysis virus, despite the shared word 'paralysis'. The name reflects the clinical course (a slowly developing paralysis), not the virus's phylogeny. Complete genome sequencing showed the SBPV genome is approximately 9.5 kb with a single open reading frame, flanked 5′ and 3′ UTRs and a naturally polyadenylated 3′ tail — the organization typical of iflaviruses (de Miranda 2010).

Strains and Genetic Variation

Two distinct SBPV strains have been characterised, named 'Rothamsted' and 'Harpenden', which are 83% identical at the nucleotide level and 94% at the amino-acid level, with the variation distributed unevenly across the genome (de Miranda 2010). Notably, the two strains were found to co-exist at different proportions within independently propagated virus preparations, indicating that mixed infections and within-host strain competition occur even in this otherwise rare virus (de Miranda 2010).

Pathology

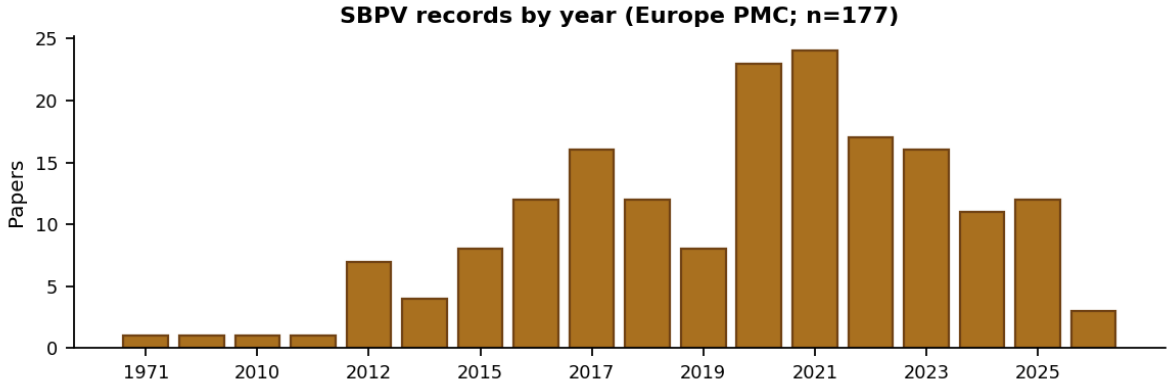
SBPV causes a characteristic, slowly progressing paralysis. The classical description is of paralysis that first affects the **anterior two pairs of legs**, rendering the bee unable to walk normally, with death following roughly ten days after infection — a slower course than the rapid kill of acute bee

paralysis virus. As with most bee viruses, infection can remain covert; overt disease and mortality are associated with elevated titres, which in practice arise in colonies where the *Varroa* mite amplifies and transmits the virus.

Rarity, Distribution and the Varroa Link

SBPV's defining epidemiological feature is its **rarity**. In the largest survey to date — 847 colonies across 162 apiaries in five European countries — natural prevalence was under 2%, and positive samples were found **only in England and Switzerland**, in colonies with variable degrees of *Varroa* infestation (de Miranda 2010). This restricted, patchy distribution contrasts sharply with the near-ubiquitous DWV, SBV and BQCV. The association of the few positive colonies with *Varroa* infestation is consistent with the mite acting as the vector that enables localised SBPV outbreaks, mirroring the *Varroa*-driven emergence pattern seen for its iflavirus relative DWV.

Research Activity



Landmark Study Catalogue

Selected by citation impact and thematic importance. Core studies:

de Miranda 2010 — Genetic characterization of slow bee paralysis virus of the honeybee (2010, J. General Virology; cited 66)

Sequenced two SBPV strains and assessed natural prevalence across Europe.

- SBPV genome ~9.5 kb, single ORF, polyadenylated — typical Iflaviridae organization.
- Two strains ('Rothamsted', 'Harpenden') are 83% identical (nt) / 94% (aa), variation uneven across the genome.
- The two strains co-existed at different proportions in independent preparations.
- Natural prevalence <2% across 847 colonies / 162 apiaries / 5 EU countries; positives only in England and Switzerland, in Varroa-infested colonies.

None · doi:10.1099/vir.0.022434-0

Supporting studies:

Beaurepaire 2020 — Diversity and Global Distribution of Viruses of the Western Honey Bee (review) (2020, Insects; cited 132)

Synthesised global diversity and distribution of A. mellifera viruses.

- Places SBPV among the recognised but much rarer and more geographically restricted bee viruses.
- Contrasts SBPV's limited distribution with the globally dominant DWV/SBV/BQCV.

PMC7240362 · doi:10.3390/insects11040239

McMahon 2015 — A sting in the spit: widespread cross-infection of RNA viruses across wild and managed bees (2015, J. Animal Ecology; cited 209)

National survey of RNA-virus prevalence in honey bees and bumblebees, incl. SBPV.

- Surveyed SBPV among a panel of honey bee RNA viruses across managed and wild bees.
- Found that honey bee RNA viruses, generally, are shared with sympatric wild bumblebees.

PMC4832299 · doi:10.1111/1365-2656.12345

Manley 2015 — Emerging viral disease risk to pollinating insects (review) (2015, J. Applied Ecology; cited 112)

Reviewed drivers of viral spillover from managed to wild pollinators.

- Frames SBPV within the set of fast-evolving RNA viruses with potential to jump between pollinator hosts.
- Highlights Varroa and commercial bee use as drivers of viral disease emergence.

PMC4415536 · doi:10.1111/1365-2664.12385

Scope note: studies excluded as off-topic

- **Cornman 2012 / Dainat 2012 / Vanengelsdorp 2009**: CCD/colony-loss studies — SBPV is one of many or absent from focus.
- **Lanzi 2006 / Highfield 2009 / McMahon 2016**: DWV-specific papers surfaced via shared surveys.
- **General RNAi / virome-method papers**: Methodology or non-bee virome studies matched via 'virus'/'paralysis' keywords.

References

de Miranda 2010. *Genetic characterization of slow bee paralysis virus of the honeybee.* J. General Virology (2010). doi:10.1099/vir.0.022434-0

Manley 2015. *Emerging viral disease risk to pollinating insects (review).* J. Applied Ecology (2015). doi:10.1111/1365-2664.12385 · PMC4415536

McMahon 2015. *A sting in the spit: widespread cross-infection of RNA viruses across wild and managed bees.* J. Animal Ecology (2015). doi:10.1111/1365-2656.12345 · PMC4832299

Beaurepaire 2020. *Diversity and Global Distribution of Viruses of the Western Honey Bee (review).* Insects (2020). doi:10.3390/insects11040239 · PMC7240362
