JOURNAL OF MULTIDISCIPLINARY SCIENCES AND INNOVATIONS
VOLUME 04, ISSUE 09
MOPNTHLY JOURNALS
ISSN NUMBER: 2751-4390
IMPACT FACTOR: 9,08

COMPARATIVE ANALYSIS OF BM64 AND BM60 GENES REVEALS DUAL STRATEGIES IN BMNPV INFECTION AND RESISTANCE IN BOMBYX MORI

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Review article

"BmNPV Bm60 is a key target gene used by a resistant strain of Bombyx mori to inhibit BmNPV proliferation" and "Bombyx mori nucleopolyhedrovirus (BmNPV) Bm64 is required for BV production and per os infection"

Background

The silkworm Bombyx mori serves as an excellent model for exploring the molecular interactions between host and virus, particularly Bombyx mori nucleopolyhedrovirus (BmNPV), a pathogen responsible for severe economic losses in sericulture. Recent findings on viral and host gene functions have deepened our understanding of this complex system. Among these, Bm64, a conserved baculovirus core gene, plays a critical role in the production of budded viruses (BVs) and occlusion-derived viruses (ODVs), influencing the efficiency of per os infection. On the other hand, the host gene Bm60 has emerged as a key antiviral target used by resistant silkworm strains to suppress BmNPV replication. Comparative studies between the susceptible strain 871 and its resistant counterpart 871C revealed that inhibition of Bm60 expression effectively blocked viral DNA replication and late gene transcription, thereby preventing viral proliferation. Together, these studies suggest a dual-layer mechanism in the B. mori – BmNPV interaction: viral genes like Bm64 promote infection and virion formation, while host-regulated pathways involving Bm60 act as defense barriers to viral propagation. Understanding these opposing molecular strategies provides valuable insights for the genetic improvement of silkworm resistance and contributes to the broader study of host–virus coevolution in Lepidoptera.

Introduction (review)

Silkworm (Bombyx mori), an essential insect for global sericulture and a classic model organism for lepidopteran research, has long fascinated me for its delicate balance between productivity and vulnerability. Despite its tremendous economic importance, this species often faces devastating viral infections that lead to massive cocoon yield losses. Among these, Bombyx mori nucleopolyhedrovirus (BmNPV) is the most destructive, capable of rapidly spreading through silkworm populations and remaining difficult to control even with modern management strategies.

Over the past decade, I have been particularly intrigued by how certain silkworm strains naturally resist BmNPV infection. Numerous studies have identified antiviral genes such as Bmlipase-1, BmSP-2, BmNOX, and BmSTING, each contributing to the defense system through digestive enzymes, oxidative pathways, or innate immune signaling. Moreover, the application of omics technologies has revealed dozens of differentially expressed genes in resistant strains, linking antiviral activity to processes such as apoptosis, detoxification, and intracellular transport. Yet, despite these advances, the central molecular mechanism that defines silkworm resistance to BmNPV infection remains only partially understood.

While exploring this question, I became particularly drawn to two genes that represent opposite sides of the host–virus interaction: Bm60 and Bm64. The Bm60 gene, a viral early gene, is strongly suppressed in resistant silkworm strains such as 871C. Its inhibition prevents viral DNA replication and late gene transcription, acting as a natural "molecular brake" on BmNPV proliferation. In contrast, Bm64, a core baculovirus gene conserved across all sequenced genomes, appears to play an indispensable role in producing budded viruses (BVs) and

occlusion-derived viruses (ODVs). Without it, per os infection cannot proceed effectively. Interestingly, even though Bm64 deletion mutants can still replicate viral DNA, they fail to spread systemically within the host, highlighting its specific role in virion formation rather than genome replication.

This duality — where Bm64 promotes infection while Bm60 is suppressed by the host to block replication — perfectly illustrates the complex evolutionary battle between B. mori and BmNPV. By reviewing these two mechanisms side by side, I aim to better understand how viral genes coordinate infection strategies and how silkworms have evolved molecular countermeasures. Such knowledge not only deepens our understanding of host–virus coevolution in Lepidoptera but also provides valuable clues for breeding virus-resistant silkworm lines and developing biological control approaches against related pest species.

Chapter

Methods and Reflections

While analyzing both studies, I found their experimental approaches remarkably complementary — one focused on the host's defensive strategy, while the other dissected the virus's offensive mechanism. The first study compared the susceptible strain 871 and the near-isogenic resistant strain 871C of Bombyx mori, both of which were maintained under identical laboratory conditions. What fascinated me most was how two genetically similar strains could behave so differently when challenged by Bombyx mori nucleopolyhedrovirus (BmNPV). The researchers conducted both oral and subcutaneous infections, and their results were astonishing — the resistant strain 871C showed nearly ten thousand times higher LD_{50} than 871. This alone demonstrated that resistance in silkworms can be dramatically influenced by molecular-level regulation rather than broad physiological traits.

As I read this, I imagined how these larvae might respond internally — perhaps through precise gene silencing or a quick immune signal that shuts down viral replication before it spreads. The discovery that 871C suppressed the expression of the viral early gene Bm60 made perfect sense to me. It was like the host had learned to "mute" a key viral command, blocking the replication process right from its initiation. Such a targeted defense mechanism felt both elegant and powerful — a natural molecular intelligence evolved through constant viral pressure.

The second study, however, turned my attention to the virus's own strategy. It focused on Bm64, a highly conserved baculovirus core gene, and its crucial role in the production of budded viruses (BVs) and occlusion-derived viruses (ODVs). I found it fascinating that when Bm64 was deleted, the virus could still replicate its DNA, yet it lost its ability to spread or infect orally. This reminded me of a "silent warrior" — a virus that can build its genetic army but cannot send it into battle without Bm64. The researchers used sophisticated techniques — RT-PCR, 5′ RACE, electron microscopy, and recombinant virus construction — all to show how vital Bm64 is for virion morphogenesis and oral infectivity.

By combining insights from both papers, I began to see an intricate molecular dialogue between the silkworm and BmNPV. On one side, Bm60 suppression acts as the host's defense brake, halting viral transcriptional cascades. On the other, Bm64 ensures successful virion formation, driving the infection forward. It feels almost poetic — one gene trying to silence the infection, another striving to sustain it. Reading both studies side by side, I could sense the evolutionary tension between survival and adaptation.

To me, these experiments go far beyond technical data — they illustrate a deep biological balance. The resistant strain 871C does not simply "fight" the virus; it understands its rhythm and intercepts it at a critical molecular point. Meanwhile, the virus, through genes like Bm64,

continues to refine its infection machinery. This dynamic battle — precise, invisible, and ancient — is what makes studying Bombyx mori and BmNPV so endlessly fascinating to me.

Chapter
Discussion

The more I delved into these two studies, the more I realized how fascinatingly complex the interaction between Bombyx mori and Bombyx mori nucleopolyhedrovirus (BmNPV) truly is. It feels like watching an ancient, silent battle between the host and the pathogen — each side evolving its own molecular strategy to outsmart the other.

From the host's perspective, I was struck by how multi-layered the defense mechanisms are. Silkworms rely not only on physical barriers such as digestive enzymes and the peritrophic membrane, but also on finely tuned innate immune responses, including apoptosis and RNA interference. When I read that the midgut juice of resistant strains contains proteins with direct antiviral activity, it immediately reminded me of the elegant simplicity of insect immunity — minimalistic yet highly efficient. The fact that 871C could suppress viral gene expression, particularly Bm60, showed that the silkworm doesn't just defend passively; it actively targets the virus at the genetic level. It's as if the silkworm "knows" where to strike — right at the command center of the virus.

On the viral side, the role of Bm64 completely absorbed my attention. The paper revealed that this gene, conserved across all baculoviruses, is indispensable for the formation of budded and occlusion-derived viruses (BVs and ODVs). I found it especially intriguing that deleting Bm64 didn't affect DNA replication — meaning the virus could still prepare its genetic material — but it couldn't assemble infectious particles or spread orally. It was like a virus stripped of its mobility: alive at the molecular level but biologically paralyzed.

Electron microscopy images showing properly formed nucleocapsids but failed egress made me imagine those trapped virions inside the nucleus — ready to invade but unable to escape. The authors' observation that Bm64 localized to the "ring zone" of infected nuclei fascinated me even more. I could almost visualize this ring zone as the virus's "control room," where assembly and exit are coordinated. Many viral envelope proteins like P33 and Ac76 are also found there, forming a molecular highway for viral egress. The suggestion that Bm64 interacts with these proteins to facilitate BV envelope formation made me think of how precisely viruses manage their internal logistics.

But what makes this even more captivating is how different baculoviruses appear to have slightly adapted Bm64's function to their own infection strategies. While the AcMNPV version (Ac78) is crucial for BV production, BmNPV's Bm64 seems more specialized in ensuring oral infectivity. This subtle difference, despite the gene's conserved nature, reminded me that evolution doesn't just conserve — it refines. Each species reshapes even its "core" genes to fit its ecological niche and infection rhythm.

When I connect these findings, I see a perfect balance between attack and defense. Bm64 pushes the virus to replicate and spread efficiently, while Bm60 suppression and host proteins like 30K-8 counteract this process, restricting viral propagation. It's a molecular dialogue — not pure conflict, but a form of co-adaptation. The silkworm has learned to silence the virus where it matters most, and the virus, in turn, has refined its tools to survive that resistance.

Reading these studies didn't feel like going through data; it felt like unfolding a story — a biological chess game where every move counts. It made me wonder how many other genes are silently playing their parts in this microscopic struggle. Each figure, each PCR curve, and each electron micrograph deepened my curiosity. The more I learned, the more I realized: BmNPV

and Bombyx mori are not enemies in a simple sense — they are two co-evolving partners, locked in a delicate, centuries-long dialogue of adaptation and survival.

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