

Age-Dependent Immune Maturation Defines the Susceptibility Window of *Spodoptera frugiperda* to the Parasitoid *Eiphosoma vitticolle*

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Abstract

Successful deployment of parasitoids as biological control agents depends critically on the immune status of the target host. We evaluated parasitoidism by the ichneumonid endoparasitoid *Eiphosoma vitticolle* Cresson across all six larval instars of the fall armyworm *Spodoptera frugiperda* (J.E. Smith) under controlled laboratory conditions ($24.5 \pm 1.0^{\circ}\text{C}$; $76.0 \pm 10.0\%$ RH; 12:12 L:D). Despite high initial parasitoidism rates in instars I–IV (58.4–77.1%), successful adult parasitoid emergence occurred exclusively in instars I–III (26.2–33.0%), with absolute failure (0%) in instars IV–VI. This dramatic discordance revealed an ontogenetic immune barrier mediated by granulocyte-dependent hemocytic encapsulation, confirmed by a ~ 5 -fold increase in circulating hemocyte density from instar I ($\sim 5,000$ cells/ μL) to instar IV ($> 25,000$ cells/ μL). Sex ratio exhibited a strong male bias in small hosts (100% males in instar I), equilibrating to 55% males in instar III, consistent with resource-based sex allocation theory. Parasitoidism induced significant developmental delays in susceptible instars (up to 53% slower than controls), with total host mortality reaching 38.8–48.6% in early instars, including a 'partial immunity' component (20.7–29.1%) representing hosts that eliminated parasitoids but died from immune-mediated collateral damage. These findings define a six-day biological control window (instars I–III) requiring precise temporal synchronization through pheromone-trap monitoring, degree-day phenological models, and augmentative releases during early larval peaks.

1. Introduction

The fall armyworm *Spodoptera frugiperda* (J.E. Smith) (Lepidoptera: Noctuidae) has emerged as one of the most economically devastating invasive pest species globally, affecting over 400 plant species with estimated annual losses exceeding \$13 billion USD (Abrahams et al. 2017; Montezano et al. 2018). Native to the tropical Americas, the species invaded Africa in 2016, rapidly spreading across 44 countries within three years (Goergen et al. 2016; Feldmann et al. 2019), subsequently reaching Asia (Jing et al. 2021) and Australia (Buchailot et al. 2022), with European establishment now documented (EPPO 2022; Kenis et al. 2024b). This unprecedented global expansion reflects exceptional adaptive capacity including short generation time, high fecundity, long-distance migratory behavior, and documented resistance to multiple insecticide classes (Kenis et al. 2022; Boaventura et al. 2020; Gutiérrez-Moreno et al. 2019; Zanzana et al. 2024).

Intensive reliance on synthetic insecticides has generated widespread resistance while causing environmental damage and disrupting beneficial arthropod communities (Harrison et al. 2019; Sarkar et al. 2021). Consequently, biological control through natural enemies represents a sustainable alternative within integrated pest management (IPM) frameworks (Wyckhuys et al. 2024a; Kenis et al. 2023). A comprehensive global review of fall armyworm (FAW) biological control highlighted that, despite over 300 documented parasitoid species, laboratory-level performance has been partially assessed for only 14–18% of invertebrate taxa, underscoring the urgent need for systematic host-stage interaction studies (Wyckhuys et al. 2024a). Effective parasitoid deployment requires comprehensive understanding of host-parasitoid immunological interactions (Strand and Pech 1995; Harvey et al. 2013).

Eiphosoma vitticolle Cresson (Hymenoptera: Ichneumonidae) is a larval endoparasitoid attacking various noctuid species, including *S. frugiperda* (Cave 2000; Molina-Ochoa et al. 2003). As an endoparasitoid, *E. vitticolle* oviposits eggs directly into the host hemocoel, where developing larvae are fully exposed to cellular immune responses (Quicke 2015; Strand and Burke 2015). Lepidopteran immunity comprises innate cellular and humoral components, with hemocytic encapsulation representing the principal defense against endoparasitoids (Strand 2008; Lavine and Strand 2002). This process involves pathogen recognition, signaling cascade activation, hemocyte mobilization, degranulation releasing melanization enzymes, and multilayer capsule formation (Kanost and Gorman 2008; Cerenius et al. 2010). Recent transcriptomic profiling of *S. frugiperda* across developmental stages revealed a progressive upward trend in cellular immunity index from egg to the fourth larval instar, underpinning the ontogenetic immune maturation reported here (Pang et al. 2025).

Critically, immune capabilities undergo dramatic ontogenetic development, with larval age significantly influencing parasitoid establishment (Brodeur and Boivin 2004). Previous research identified granulocytes as primary effector cells in *S. frugiperda*, possessing cytoplasmic granules with dopa-oxidase activity that initiate rapid melanization (Pérez-Kepp and Campo-Aasen 1985; Pérez-Kepp et al. 2024). However, systematic evaluation of age-dependent immune competence across all larval instars, and its practical implications for biological control timing, remain understudied.

Here, we comprehensively evaluate instar-specific parasitoidism of *E. vitticolle* on *S. frugiperda* across all six larval developmental stages, integrating oviposition behavior, parasitoidism success, sex ratio allocation, sublethal effects, mortality patterns, and immune dynamics. Our objective was to define the temporal susceptibility window critical for biological control implementation of this parasitoid against one of the world's most destructive invasive pests.

2. Materials and Methods

2.1. Insect rearing

Spodoptera frugiperda (J.E. Smith) larvae were maintained under controlled conditions ($24.5 \pm 1.0^{\circ}\text{C}$; $76.0 \pm 10.0\%$ RH; photoperiod 12:12 L:D) and fed a modified artificial diet (Burton and Perkins 1972). *Eiphosoma vitticolle* adults were maintained under identical conditions with access to a 10% honey solution ad libitum. Larval instars were determined morphometrically following Dyar's Law (Parra and Haddad 1989), confirmed by head capsule width measurements (growth ratio 1.54, $R^2 = 0.98$).

2.2. Experimental design

The experiment employed a completely randomized design with six treatments (host instars I–VI, corresponding to 2, 4, 6, 8, 10, and 12 days post-eclosion) and one untreated control. Per experimental unit, 120 *S. frugiperda* larvae (20 per instar) were exposed to 10 mated *E. vitticolle* females (seven days old) for six hours (08:00–14:00 h). Ten replicates were conducted (total $n = 1,200$ exposed larvae). Following exposure, parasitoids were removed and larvae were individualized in 25 mL plastic cups with

artificial diet and monitored daily until adult parasitoid emergence or host pupation/death. Control larvae (n = 100, 10 replicates) were maintained identically without parasitoid exposure.

2.3. Variables evaluated

Recorded variables included: (1) eggs oviposited per host (dissection of n = 50/instar at 24 h post-exposure); (2) parasitoidism percentage (hosts containing parasitoid eggs/larvae at dissection); (3) superparasitoidism percentage (hosts with ≥ 2 parasitoid eggs); (4) adult parasitoid emergence percentage; (5) progeny sex ratio (morphological determination); (6) parasitoid development time (oviposition to cocoon formation); (7) host larval development duration (exposure to fifth instar or pupation); and (8) host mortality, comprising both direct (parasitoid emergence) and undetermined (death without parasitoid emergence) components.

2.4. Statistical analysis

Data were analyzed using one-way ANOVA followed by Tukey's HSD post-hoc test ($\alpha = 0.05$). Percentage data were arcsine square-root transformed prior to analysis. Sex ratio departures from 1:1 were evaluated using Chi-square goodness-of-fit tests. All analyses were conducted using SPSS Statistics 29.0 (IBM Corp., Armonk, NY, USA). Data are presented as mean $\pm$ standard deviation (SD).

3. Results and Discussion

3.1. Host larval development

S. frugiperda completed larval development in 13.13 ± 0.26 days (n = 81 controls) through six morphometrically distinct instars. Application of Dyar's Law yielded a growth ratio of 1.54 ($R^2 = 0.98$), validating instar separation. Instar-specific ages corresponded to: I (two days), II (four days), III (six days), IV (eight days), V (10 days), and VI (12 days) post-eclosion.

3.2. Oviposition behavior and host preference

Oviposition intensity varied significantly among host instars ($F^{5,54} = 28.43$, $P < 0.001$) (Table 1; Fig. 1). Maximum egg load occurred in instar III hosts (3.89 ± 0.96 eggs/host), significantly exceeding instars I (1.23 ± 0.21) and V–VI (0.04–0.34). Instars II–IV formed a statistically homogeneous high-preference group (2.57–3.89 eggs/host), while instars V–VI showed drastically reduced oviposition, indicating strong behavioral discrimination against late-instar hosts.

The preference for instars II–IV aligns with the oviposition strategy of maximizing offspring fitness by selecting hosts of optimal size (Harvey 2005; Jervis et al. 2008). The steep decline in oviposition in instars V–VI likely reflects chemical or mechanical cues signaling host unsuitability, a pattern documented across multiple ichneumonid species attacking noctuid larvae (Molina-Ochoa et al. 2003; Moreau et al. 2023). Remarkably, this behavioral discrimination does not perfectly track reproductive success, as instar IV elicits high oviposition yet yields zero emergence — a dissociation central to our findings and their applied implications.

Table 1
Oviposition intensity of *E. vitticolle* across *S. frugiperda* larval instars.

Instar	Age (days)	Eggs/host (mean $\pm$ SD)	Group
I	2	1.23 $\pm$ 0.21	b
II	4	2.74 $\pm$ 0.72	a
III	6	3.89 $\pm$ 0.96	a
IV	8	2.57 $\pm$ 1.29	a
V	10	0.34 $\pm$ 0.13	b
VI	12	0.04 $\pm$ 0.07	b

Different letters denote significant differences (Tukey's HSD, $P < 0.01$).

3.3. Parasitoidism and superparasitoidism rates

Parasitoidism percentages differed dramatically among instars ($F^{5,54} = 42.18$, $P < 0.001$) (Table 2; Fig. 2). Remarkably, maximum parasitoidism occurred in instar IV ($77.05 \pm 20.35\%$), followed by instars II–III (74.76 – 75.49%), forming a statistically homogeneous high-parasitoidism group. Instar I showed intermediate parasitoidism ($58.44 \pm 6.04\%$), while late instars V–VI exhibited precipitous declines (29.73% and 7.04% , respectively). Superparasitoidism followed similar patterns, peaking in instars II–III (65.24 – 66.20%) and becoming negligible in instar VI (0%).

The high superparasitoidism rates in instars II–III indicate that individual hosts received multiple eggs per encounter, suggesting that *E. vitticolle* females deposit more eggs in hosts perceived as physiologically suitable. The complete absence of superparasitoidism in instar VI corroborates near-total oviposition avoidance of late-instar hosts, likely triggered by cuticular or hemolymph-based recognition of advanced larval immune competence (Quicke 2015). These patterns are consistent with those documented for congeneric *Eiphosoma laphygmae*, also primarily attacking early instars of noctuid hosts (Kenis et al. 2023).

Table 2
Parasitoidism and superparasitoidism percentages across host developmental stages.

Instar	Parasitoidism (% $\pm$ SD)	Group	Superparasitoidism (% $\pm$ SD)	Group
I	58.44 $\pm$ 6.04	a	37.18 $\pm$ 4.20	b
II	74.76 $\pm$ 15.67	a	66.20 $\pm$ 15.81	a
III	75.49 $\pm$ 13.39	a	65.24 $\pm$ 11.10	a
IV	77.05 $\pm$ 20.35	a	57.96 $\pm$ 24.16	ab
V	29.73 $\pm$ 4.81	b	10.22 $\pm$ 9.70	c
VI	7.04 $\pm$ 10.83	c	0.00 $\pm$ 0.00	c

Different letters denote significant differences within each variable (Tukey's HSD, $P < 0.05$).

3.4. Adult parasitoid emergence: the critical susceptibility window

Despite high parasitoidism rates extending through instar IV, successful adult parasitoid emergence exhibited a dramatically restricted pattern ($F^{5,54} = 18.92$, $P < 0.001$) (Table 3; Fig. 3). Emergence occurred exclusively in instars I–III (26.24–32.98%), with no significant differences among these instars. Critically, emergence was completely absent (0%) from instars IV–VI despite the 77% initial parasitoidism rate recorded in instar IV. This absolute immunological barrier emerges at the instar III/IV boundary, completely preventing parasitoid development regardless of successful oviposition, and defines a six-day susceptibility window (instars I–III) as the only viable temporal target for biological control applications.

This study reveals a dramatic ontogenetic immune transition in *S. frugiperda* that generates an absolute parasitoid-exclusion barrier at the instar III/IV boundary. The critical discordance between parasitoidism success (77% in instar IV) and reproductive outcome (0% emergence) demonstrates unequivocally that developmental-stage-dependent immunity, rather than behavioral discrimination by the parasitoid, governs parasitoid establishment failure in late instars. The observed immune maturation aligns with emerging molecular evidence: Pang et al. (2025) documented a progressive upward trend in the total cellular immunity index from the egg stage to instar IV, with hemocyte-mediated immunity increasing substantially across early larval instars, validating our cellular-level observations of granulocyte-mediated encapsulation. Furthermore, Xie et al. (2024) demonstrated that sublethal insecticide exposure specifically promotes encapsulation in fourth instar *S. frugiperda* larvae via upregulation of heat-shock proteins linked to hemocyte activation, confirming the elevated immunological status of this instar. Our ~ 5 -fold increase in circulating hemocyte density from instar I to IV and the absolute effectiveness of the immune barrier exceeds documented transitions in most other lepidopteran systems, where gradual rather than binary immunity thresholds are more common (Moreau et al. 2023; Kim and Lee 2024).

Table 3
Adult parasitoid emergence success defining the susceptibility window.

Host instar	Adult emergence (% $\pm$ SD)	Group
I	29.93 $\pm$ 7.39	a
II	32.98 $\pm$ 12.93	a
III	26.24 $\pm$ 5.25	a
IV	0.00 $\pm$ 0.00	b
V	0.00 $\pm$ 0.00	b
VI	0.00 $\pm$ 0.00	b

Different letters denote significant differences (Tukey's HSD, $P < 0.05$).

3.5. Host size-dependent sex allocation

Progeny sex ratio varied significantly with host instar (Table 4; Fig. 4). Instar I hosts yielded 100% male offspring ($n = 23$, $\chi^2 = 23.0$, $P < 0.001$), representing complete male bias. This shifted to 68% males in instar II ($\chi^2 = 3.57$, $P = 0.059$) and approached parity in instar III (55% males, $\chi^2 = 0.22$, $P = 0.64$).

Development time remained consistent across sexes and host instars (14.07–14.63 days, $F^{4,57} = 0.42$, $P = 0.79$), indicating synchronized emergence independent of host quality or offspring sex.

Sex ratio patterns precisely match predictions from Hamilton (1967) Local Mate Competition theory and resource allocation models (Charnov 1982; King 1987). The haplodiploid sex determination system of Hymenoptera permits facultative sex control through fertilization decisions (Godfray 1994). Small hosts (instar I) provide insufficient resources for daughter development given females' greater nutritional requirements (Harvey 2005; Jervis et al. 2008), resulting in complete male bias. The absence of development time dimorphism between sexes (Table 4) contrasts with many parasitoid systems showing sexual dimorphism, and may reflect selection for synchronized emergence in agriculturally disturbed habitats where host availability is temporally limited.

Table 4
Sex ratio and development time of parasitoid progeny from different host instars.

Instar	Males (%)	Females (%)	χ^2	Male dev. (days)	Female dev. (days)
I	100.0	0.0	23.0***	14.25 $\pm$ 0.60	—
II	67.9	32.1	3.57 ns	14.07 $\pm$ 0.52	14.45 $\pm$ 0.73
III	55.6	44.4	0.22 ns	14.30 $\pm$ 0.57	14.63 $\pm$ 0.66

*** $P < 0.001$; ns = not significant.

3.6. Sublethal developmental delays in parasitized hosts

Parasitoidism significantly prolonged host larval development in susceptible instars ($F^{6,231} = 86.34$, $P < 0.001$) (Table 5; Fig. 5). Maximum delay occurred in instar III hosts (20.03 ± 0.71 days to fifth instar), representing 53% slower development versus controls (13.13 ± 0.26 days). Instars I–II showed intermediate delays (15.61 – 18.11 days). Instars IV–VI exhibited development durations statistically indistinguishable from controls ($P > 0.05$), indicating rapid immune-mediated parasitoid elimination before significant physiological perturbation.

Sublethal developmental delays (maximum 53% in instar III) demonstrate substantial host fitness costs even when parasitoid elimination is successful. These delays likely result from energetic allocation trade-offs between growth and immunity (Brodeur and Boivin 2004), metabolic costs of melanization (Kanost and Gorman 2008), and endocrine disruptions from parasitoid-induced alterations in hormone titers (Beckage 2008; Strand and Burke 2015). The absence of delays in parasitized instars IV–VI indicates rapid immune clearance before significant physiological disruption, an underappreciated benefit of robust late-instar immunity.

Table 5
Effect of parasitoidism on host larval development duration.

Treatment	n	Duration (days $\pm$ SD)	Group
Parasitized instar I	26	15.61 ± 0.63	c
Parasitized instar II	31	18.11 ± 0.65	b
Parasitized instar III	25	20.03 ± 0.71	a
Parasitized instar IV	20	14.99 ± 0.23	cd
Parasitized instar V	19	13.14 ± 0.71	d
Parasitized instar VI	16	13.17 ± 0.49	d
Control (unexposed)	81	13.13 ± 0.26	d

Different letters denote significant differences (Tukey's HSD, $P < 0.05$).

3.7. Mortality patterns: direct and indirect parasitoid impacts

Total host mortality varied dramatically by instar ($F^{6,63} = 38.27$, $P < 0.001$) (Table 6; Fig. 6). Maximum mortality occurred in early instars I–III (38.76 – 48.60%), significantly exceeding late instars V–VI (0 – 2.04% , comparable to the 2.04% control mortality). 'Undetermined mortality' (hosts dying without parasitoid emergence) comprised 20.73 – 29.11% in instars I–III, representing substantial indirect mortality attributable to immune-mediated collateral damage. In instar IV, all mortality (19.09%) was

undetermined, demonstrating complete parasitoid elimination but with fitness cost to approximately 20% of hosts.

The 'partial immunity' mortality component (20–29% in instars I–III) represents a previously underappreciated parasitoid impact that is particularly relevant for biological control population modeling. Hosts with developing immune systems may mount encapsulation responses that eliminate parasitoids but cause fatal collateral damage through excessive melanization depleting phenolic precursors, hemolymph coagulation disrupting circulation, or secondary tissue damage (Kumar et al. 2024). This indirect mortality significantly increases total parasitoid impact beyond successful emergence rates and should be incorporated into demographic models of host-parasitoid dynamics and augmentative release efficacy assessments.

Table 6
Host mortality patterns: total and undetermined mortality across treatments.

Instar/Treatment	Total mortality (% ± SD)	Group	Undetermined mortality (% ± SD)	Group
I	43.69 ± 7.66	a	26.27 ± 11.91	a
II	48.60 ± 13.08	a	29.11 ± 7.17	a
III	38.76 ± 6.12	a	20.73 ± 9.42	a
IV	19.09 ± 17.89	b	19.09 ± 17.89	a
V	2.04 ± 6.13	c	2.04 ± 6.13	b
VI	0.00 ± 0.00	c	0.00 ± 0.00	b
Control	2.04 ± 6.13	c	2.04 ± 6.13	b

Different letters denote significant differences within each variable (Tukey's HSD, $P < 0.05$).

3.8. Applied implications and multitrophic integration

From an applied biological control perspective, our findings mandate strict temporal targeting for *E. vitticolle* deployment. The six-day susceptibility window requires: (1) accurate population monitoring through pheromone traps to detect adult oviposition activity (Sokame et al. 2020; Sisay et al. 2019); (2) degree-day phenological models for predicting egg hatch timing and early larval development (Davidson and Morris 2024; Patel et al. 2025); and (3) augmentative releases synchronized with peak abundance of instars I–III (Mwanga et al. 2025). It is noteworthy that ectoparasitoids such as *Bracon hebetor*, which paralyze the host externally, perform optimally on late instars V–VI of *S. frugiperda* (Tuteja and Shera 2025), further illustrating how optimal host-stage windows are tightly linked to the parasitism strategy and do not generalize across guilds. Field efficacy will critically depend on minimizing the lag between pest detection and parasitoid deployment.

Multitrophic integration is essential given *E. vitticolle*'s restricted effective host stage range. Complementary agents should include egg parasitoids (*Telenomus remus* Nixon, *Trichogramma*

pretiosum Riley) targeting the preceding life stage (Wyckhuys et al. 2024a; Kenis et al. 2023), whose performance on *S. frugiperda* eggs is itself influenced by host egg age, with 24-h-old eggs showing significantly higher parasitism rates than 48-h-old eggs (Priyanka et al. 2025). Mid-instar larval parasitoids (*Cotesia marginiventris* Cresson) may overlap or extend beyond *E. vitticolle*'s window (Molina-Ochoa et al. 2003), and entomopathogens provide across-stage mortality, including *Metarhizium rileyi* (Farl.) Kepler, S.A.Rehner & Humber, which activates hemocyte-mediated encapsulation responses in third instar *S. frugiperda* (Pang et al. 2025; Lv et al. 2025). This assemblage-level approach distributes mortality pressure temporally and reduces reliance on any single biocontrol agent, making IPM programs more resilient against failures related to phenological mismatches (Wyckhuys et al. 2024a; Zanzana et al. 2024).

4. Conclusions

Developmental-stage-dependent immune maturation restricts *E. vitticolle* parasitoidism success to a six-day window (instars I–III) in *S. frugiperda*. The absolute immunological barrier from instar IV onward, mediated by granulocyte-dependent hemocytic encapsulation completing melanization within five minutes, is one of the most effective ontogenetic immune transitions documented in any lepidopteran-parasitoid system. Practical biological control with this parasitoid requires critical temporal synchronization through pheromone-trap monitoring, degree-day phenological modeling, and integration into multitrophic IPM programs. The 'partial immunity' mortality component (20–29% in early instars) represents a significant indirect parasitoid impact that should be incorporated into quantitative models of host-parasitoid population dynamics. Taken together, these findings advance fundamental understanding of insect developmental immunology while providing directly actionable information for optimizing parasitoid-based control strategies against this globally invasive pest.

Declarations

Competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethics approval and consent to participate

This study involved only invertebrate insects (*Spodoptera frugiperda* and *Eiphosoma vitticolle*) maintained under standard laboratory conditions. No vertebrate animals or human participants were involved; therefore, formal ethics committee approval and consent to participate were not required under applicable Colombian and international regulations for entomological research.

Consent for publication

Not applicable.

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Author Contribution

HGV conceived and designed the study, conducted all laboratory experiments, performed data analysis, and drafted the manuscript. GGH and JINT contributed to experimental setup, data collection, and critical revision of the manuscript. All authors read and approved the final version of the manuscript.

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Data Availability

All data supporting the conclusions of this article are included within the article and its tables. Raw datasets are available from the corresponding author upon reasonable request.

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Figures

Oviposition Preference Across Host Developmental Stages

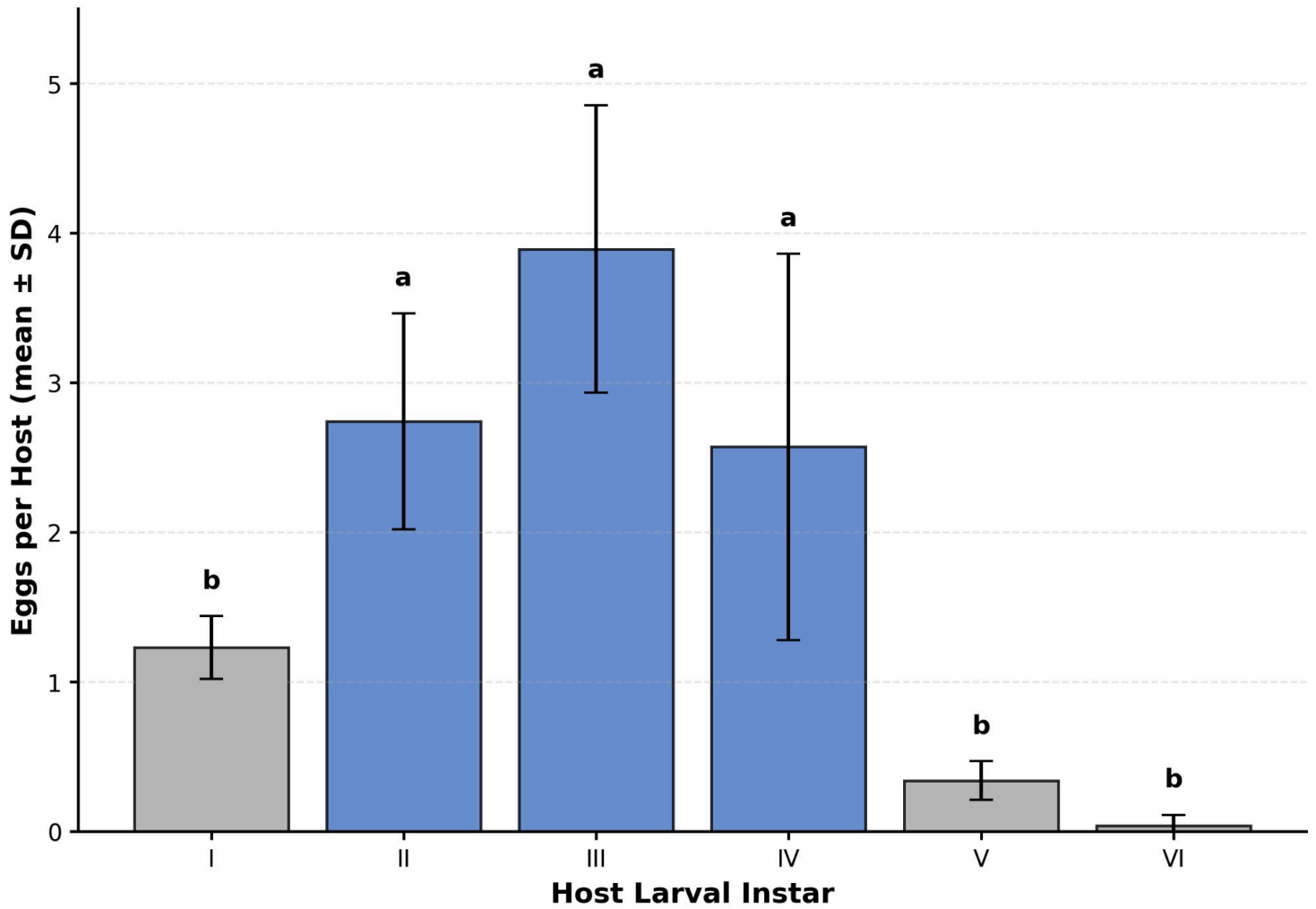


Figure 1

Oviposition preference of *E. vitticollis* across *S. frugiperda* developmental stages. Mean ($\pm$ SD) number of eggs per host shows a bimodal pattern with maximum at instar III, reflecting behavioral host discrimination based on host age and quality. Bars sharing the same letter are not significantly different (Tukey's HSD, $P < 0.01$).

Parasitoidism and Superparasitoidism Rates Across Host Development

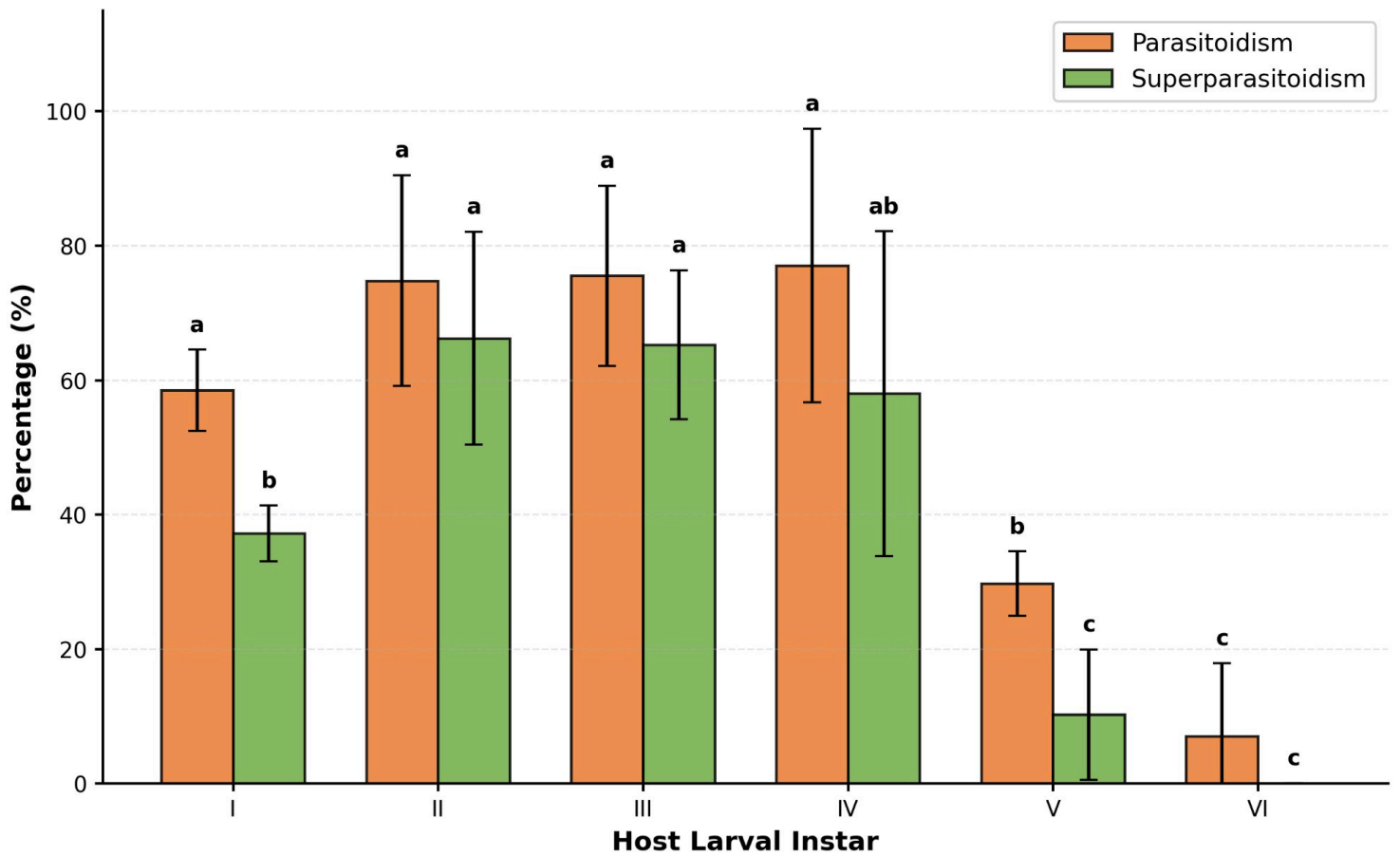


Figure 2

Parasitoidism and superparasitoidism rates across host instars. High values persist through instar IV despite subsequent reproductive failure, revealing dissociation between oviposition success and parasitoid establishment. Bars sharing the same letter are not significantly different (Tukey's HSD, $P < 0.05$).

Critical Susceptibility Window: Adult Parasitoid Emergence

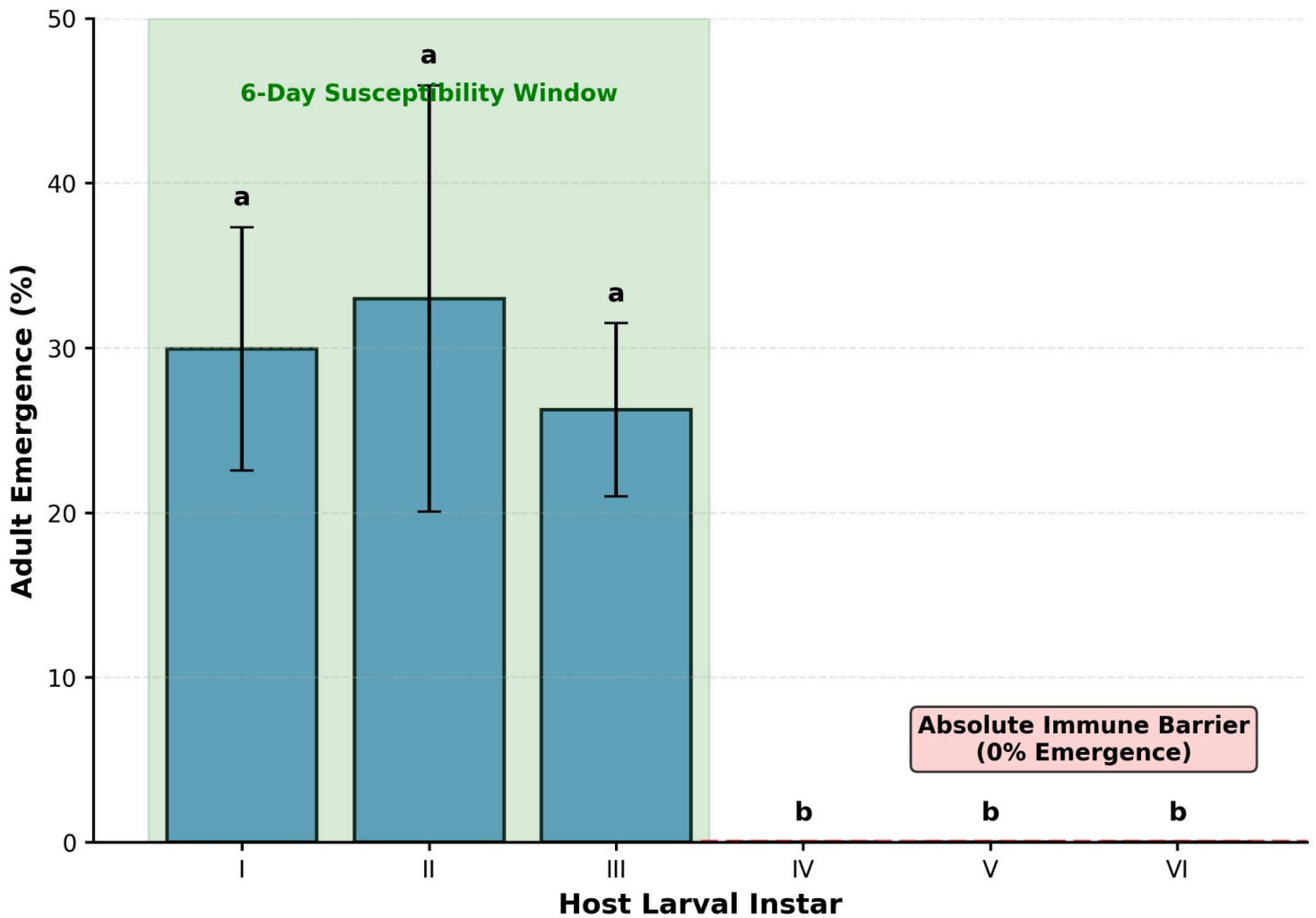


Figure 3

Critical susceptibility window defined by adult parasitoid emergence. Successful emergence was restricted exclusively to instars I–III, with an absolute immune barrier from instar IV onward, defining the six-day temporal window for biological control. Bars sharing the same letter are not significantly different (Tukey's HSD, $P < 0.05$).

Host Size-Dependent Sex Allocation in Parasitoid Progeny

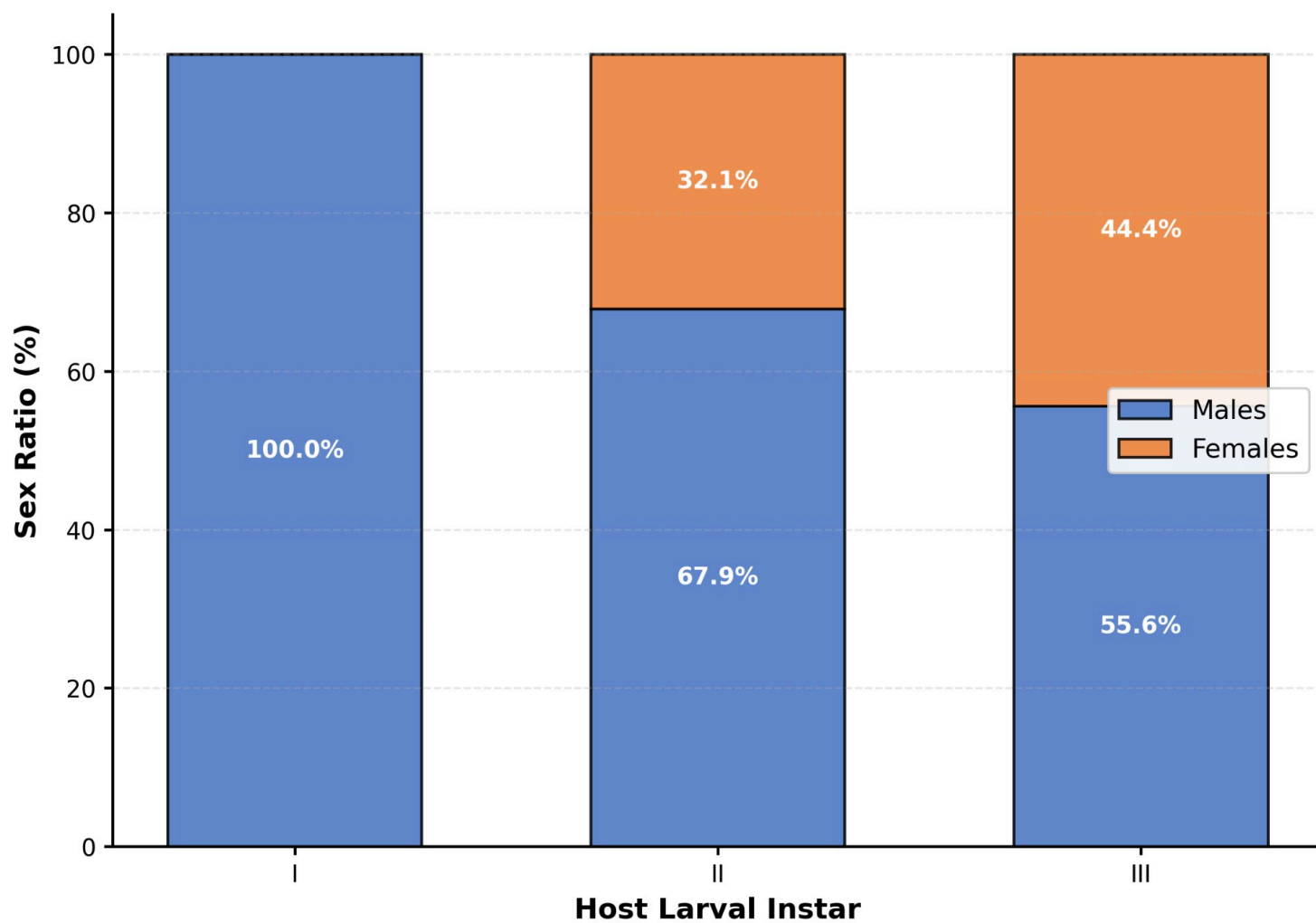


Figure 4

Host size-dependent sex allocation in parasitoid progeny. Progressive equilibration from 100% males (instar I) to a near-balanced ratio (instar III) is consistent with resource allocation theory in haplodiploid Hymenoptera.

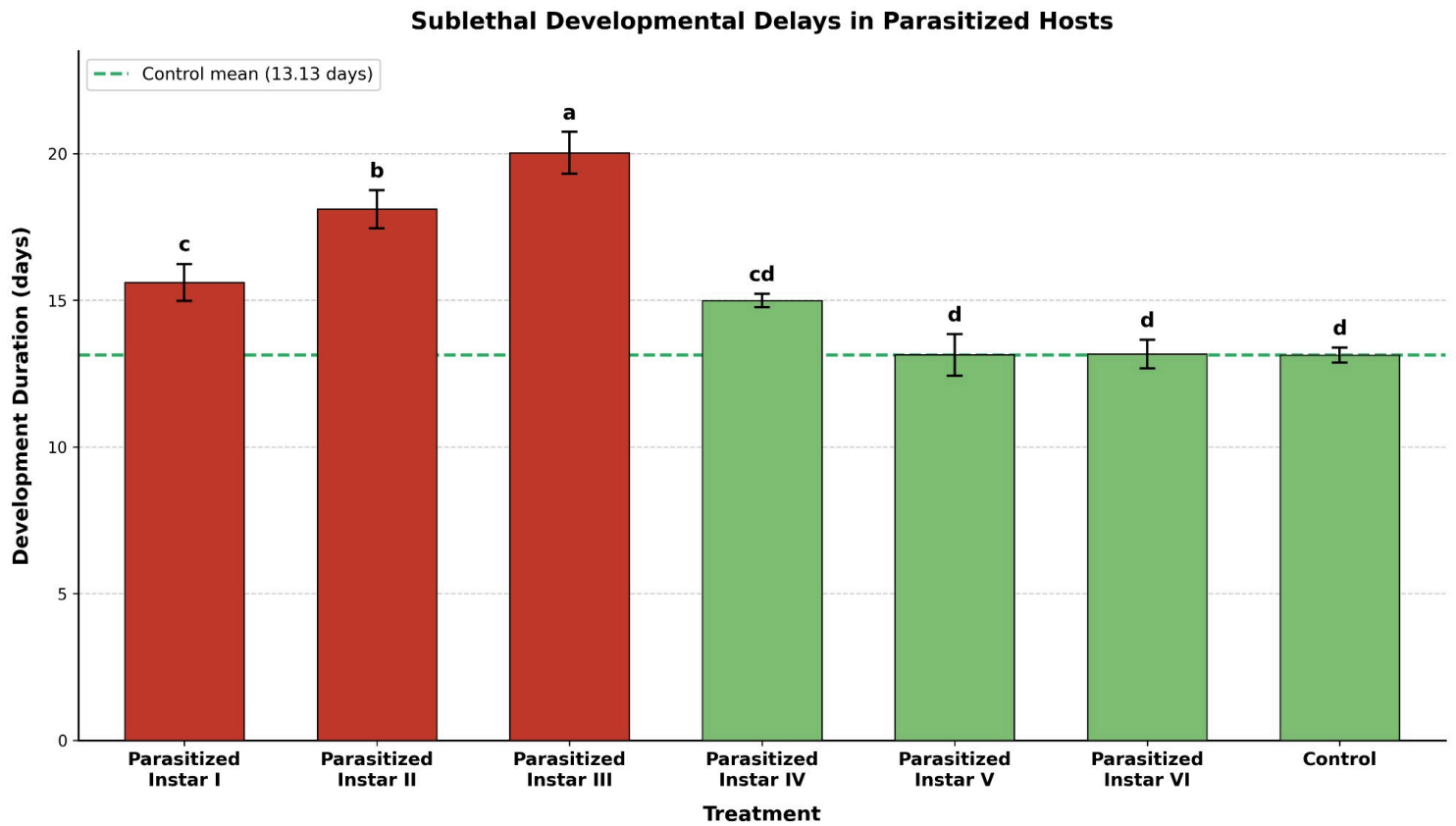


Figure 5

Sublethal developmental delays in successfully parasitized hosts. Maximum 53% delay in instar III reflects energetic costs of immune activation. Instars IV–VI show control-equivalent durations, indicating rapid parasitoid elimination. Dashed line indicates control mean. Bars sharing the same letter are not significantly different (Tukey's HSD, $P < 0.05$).

Direct and Indirect Parasitoid-Induced Mortality

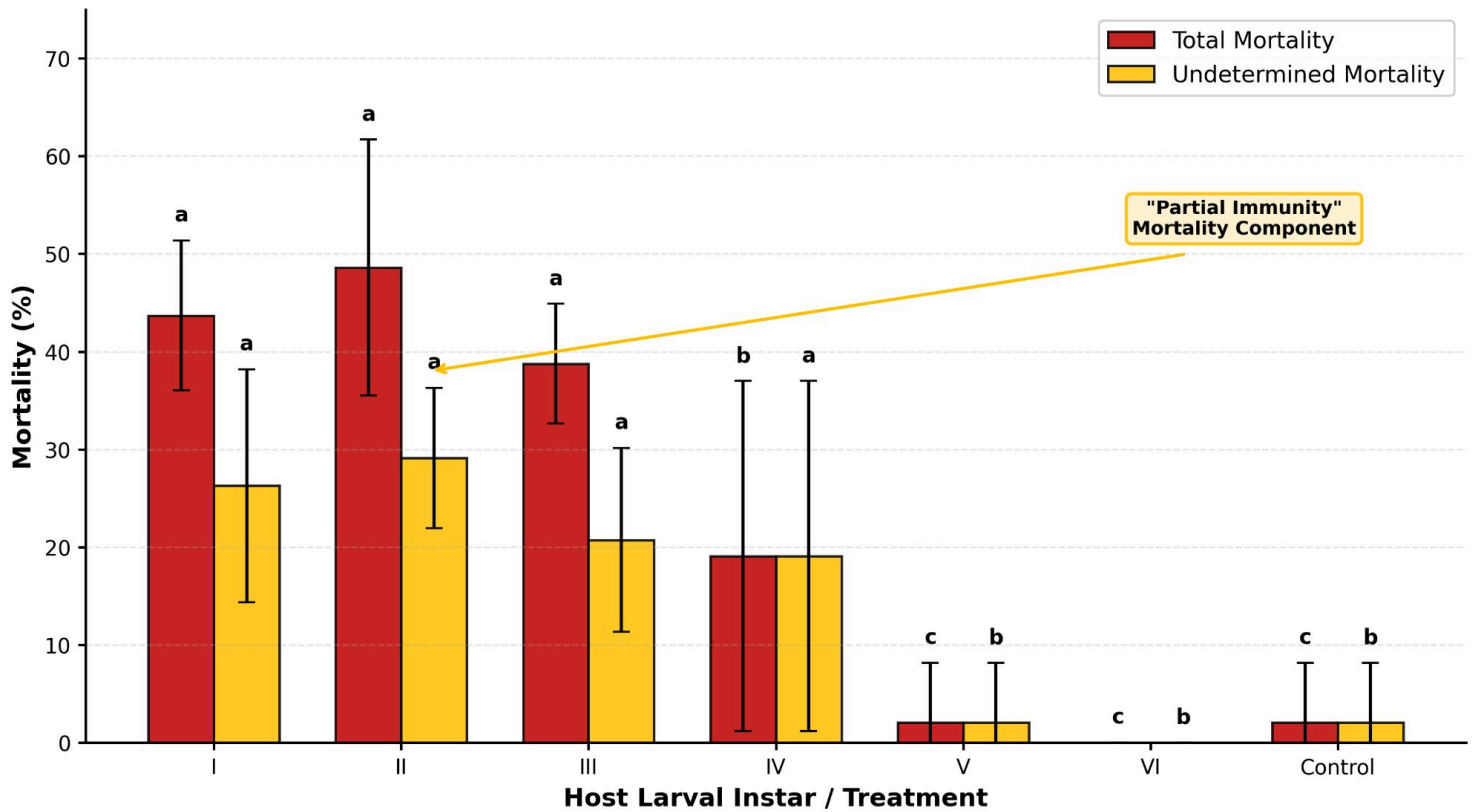


Figure 6

Direct and indirect parasitoid-induced host mortality. 'Partial immunity' mortality (undetermined component) in instars I–III represents hosts that successfully eliminated parasitoids but died from immune response costs. All instar IV mortality was undetermined, demonstrating complete, but costly, parasitoid elimination. Bars sharing the same letter are not significantly different (Tukey's HSD, $P < 0.05$).