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ADULT-STAGE EXPRESSION OF EMAMECTIN BENZOATE RESISTANCE IN *Spodoptera frugiperda* (J. E. SMITH) FOLLOWING ORAL EXPOSURE

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ABSTRACT
The fall armyworm, Spodoptera frugiperda (J. E. Smith) (Lepidoptera: Noctuidae), has developed resistance to insecticides, including emamectin benzoate (EB). Most EB resistance studies use larval assays, whereas expression remains less characterized. We evaluated adult responses to exposure to EB-treated 10% sucrose solution (0.2 g L^{-1}) in susceptible (SS), resistant (RR), and reciprocal F_1 populations at one and three days of age. Mortality was assessed at 24 and 48 h. At 24 h, SS mortality was 100% at both ages, whereas pooled RR mortality was 70% and 65% in one- and three-day-old adults, respectively. Reciprocal F_1 mortality ranged from 85–95%. Fisher's exact tests showed lower RR mortality than SS at both ages ($P = 0.020$ and 0.008) and after pooling ages and sexes ($P < 0.001$). By 48 h, mortality reached 100% in all groups except three-day-old RR females (90%). Resistance expression was most clearly detected during the first 24 h.

KEYWORDS: fall armyworm; insecticide resistance; emamectin benzoate; adult susceptibility; oral exposure; reciprocal crosses

INTRODUCTION
The fall armyworm, *Spodoptera frugiperda* (J. E. Smith), is one of the most important invasive agricultural pests of the modern era. Native to the Americas, the species was first detected in Africa in 2016 and subsequently expanded rapidly across Asia and other regions (Goergen et al. 2016). In India, its detection was reported in May 2018 followed by rapid establishment in maize-producing regions (Sharanabasappa et al. 2018 ; Suby et al. 2020). Its importance is associated not only with its broad host range but also with high reproductive capacity, migratory ability, and the capacity to adapt to diverse agroecosystems (Montezano et al. 2018; Nagoshi et al. 2019).
Management of *S. frugiperda* has relied heavily on insecticides, particularly where biological and cultural control measures are insufficient. The intensive and repeated use of insecticides creates strong selection pressure favoring resistant individuals. Insecticide resistance is a major evolutionary challenge in agricultural pest management because resistance can develop through target-site modification, enhanced detoxification, reduced penetration, behavioral mechanisms, or combinations of these processes (Hawkins et al. 2019). The problem is particularly relevant in *S. frugiperda*, which has developed resistance to several insecticide classes and Bt proteins in different geographic regions.
The rapid development of insecticide resistance in *S. frugiperda* is associated with several biological characteristics of the species, including high fecundity, multiple generations per year, dispersal capacity and intensive exposure to insecticides. Resistance management consequently requires not only monitoring changes in susceptibility but also understanding the inheritance, fitness consequences and expression of resistance across the insect's life cycle (Sparks and Nauen 2015; Hawkins et al. 2019).
Emamectin benzoate (EB) is an avermectin insecticide widely used against lepidopteran pests. Its insecticidal activity is associated primarily with disruption of ligand-gated chloride channels, particularly glutamate-gated chloride channels, resulting in neurological dysfunction and eventual paralysis and mortality. Molecular studies in *S. frugiperda* have demonstrated an important role for the glutamate-gated chloride channel in EB susceptibility, providing evidence that alterations in this pathway can influence insecticide response. EB has become particularly important in *S. frugiperda* management. In India, EB was among the insecticides given approval for management of the invasive pest following its establishment in maize, and susceptibility monitoring has subsequently demonstrated variation among geographical populations (Kulye et al. 2021; Kumar et al. 2022). Kulye et al. (2021) evaluated 47 *S. frugiperda* populations collected from different regions of India and established baseline susceptibility to several insecticide classes, providing an important reference for subsequent resistance monitoring. (Kumar et al. 2022) subsequently reported geographical variation in insecticide susceptibility together with molecular variation among Indian *S. frugiperda* populations.
The ability of *S. frugiperda* to develop resistance to EB has been demonstrated experimentally. (Muraro et al. 2021) selected an EB-resistant strain under laboratory conditions and reported approximately 2340-fold resistance after ten generations of selection. Reciprocal crosses indicated incomplete dominance and the authors estimated

a polygenic contribution to resistance. The resistant and heterozygous populations also showed high survival on EB-treated maize, demonstrating that laboratory resistance could translate into survival under plant exposure conditions. Field evolution of EB resistance has subsequently been demonstrated in *S. frugiperda*. Muraro et al. (2022) reported a substantial decline in EB susceptibility among Brazilian populations sampled over time, with resistance ratios reaching approximately 632-fold in populations collected during 2019–2021. Increased larval survival at diagnostic concentrations and on EB-treated maize further supported the occurrence of field-evolved resistance. These observations demonstrate that EB resistance is not simply a laboratory phenomenon and emphasize the importance of continued characterization of resistance phenotypes.

An additional consideration in resistance biology is the stage at which resistance is expressed. Most insecticide resistance assays in *S. frugiperda* are conducted using larvae because larvae are the principal crop-damaging stage and can be conveniently exposed using diet-overlay, leaf-dip or treated-plant assays. Consequently, resistance phenotypes are usually described in terms of larval mortality or larval survival. Although such assays are essential for resistance monitoring, they provide limited information about whether an established resistance phenotype remains detectable after adult emergence. Adult *S. frugiperda* have a different biology and exposure ecology from larvae. Adults primarily obtain carbohydrate resources from liquid food sources, and their feeding behavior creates an opportunity to assess insecticide susceptibility through oral exposure to treated sucrose. An adult oral-exposure assay therefore provides a complementary route of exposure that differs from conventional larval diet-overlay bioassays.

The distinction is important because resistance is not necessarily expressed identically across life stages. Physiological processes associated with insecticide absorption, distribution, metabolism, excretion and target-site sensitivity can vary during development. Fitness costs associated with resistance can likewise vary according to genetic background, host plant and environmental conditions (Padovez et al. 2022; Barbosa et al. 2020). Studies of resistant *S. frugiperda* populations have documented changes in developmental rate, survival, longevity, fecundity and population growth, demonstrating that resistance can influence biological performance beyond the immediate toxicological response (Padovez et al. 2022; Barbosa et al. 2020). The present study therefore examined the adult response of susceptible, EB-resistant and reciprocal F_1 populations of *S. frugiperda* following oral exposure to EB-treated sucrose solution. One-day-old and three-day-old adults were evaluated to determine whether resistance-associated differences were detectable at different early adult ages. The study was specifically designed to characterize adult-stage phenotypic expression and the temporal pattern of mortality following oral exposure. It was not intended to re-estimate larval resistance ratios or inheritance parameters.

MATERIALS AND METHODS

Insect populations

Four populations of *S. frugiperda* were used which were maintained at division of Entomology, ICRISAT, Hyderabad. The different populations were : susceptible (SS), EB-resistant (RR), SS♀ × RR♂, and RR♀ × SS♂ reciprocal F_1 populations. The SS population was maintained without insecticide selection, while the RR population had been developed through laboratory selection with EB. Reciprocal F_1 populations were produced by crossing susceptible and resistant parental adults. The populations were maintained under controlled laboratory conditions prior to the adult exposure experiment. Because the purpose of the present study was to examine adult-stage expression of an established resistance phenotype, previously determined larval resistance ratios and inheritance statistics are not presented as primary results in this study.

Adult oral-exposure bioassay

Adult susceptibility was evaluated using an oral exposure system in which EB was supplied through treated sucrose solution. A plastic cup containing a cotton wick soaked in 10% (w/v) sucrose solution served as the lower component of the exposure chamber. EB was incorporated into the sucrose solution at 0.2 g/L, corresponding to one-half of the concentration used as the experimental field-dose reference in the associated resistance studies. The upper component consisted of a glass cylinder with its lower end covered with fine mesh. The cylinder was positioned over the lower cup so that adults could access the sucrose solution through the mesh while remaining confined within the chamber. One-day-old and three-day-old adult males and females from each population were tested. One male and one female were released into each chamber, and 10 replicate chambers were maintained for each population and adult age. Corresponding untreated control chambers containing 10% sucrose solution without EB were maintained in parallel. The chambers were maintained at 26 ± 1 °C, 75 ± 5% relative humidity and a 12:12 h light photoperiod.

Mortality correction

Control mortality at 24 h was zero for all population, age and sex combinations. At 48 h, control mortality ranged from 0 to 20% depending on population and age. Treatment mortality was corrected using Abbott's formula (Abbott 1925) where control mortality exceeded 5%:

$$\text{Corrected mortality} = \frac{T - C}{100 - C} \times 100$$

where T represents mortality in the treated group and C represents mortality in the corresponding untreated control.

Statistical analysis

Mortality at 24 h was analyzed as a categorical survival outcome. Male and female observations were pooled within each population and adult-age group, giving 20 adults per population within each age category. Fisher's

exact test was used to compare mortality between populations because the response variable was binomial and the experimental sample size was limited. Comparisons between RR and SS populations were performed separately for one-day-old and three-day-old adults. Additional pooled comparisons were performed across both ages and sexes. Statistical analyses were performed using R version 4.4.3. Differences were considered significant at $P < 0.05$.

RESULTS

Adult mortality at 24 h

A pronounced difference in mortality among population backgrounds was observed during the first 24 h of EB exposure (Table 1; Fig. 1). Mortality in the SS population was 100% in both one-day-old and three-day-old adults. Complete mortality was observed in both sexes. In contrast, RR adults showed substantially lower mortality. Among one-day-old RR adults, mortality was 80% in males and 60% in females, giving a pooled mortality of 70%. Among three-day-old RR adults, mortality was 70% in males and 60% in females, corresponding to a pooled mortality of 65%.

The reciprocal F_1 populations showed intermediate mortality. For one-day-old SS♀ × RR♂ adults, mortality was 90% in males and 100% in females, resulting in 95% pooled mortality. The RR♀ × SS♂ population showed 100% mortality in males and 80% in females, corresponding to 90% pooled mortality. At three days of adult age, both reciprocal F1 populations showed 85% pooled mortality. The SS♀ × RR♂ population showed 80% mortality in males and 90% in females, whereas the RR♀ × SS♂ population showed 90% mortality in males and 80% in females. Fisher's exact test showed that mortality in RR adults was significantly lower than mortality in SS adults at one day ($P = 0.020$) and three days ($P = 0.008$). When both adult ages and sexes were pooled, RR mortality remained significantly lower than SS mortality ($P < 0.001$). Pooled RR mortality was also significantly lower than that of the SS♀ × RR♂ cross ($P = 0.027$), whereas the difference between RR and RR♀ × SS♂ was not significant ($P = 0.059$). Thus, the strongest population-level discrimination occurred during the first 24 h, with the RR population consistently showing greater survival than the susceptible population. Fisher's exact test showed that mortality in RR adults was significantly lower than in SS adults at one day ($P = 0.020$) and three days ($P = 0.008$). When both adult ages and sexes were pooled, RR mortality remained significantly lower than SS mortality ($P < 0.001$) and was also lower than the SS♀ × RR♂ cross ($P = 0.027$), whereas the difference between RR and RR♀ × SS♂ was not significant ($P = 0.059$).

Adult mortality at 48 h

The separation between population backgrounds decreased markedly with prolonged exposure. At 48 h, mortality reached 100% in all population, age and sex combinations except three-day-old RR females, which showed 90% raw mortality. Because control mortality for this group was zero, Abbott correction did not change the value. Thus, the major discriminatory feature of the assay was the rate of mortality during the first 24 h rather than complete protection from mortality throughout the 48-h exposure period (Table 2).

Survival and delayed mortality

The 24-h results can also be expressed in terms of survival. Among one-day-old adults, survival was 0% in SS, 30% in RR, 5% in SS♀ × RR♂, and 10% in RR♀ × SS♂. Among three-day-old adults, survival was 0% in SS, 35% in RR, and 15% in each reciprocal F_1 population. The greatest increase in mortality between 24 and 48 h was observed in the RR population. The resistant phenotype under these conditions was expressed primarily as prolonged survival or delayed mortality, rather than absolute survival following EB exposure. Adults were considered dead when they failed to respond to gentle prodding or were unable to right themselves after being placed on the dorsal surface. Mortality was recorded at 24 and 48 h following exposure.

DISCUSSION

The present study demonstrates that an established EB-resistant phenotype in *S. frugiperda* remains detectable during the adult stage following oral exposure to EB-treated sucrose. The most pronounced difference occurred during the first 24 h of exposure, when all susceptible adults died whereas 30–35% of RR adults remained alive. The difference was statistically significant at both one and three days of adult age. The result complements the extensive body of EB resistance research in *S. frugiperda*, which has primarily examined larval susceptibility. Laboratory selection experiments have demonstrated that the species can evolve high levels of EB resistance, while reciprocal crosses have shown incomplete dominance and a polygenic contribution to resistance (Muraro et al. 2021). Field monitoring subsequently confirmed that EB susceptibility can decline substantially in natural populations, with resistance ratios reaching hundreds-fold in Brazilian populations (Muraro et al. 2022).

The present experiment addresses a different question from these concentration-response and inheritance studies. Rather than quantifying the magnitude or genetic architecture of larval resistance, it asks whether the established resistant phenotype remains expressed after adult emergence when the insect encounters EB through an oral liquid-food route. The significant difference between RR and SS adults provides evidence that resistance-associated survival is not restricted to the larval stage under the conditions tested.

This finding is particularly relevant because adult exposure ecology differs from larval exposure ecology. Larvae are directly exposed to insecticide residues on crop foliage, whereas adults are primarily associated with dispersal, mating and feeding on liquid carbohydrate resources. An oral-exposure assay therefore provides a physiologically distinct route for examining the expression of resistance.

The response of the RR population also provides an important insight into the temporal nature of resistance expression. At 24 h, RR mortality was 65–70%, compared with 100% in SS. By 48 h, mortality was essentially complete in most groups. Thus, resistance under the experimental conditions did not prevent EB toxicity indefinitely. Instead, it resulted in a measurable delay in mortality. This distinction is important for interpretation of resistance assays. If mortality had been recorded only at 48 h, the difference between susceptible and resistant populations would have been substantially less apparent. The 24-h observation therefore appears to be the more informative endpoint for discriminating the resistance phenotype in this adult oral-exposure system.

The delayed response is biologically plausible given the mode of action of EB. Avermectin insecticides affect ligand-gated chloride channels, resulting in disruption of neuronal function. Recent molecular evidence in *S. frugiperda* has specifically implicated the glutamate-gated chloride channel in EB susceptibility. Consequently, differences in the magnitude or timing of toxicity could theoretically arise from changes in target-site sensitivity or other processes affecting the internal concentration of active insecticide. However, the present experiment was not designed to distinguish among these mechanisms. This limitation is important because EB resistance is not necessarily attributable to a single mechanism. Muraro et al. (2021) found no strong evidence for a major contribution of the metabolic pathways tested using synergists in their laboratory-selected EB-resistant strain, illustrating that resistance mechanisms can differ among populations. Similarly, broader studies of FAW resistance have demonstrated involvement of metabolic enzymes, target-site changes and other physiological processes depending on the insecticide and genetic background.

The intermediate response of the reciprocal F_1 populations is another notable result. At 24 h, F_1 mortality ranged from 85 to 95%, compared with 100% in SS and 65–70% in RR. This pattern places the F_1 populations between the two parental phenotypes. The result is consistent with the intermediate phenotype expected from the previously characterized resistant and susceptible parental backgrounds. The current experiment was not a concentration-response inheritance assay, and the mortality endpoint was evaluated at a single EB concentration. Therefore, the F_1 response is interpreted here as evidence of an intermediate adult phenotype rather than as an independent genetic estimate.

The similarity between reciprocal F_1 populations is also noteworthy. Both reciprocal crosses produced comparable pooled mortality at three days, and their one-day responses differed only modestly. This indicates that the adult phenotype was broadly similar between the two reciprocal genetic backgrounds. Nevertheless, because the experiment was not specifically designed to test maternal effects or sex-linked inheritance, the results should not be interpreted as independent confirmation of autosomal inheritance. The Indian context makes characterization of EB resistance particularly relevant. EB was among the insecticides approved for FAW management following its establishment in India, creating substantial selection opportunities in maize production systems. Kumar et al. (2022) documented geographical variation in insecticide susceptibility among Indian *S. frugiperda* populations, while Kulye et al. (2021) established baseline susceptibility profiles across multiple Indian populations. Continued monitoring is therefore necessary as insecticide use patterns and resistance frequencies change.

The present study complements such monitoring by demonstrating a possible adult-stage endpoint. Conventional larval assays remain indispensable because larvae are the major crop-damaging stage and the principal target of foliar insecticide applications. However, adult-stage assays may provide additional information about whether resistance-associated phenotypes persist after metamorphosis. The biological consequences of resistance can extend beyond insecticide survival. Studies in *S. frugiperda* have shown that resistance can be associated with changes in developmental duration, longevity, fecundity and population growth, although the magnitude and direction of these costs can depend strongly on genetic background and environmental conditions (Barbosa et al. 2020; Padovez et al. 2022). Barbosa et al. (2020), for example, reported altered developmental and adult biological traits following repeated insecticide selection, whereas Padovez et al. (2022) demonstrated that the apparent fitness cost of resistance could differ substantially depending on genetic background.

These findings are relevant to the interpretation of adult-stage resistance because adult survival may influence subsequent reproductive opportunity and population dynamics. Nevertheless, the present experiment did not measure reproduction after EB exposure and therefore does not establish effects on fecundity, mating success or population growth. Fitness consequences of insecticide resistance can depend on genetic background and environmental conditions (Barbosa et al. 2020; Padovez et al. 2022), so such effects should be tested directly in future adult-exposure studies.

The adult oral-exposure assay used here also has several limitations. First, a single EB concentration was used; therefore, adult LC_{50} values and the quantitative magnitude of resistance cannot be derived from the experiment. Second, the amount of EB actually ingested by individual adults was not quantified. Third, mortality was recorded at two discrete time points rather than continuously, preventing formal estimation of individual survival curves. Finally, the assay was conducted under controlled laboratory conditions and should not be directly equated with field exposure. Despite these limitations, the experiment provides a clear biological observation: the difference between EB-resistant and susceptible *S. frugiperda* populations is detectable during the adult stage and is most evident during the early period following oral exposure. Future studies could strengthen this observation by developing adult concentration-response curves, quantifying ingestion, recording individual time-to-death, and comparing physiological or molecular responses between resistant and susceptible adults.

In particular, a time-to-event approach such as Kaplan–Meier survival analysis would be valuable if individual mortality times were recorded in future experiments. Such an approach could distinguish populations not simply

according to final mortality but according to the rate at which mortality accumulates following exposure. Overall, the findings broaden the phenotypic characterization of EB resistance in *S. frugiperda* from the conventional larval stage to the adult stage. The results do not indicate that adults are immune to EB exposure; rather, they show that resistant adults survive longer during the early exposure period than susceptible adults. This distinction provides a useful framework for future studies investigating the persistence, physiological basis and ecological significance of insecticide resistance across the life cycle.

CONCLUSION

Adult *S. frugiperda* from the EB-resistant population exhibited significantly greater survival during the first 24 h of oral exposure to EB-treated sucrose than adults from the susceptible population. This difference was observed in both one-day-old and three-day-old adults, while reciprocal F_1 populations showed intermediate mortality. The convergence of mortality by 48 h indicates that the principal phenotype was delayed mortality rather than complete protection from EB toxicity. These findings demonstrate detectable adult-stage expression of the EB-resistant phenotype and support further evaluation using adult concentration-response and time-to-event approaches.

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Conflict of Interest

The authors declare that they have no conflict of interest.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

LITERATURE CITED

Abbott, Walter S. 1925. A Method of Computing the Effectiveness of an Insecticide. Journal of Economic Entomology 18 (2): 265–67.

Barbosa, M. G., T. P. P. André, A. D. S. Pontes, S. A. Souza, N. R. X. Oliveira, and P. L. Pastori. 2020. Insecticide rotation and adaptive fitness cost underlying insecticide resistance management for Spodoptera frugiperda (Lepidoptera: Noctuidae) Barbosa et Al. Neotropical Entomology 49 (6): 882–92.

Elias Oliveira Padovez, Fernando, Rubens Hideo Kanno, Celso Omoto, and Aline Sartori Guidolin. 2022. Fitness Costs Associated with Chlorantraniliprole Resistance in Spodoptera frugiperda (Lepidoptera, Noctuidae) Strains with Different Genetic Backgrounds. Pest Management Science 78 (3): 1279–86. https://doi.org/10.1002/ps.6746.

Goergen, Georg, P. Lava Kumar, Sagnia B. Sankung, Abou Togola, and Manuele Tamò. 2016. First Report of Outbreaks of the fall armyworm Spodoptera frugiperda (JE Smith)(Lepidoptera, Noctuidae), a New Alien Invasive Pest in West and Central Africa. PloS One 11 (10): e0165632.

Hawkins, Nichola J., Chris Bass, Andrea Dixon, and Paul Neve. 2019. The Evolutionary Origins of Pesticide Resistance. Biological Reviews 94 (1): 135–55.

Kulye, Mahesh, Sonja Mehlhorn, Debora Boaventura, Nigel Godley, Sreedevi Kodikoppalu Venkatesh, Thimmaraju Rudrappa, Tara Charan, Dinesh Rathi, and Ralf Nauen. 2021. Baseline Susceptibility of Spodoptera frugiperda Populations Collected in India towards Different Chemical Classes of Insecticides. Insects 12 (8): 758.

Kumar, Sandeep, S. B. Suby, Naveen Kumar, J. C. Sekhar, Suresh Nebapure, and G. K. Mahapatro. 2022. Insecticide Susceptibility Vis-à-Vis Molecular Variations in Geographical Populations of Fall Armyworm, Spodoptera frugiperda (JE Smith) in India. 3 Biotech 12 (9): 241.

Montezano, D. G., A. Specht, D. R. Sosa-Gómez, A. Specht, V. F. Roque-Specht, J. C. Sousa-Silva, S. V. Paula-Moraes, J. A. Peterson, and T. E. Hunt. 2018. Host Plants of Spodoptera frugiperda (Lepidoptera: Noctuidae) in the Americas. African Entomology 26 (2): 286–300. https://doi.org/10.4001/003.026.0286.

Muraro, Dionei S., Dyrson De Oliveira Abbade Neto, Rubens H. Kanno, Ingrid S. Kaiser, Oderlei Bernardi, and Celso Omoto. 2021. Inheritance Patterns, Cross-resistance and Synergism in Spodoptera frugiperda (Lepidoptera: Noctuidae) Resistant to Emamectin Benzoate. Pest Management Science 77 (11): 5049–57. https://doi.org/10.1002/ps.6545.

Muraro, Dionei S., Eloisa Salmeron, João VS Cruz, Fernando Semmelroth de Assunção e Amaral, Aline Sartori Guidolin, Antonio Rogerio Bezerra do Nascimento, José B. Malaquias, Oderlei Bernardi, and Celso Omoto 2022. Evidence of Field-Evolved Resistance in Spodoptera frugiperda (Lepidoptera: Noctuidae) to Emamectin Benzoate in Brazil. Crop Protection 162: 106071.

Nagoshi, Rodney N., Isabel Dhanani, R. Asokan, H. M. Mahadevaswamy, Chicknayakanahalli M. Kalleshwaraswamy, Sharanabasappa, and Robert L. Meagher. 2019. Genetic Characterization of Fall Armyworm Infesting South Africa and India Indicate Recent Introduction from a Common Source Population. PLoS One 14 (5): e0217755.

Sharanabasappa, S. D., C. M. Kalleshwaraswamy, R. Asokan, H. M. M. Swamy, M. S. Maruthi, H. B. Pavithra, K. Hegde, S. Navi, S. T. Prabhu, and G. E. Goergen. 2018. First Report of the Fall Armyworm, Spodoptera frugiperda (JE Smith) (Lepidoptera: Noctuidae), an Alien Invasive Pest on Maize in India.
Sparks, Thomas C., and Ralf Nauen. 2015. IRAC: Mode of Action Classification and Insecticide Resistance Management. Pesticide Biochemistry and Physiology 121: 122–28.
Suby, S. B., P. Lakshmi Soujanya, Pranjal Yadava, Jagadeesh Patil, K. Subaharan, G. Shyam Prasad, K. Srinivasa Babu, S. L. Jat, K. R Yathish, Jyothi lakshmi Vadassery, Vinay K. Kalia, N. Bakthavatsalam, J. C. Shekhar and Sujay Rakshit. 2020. Invasion of fall armyworm (Spodoptera frugiperda) in India: nature, distribution, management and potential impact. Current Science. 119(1): 44-51.

Table 1. Adult mortality of *Spodoptera frugiperda* populations following oral exposure to emamectin benzoate (0.2 g L^{-1}) through 10% sucrose solution after 24 h.

Population	Adult age	Mortality (%)	Survival (%)
SS	1-day-old	100.0	0.0
RR	1-day-old	70.0	30.0
SS♀ × RR♂	1-day-old	95.0	5.0
RR♀ × SS♂	1-day-old	90.0	10.0
SS	3-day-old	100.0	0.0
RR	3-day-old	65.0	35.0
SS♀ × RR♂	3-day-old	85.0	15.0
RR♀ × SS♂	3-day-old	85.0	15.0

Note: Mortality values are pooled across males and females (n = 20 adults per population within each age group).

Table 2. Sex- and age-specific mortality of adult *Spodoptera frugiperda* populations following oral exposure (0.2 g L^{-1}) to emamectin benzoate after 48 h.

Population	Age	Sex	Mortality (%)
SS	1-day-old	Male	100
SS	1-day-old	Female	100
RR	1-day-old	Male	100
RR	1-day-old	Female	100
SS♀ × RR♂	1-day-old	Male	100
SS♀ × RR♂	1-day-old	Female	100
RR♀ × SS♂	1-day-old	Male	100
RR♀ × SS♂	1-day-old	Female	100
SS	3-day-old	Male	100
SS	3-day-old	Female	100
RR	3-day-old	Male	100
RR	3-day-old	Female	85.7

SS♀ × RR♂	3-day-old	Male	100
SS♀ × RR♂	3-day-old	Female	100
RR♀ × SS♂	3-day-old	Male	100
RR♀ × SS♂	3-day-old	Female	100

Note: Mortality values are Abbott-corrected where control mortality exceeded 5%. The three-day-old RR female control had zero mortality at 48 h; therefore, the corrected mortality was 90%, identical to the raw mortality.

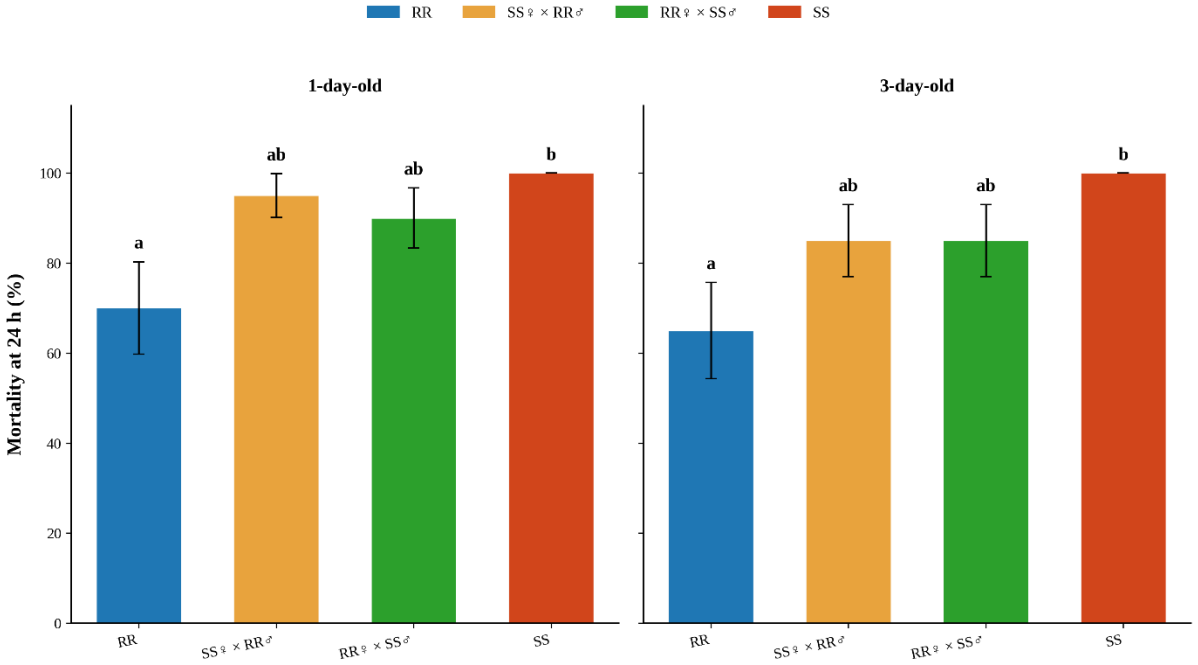


Fig. 1. Mortality of one-day-old and three-day-old adult susceptible (SS), resistant (RR) and F_1 cross (SS♀ × RR♂ and RR♀ × SS♂) *Spodoptera frugiperda* following oral exposure to emamectin benzoate (0.2 g L^{-1}) treated sucrose solution.