
Complications with Controlling Insect Eggs

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Abstract

Eggs are difficult to kill because of the unique structure of the eggshell, comprised of multiple layers that have evolved to allow the embryo to breathe while simultaneously limiting water loss. The eggshell has been shown to be an excellent barrier to insecticides, fungal pathogens, and some fumigants. The insect eggshell contains only a few areas that could allow penetration of insecticides, the aeropyles and micropyles, which seem to be either so few in number or small in size that they do not allow a sufficient amount of insecticide through the eggshell. Resistance is also a contributing factor to control failures of insect eggs. Resistance in eggs has been documented in several insect species and a few studies have shown that some insect eggs produce elevated numbers of enzymes to break down insecticides. This chapter focuses on the structure and respiration of the insect eggshell as a barrier to insecticides and also covers various management strategies against insect eggs. Lastly, we discuss the few documentations of resistance in insect eggs thus far.

Keywords: Insect, egg, eggshell, resistance, management, insecticide

1. Introduction

The predominant reproductive type of most insects is egg laying, called oviparity. There is limited literature on controlling insect eggs, although eggs are considered the most difficult life stage to kill. Examples of this can be found in both agricultural and urban pest species. Oftentimes, the management strategy is to essentially ignore the eggs, wait for them to hatch, and then treat the nymphs that emerge from the eggs because they are easier to kill. During many treatments, the eggs are left unaccounted for because they are not the nuisance stage (they do not bite or feed) and are not as visible as other stages. However, the eggs left behind that were not killed by the treatment will soon begin to hatch and cause a reinfestation. It may take time before the infestation grows to become a problem again, at which time reevaluation or restart of the treatment may be necessary.

Insect eggs have adapted mechanisms to enhance their survival, including the enclosure of the embryo within an eggshell. The eggshell, also referred to as the chorion, is the first line of defense for the developing embryo against environmental stressors. One main environmental stressor that pest insect species have to face is human use of pesticides. The unique structure of the eggshell renders most insecticidal products impenetrable through the eggshell.

Insecticides that do penetrate the eggshell have to reach their target site within the embryo to be effective. A few insects have been shown to have developed enzymatic resistance to insecticides in the egg stage. Eggs treated with insecticides repeatedly were shown to produce high levels of enzymatic activity to break down insecticides. The combination of reduced penetration through the eggshell and pesticide resistance makes eggs an extremely difficult life stage to kill.

2. Insect eggshell morphology

Insect eggs are remarkably structured to provide the developing insect embryo with protection, simultaneously providing a barrier against insecticide penetration. This is evident from the array of structural designs observed on eggshells across insect species, which have specifically evolved to provide the developing insect ultimate protection in the environment. There are many variations in the respiratory structures, shape, size, coloration, and the chorionic structure of eggs, all seemingly evolved to withstand environmental stress. The diversity of eggshells and the structural complexity of the eggshell (Figure 1) are evident in three butterfly eggs in the families Nymphalidae and Saturniidae. The amazing diversity of insect eggs has received little attention from the scientific community. H. E. Hinton's three-volume work on the biology of insect eggs [1] represents one of the most significant contributions to understanding insect eggs.

Various studies have attempted to characterize the insect eggshell, or chorion, and its layers. Eggshell morphological descriptions have revealed that many insect eggs have detailed sculpturing on the outside of the eggshell. The sculpturing is usually comprised of multisided geometrical shapes, arranged on the eggshell in an aesthetically pleasing pattern.

Not all insect eggshells are perfectly symmetrical. For instance, the outer surfaces of the chorion in true bugs oftentimes are geometrical but the shapes are irregular in shape and size. Furthermore, some insect eggs lack the geometrical shapes extending from the eggshell and are completely smooth. The formation and shape of the eggshell is highly dependent on the outline shape of the mother's follicle cells that synthesize the eggshell.

In the family Pentatomidae (stink bugs), the chorion is characterized by the surface structure, termed either "spinose" or "coarse" [2]. "Spinose" refers to insect eggs that have projections arranged in patterns that extend outwardly from the surface. The term "coarse" refers to indented pit structures on the outer eggshell surface [2].

The chorion is produced within the female's ovariole by the follicular epithelium. In the simplest form, the chorion is typically comprised of three layers (exochorion, endochorion



Figure 1. Automontage photographs of three butterfly eggs. (A) An egg from the gulf fritillary, *Agraulis vanillae*; family: Nymphalidae. (B) Eggs from the luna moth, *Actias luna*; family: Saturniidae. Note the exuvia from the recent molt of the larvae. (C) Eggs from the zebra longwing, *Heliconius charithonia*; family: Nymphalidae.

[inner and outer], and vitelline membrane) [2, 3]. The vitelline membrane is the innermost layer that surrounds the embryo. A few insect studies have further subdivided the eggshell layers into waxy layers and crystal chorionic layers, which most probably serve as the main barriers in the eggshell against water loss. However, these structures and layers differ between insect families and species, depending on their habitat and individual respiratory and water requirements.

Gravid female insects have accessory glands that secrete glue-like substances, sometimes referred to as “cement” to adhere their oviposited eggs to a substrate. These glue substances have to be able to withstand environmental stress for the duration of embryonic development before the larvae is ready to emerge from the eggshell. Much like the eggshell, these glue substances are primarily comprised of proteins [4]. When considering the penetration of insecticides, the glue sheath adds an extra layer of protection for the developing embryo. The head louse glue sheath is especially problematic from the perspective of lice control.

Anyone who has experienced head lice is well aware of the glue sheath that surrounds a head louse egg, called a nit. The glue sheath laid down by the mother from her collateral glands adheres the nit to the hair shaft. This makes nits incredibly difficult to remove from hair. The nit comb, which is a widely used management technique for head lice, has very fine teeth designed to brush nits from the hair shaft. The sheath covers the entire egg, except at the operculum where respiration occurs [5]. Understanding the components of the sheath could

allow researchers to develop novel ways to denature the protein components of the sheath or to coat the sheath and prevent the embryo from breathing [5].

The embryo inside of the eggshell hatches, or encloses, through the egg cap, called the operculum. The operculum is usually located on the anterior pole of the egg. The border of the operculum is comprised of multiple, small, uniform-shaped holes along the circumference of an egg, usually aligned side by side. The appearance of these holes is similar to a loose-leaf notebook with a perforated edge on each page that allows pages to easily be torn out. The perforations on the egg make the operculum easier to break open, thus allowing the first instar larvae to push through the operculum during eclosion. This process is taxing to the small larvae, so many larvae have a specialized spine, or egg burster, on their heads to assist with hatching from the operculum [1]. In addition to an egg burster, the larvae will grow in size by engulfing air and amniotic fluid, creating pressure inside of the egg until they expand enough to break free from the eggshell.

In addition to the eggshell layers, there are structures present on the eggshell for respiration (aeropyles) and fertilization (micropyles) and also inner eggshell structures for the movement of oxygen (pillars, sometimes also referred to as struts or columns). The pillar, or column structures, can be observed easily in the scanning electron micrograph of a hatched bed bug egg (Figure 2). These structures that open into the eggshell are potentially sites that would allow insecticides to enter the insect egg. The insect eggshell must maximize embryo respiration while preventing water loss. The eggshell is designed not only to limit water loss from the egg but also to limit excessive water from entering the eggshell. Consequently, water-based insecticidal preparations do not easily penetrate insect eggshells.

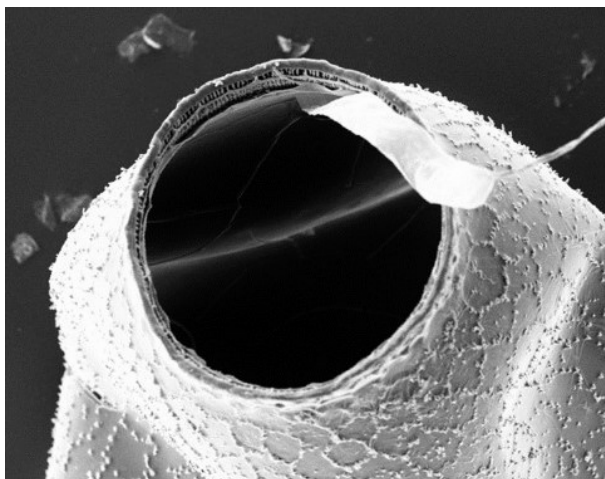


Figure 2. Scanning electron micrograph of a hatched bed bug egg. The egg has been cut in half, and the operculum has been removed for visualization purposes. The inner layers, including the respiratory struts and columns, can be observed.

Insect eggs are very small; therefore, they have an increased surface area-to-volume ratio [6]. Having a large proportion of the insect egg's surface exposed to the environment in relation to the internal volume makes the egg even more prone to desiccation. Multiple studies have been conducted to quantify the balance between respiration and water conservation of insect eggs [6–8]

3. Respiration and water conservation of insect eggs

The respiratory systems in insect eggs are very different from systems in other insect life stages. The principle idea is still the same; there must be openings on the eggshell to allow the exchange of gases with the atmosphere and mechanisms to move gases throughout the eggshell. Typically, insect embryos respire through tiny pores called aeropyles located on the outer eggshell, which allows for efficient gas exchange and reduces water loss during respiratory activities (Figure 3). To reduce water loss, many terrestrial insect eggs have a reduced numbers of aeropyles [6]. The aeropyles connect to an inner air-filled space that is referred to as the pillar system, which moves oxygen from the outer eggshell layers to the inner embryo. Insect eggs may have their aeropyles located on the outer rim of the operculum, as seen on many hemipteran eggs, or, if there is no defined rim, they will be located on the border of the operculum. Alternatively, aeropyles can also be scattered across the egg surface.

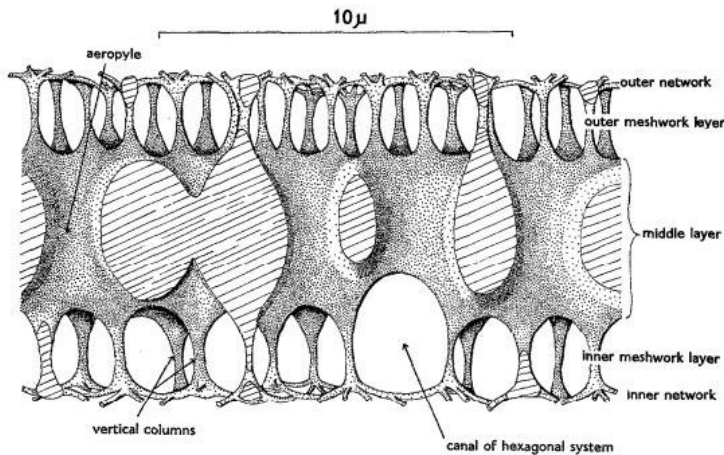


Figure 3. Respiratory system of the common house fly. Aeropyles can be seen extending through the eggshell and the various columnar sections and meshwork that compose the entire respiratory structure (Hinton 1967 [7]).

Considering there are an estimated 1 million insects on earth, there are numerous modifications and exceptions to the respiratory systems of insect eggs dependent upon the environment and respiratory requirements of individual species. While terrestrial insects have an inner pillar system, aquatic insect eggs primarily respire with a plastron. The plastron is a gas filled

air layer below the outer chorion of the egg shell. The plastron acts as a physical gill that allows eggs to respire under water [6]

Some terrestrial insect eggs that are oviposited in environments flooded with water may also have a plastron. In eggs with a large plastron membrane, only small parts of the plastron are permeable, limiting water loss. Terrestrial eggs typically do not have a plastron but do have a gas layer directly under the outer surface of the chorion and connected to the aeropyles. Aeropyles of terrestrial insects occasionally flooded by rainwater are located on the ends of respiratory horns. These horns extend above the water surface to allow for more efficient uptake of oxygen when the egg is surrounded by a layer of water [6]. These respiratory horns are essential to allow the insect to breathe during periods of heavy rain when an insect egg lacks the plastron respiratory system.

The eggshell layers and elaborate respiratory systems of insect eggs are modified to conserve water for the insect egg. Terrestrial insect eggs are provided from their mother all of the water necessary for survival and development at the time of oviposition. Therefore, the embryos must conserve this limited amount of water during their development while also respiring. Water loss occurs across the chorion and is correlated to oxygen consumption requirements of the embryo. The more gas exchange that occurs, the more vulnerable the insect embryo is to water loss.

The waxy layer in the insect eggshell most probably is the main barrier in the eggshell that prevents water loss. There was a significant increase in water loss in *Manduca sexta* eggs when the waxy layer was dissolved by organic solvents [8]. In addition to the waxy layer, the serosal membrane that envelopes the embryo has been shown to also protect insect eggs against desiccation [9]. RNAi technology has been used to prevent the development of the serosa in the beetle *Tribolium castaneum* [9]. Following RNAi treatment, the serosa-less eggs were subjected to a range of different humidities. Eggs exposed to the lowest humidities could not retain water and fewer eggs hatched compared to eggs that still had their serosal membrane intact.

Environmental factors and embryonic development can exacerbate water loss in insect eggs. Elevated environmental temperatures and low relative humidity can increase water loss greatly. Insect eggs have adapted to these conditions by having an altered density or number of eggshell chorionic layers. The developing embryo also has different metabolic requirements as it progresses in development. As insect embryos develop into larvae, the metabolic rates and water loss rates increase [8]. These metabolic requirements peak shortly before the embryo hatches from the eggshell.

4. Insect egg management

Eggs are undoubtedly the hardest insect life stage to kill with insecticides. Regardless of the application method, the tough eggshell that covers the egg prevents the entry of many insecticides, including water-based, oil-based, fumigants, and even some mechanical control methods.

An ovicide is a term used for an insecticide that specifically targets the egg stage. Smith and Salkeld [10] proposed three requirements for an ovicide to work: (1) the egg has to be in a location where it will be exposed so lethal concentrations of toxicant can reach it, (2) the egg has to be susceptible to the toxicant, and (3) a large enough proportion of eggs have to die from the toxicant in order for the treatment to be justified. The first requirement, exposure, is very important when considering insect eggs.

Agricultural pests, especially those that feed on plants, will commonly hide their eggs under leaves or may insert their eggs inside of the plant. Consideration of where the insect lays its egg in relation to an insecticide application is needed to guarantee an efficient treatment. The microclimate that the leaves create for the insect eggs is essential for their survival. Larger leaves, which absorb more sunlight, get much hotter than smaller leaves and can potentially kill eggs [11]. To limit sun exposure, insect eggs are often hidden underneath leaves and thus escape direct spray during an insecticidal treatment, resulting in an insect outbreak once those eggs hatch.

Systemic insecticides are often used for control of agricultural pests, but the use of these formulations presents a large problem for controlling the egg stage as far as exposure is concerned. These products are applied to the soil and then are taken up into the plant's xylem. Systemic products work well against immature and adult stages of sap-sucking pest insects, but these products do not work on eggs because the egg stage does not feed on the plant. Thus, the egg stage will survive and be a potential source for reinfestation.

If the eggs are not well hidden and the toxicant reaches the egg, the chemical still has to penetrate the eggshell and ultimately reach the embryo in order to be effective. Thus, the egg has to be susceptible to the toxicant. Variations in egg susceptibility could be due to the eggshell and differences in the chemical composition between eggs of different species [12] or could be due to enhanced resistance mechanisms of the embryo. Lastly, the toxicant must work well enough to kill a majority of the eggs in the population to be considered a viable option for control.

4.1. Insecticides for insect egg control

Normally, oil-based insecticides penetrate the insect eggshell more readily than water-based insecticides. The eggshell is comprised of a waxy component that allows the passage of oil-based products rather than water-based formulations. Early research has suggested that petroleum oils may also act by covering the aeropyles and causing egg mortality by limiting oxygen supply to the embryo [10]. Although most oils can be expected to penetrate the insect eggshell more easily, essential oils have been found to have difficulty penetrating eggs of the confused flour beetle and the Mediterranean flour moth compared to penetration through the cuticle of other life stages of the same insects, thus resulting in lower toxicity to eggs of these organisms [13]. Although oil-based products normally work better than water-based formulations in killing insect eggs, water has been shown to penetrate some insect eggshells. For instance, eggshell permeability to water has been demonstrated in the migratory locust, *Locusta migratoria migratorioides* [14]. Empty, hatched migratory locust eggs were filled with water then placed into an osmotic solution, and water was observed both entering and leaving the

eggshells. However, there is limited information regarding insect eggshells and their permeability to various chemicals. Consequently, the permeability of the insect eggshell to water, oil, or other chemical constituents of insecticides is not well established in the scientific literature.

Determining the mode of action of ovicidal insecticides is difficult. Formamidine insecticides, which have been evaluated against tobacco budworm (Lepidoptera: Noctuidae) eggs, were found to increase the levels of octopamine titers in eggs after treatment [15]. Formamidine insecticides have been shown to have a novel mode of action on insects by mimicking the actions of octopamine [16], which regulates insect behavior and energy metabolism. These results suggested that an increase in octopamine during embryogenesis could be playing a role in increased mortality of eggs treated with formamidine insecticides.

Paraoxon actively inhibits cholinesterase in *Pieris* eggs and was shown to prevent 100% of eggs from hatching [17]. Similarly, when house cricket eggs were exposed to carbamate insecticides, there was also a decrease in cholinesterase activity in eggs following exposure, but the insecticides did not prevent eggs from hatching [18].

Triatoma infestans eggs may be capable of detoxifying the organophosphate parathion with acetylcholinesterase enzymes [19]. The eggs produced elevated levels of acetylcholinesterase after being treated with parathion. The embryos were fully developed within the egg following treatment with parathion but never hatched from the eggshell. Therefore, the authors suggested that the embryos developed their nervous system during a later developmental stage and the parathion did not have an effect until the nervous system was fully developed.

All of these studies provide examples of insecticides permeating the eggshell and reaching the embryo, resulting in a physiological response to the insecticide. Unfortunately, most studies have only evaluated whether or not a particular insecticide has ovicidal action, thus lacking information on mode of action and penetration of the ovicide.

4.2. Fumigation of insect eggs

Fumigation has been found to be highly effective against eggs of several stored product pests. Eggs of four different species of common stored product pests, the almond moth (*Cadra cautella*), the Indian mealmoth (*Plodia interpunctella*), the lesser grain borer (*Rhyzopertha dominica*), and the red flour beetle (*T. castaneum*), have been evaluated to determine time, temperature, and pressures that were required for mortality by fumigation [20]. As temperatures increased and pressure decreased, time to mortality was reached in a shorter amount of time. Pressures above 100 mmHg and temperatures below 22.5°C were not practical to reach mortality because exposure times had to be increased drastically for the fumigant to cause egg mortality.

As is the case with liquid insecticides, studies have also shown that the egg stage is the most problematic life stage to kill with fumigants [12]. The main cause for low mortality is the impermeability of the eggshell. When eggs of *Schistocerca gregaria* were treated with sulfuryl fluoride, the gas was retained primarily in the proteinaceous portion of the eggshell instead of penetrating into the embryo [12]. In addition, egg age influences the efficacy of sulfuryl

fluoride [20]. Eggs of the Mediterranean flour moth aged 1–2 days were the most tolerant to sulfuryl fluoride compared to younger and older eggs. Lower doses of sulfuryl fluoride were required to kill the Mediterranean flour moth as temperatures were increased from 15°C to 25°C.

Fumigation of stored product pests has been limited by EPA registration and regulations [20]. This concern has instigated investigations for alternative nonchemical control methods for treating stored-product insects. An alternative control method to fumigation for stored-product pests that has been evaluated is the use of a vacuum system in storage bins to limit oxygen availability. However, eggs were also the most difficult life stage to kill using this method. This is not too surprising because an insect egg is very small and thus requires minute amounts of oxygen compared to the immature and adult stages of insects [20]. Therefore, extremely low levels of oxygen are required to create a hypoxic environment that will kill eggs.

4.3. Transovarial transport of insecticides

The transovarial transport of insect growth regulators from mother to offspring has been shown to cause a considerable reduction in egg hatch and viability. Insect growth regulators affect the development and occasionally the reproduction of insects. There are two main types of insect growth regulators: juvenile hormone analogs and chitin synthesis inhibitors. Juvenile hormone analogs (JHAs) mimic the natural juvenile hormone present in insects and can cause multiple physiological and morphological problems. Chitin synthesis inhibitors (CSIs), as the name suggests, inhibit the proper formation of chitin between insect molts. CSIs can cause insects to have malformed, thinner cuticles and ruptured intestines. Many of the symptoms will lead to eventual death of the insect if they do not die at the time of molt.

The transovarial transport of insect growth regulators from adult female insects to eggs have been evaluated in several insect species. Different insect species have varying tolerances and responses to pyriproxifen. For example, none of the eggs of gravid whitefly, *Bemisia tabaci*, females treated with the juvenile hormone analog pyriproxifen hatched [21]. Alternatively, when pyriproxifen was applied to female adults of the common green lacewing, *Chrysoperla carnea*, it had little effect on preventing their eggs from hatching [22]. Transovarial transport has also been documented with diflubenzuron, a chitin synthesis inhibitor. Treatment of adult female *C. carnea* with diflubenzuron resulted in 100% mortality of eggs at the highest tested dose.

The penetration of insecticides into the female's ovaries and elsewhere in the female body has been evaluated by using [¹⁴C]-labeled isotopes [22]. Most of the pyriproxifen in the female adult *C. carnea* was excreted within a couple of days, so it was probably not present before oviposition [22]. The use of the radio-labeled isotopes showed that diflubenzuron, unlike pyriproxifen, was absorbed more slowly and was retained within the female's body, which explains the high levels of toxicity to eggs with this insecticide.

4.4. Fungi as biological control agents for eggs

Entomopathogenic fungi have been used on a variety of insect species as a biological control agent, but the egg stage has not been found to be highly susceptible to fungal pathogens in

several cases. Eggs of the greenhouse whitefly, *Trialeurodes vaporariorum*, were found to be nonsusceptible to the fungus *Aschersonia aleyrodinis* [23]. No fungal spores or discoloration of the eggs was observed, and there were no differences in mortality between eggs that were treated with the fungus compared to eggs that had not been treated. However, the fungal spores were persistent for several days, so when the first instar larvae emerged from the egg, they became infected with the fungus.

Five different fungi, (*Beauveria bassiana*, *Metarhizium anisopliae*, *Metarhizium flavoviride*, *Paecilomyces farinosus*, and *Paecilomyces fumosoroseus*) were tested against eggs of the curculionidae beetles, *Otiorhynchus sulcatus* and *Sitona lineatus* [24]. *S. lineatus* eggs were much more tolerant of the fungal pathogens compared to *O. sulcatus* eggs. *O. sulcatus* eggs were found to be susceptible to all fungi except for *B. bassiana*. Only one fungus, *M. flavoviride*, was found to be moderately effective against *S. lineatus* eggs, causing 32% egg mortality. No other fungal treatments resulted in egg mortality to *S. lineatus* eggs.

5. Insecticide resistance in insect eggs

Insect eggs, like other insect stages, vary in their susceptibility to insecticides. Susceptibility differences between eggs from different insect species may be due to variability in chorionic adaptations that facilitate the uptake of oxygen [10]. Fewer aeropyles or smaller aeropyles may reduce the amount of insecticide that can enter the egg. In addition to respiratory structures, insecticides may also enter the chorion through the micropyles that allow fertilization of the egg [25]. Furthermore, modifications of the chorionic structures of the insect eggshell may enhance or reduce the penetration of insecticides.

Another important consideration when determining susceptibility of eggs to insecticides is the age of the egg. A freshly laid egg that has not fully developed is usually more susceptible to insecticides compared to an egg that has aged several days. Insect egg susceptibility to insecticides also changes during embryonic development [9]. The eggshell itself may harden during development, and the embryo may produce enzymes that break down insecticides.

Few studies have focused on insecticide resistance in eggs. Insecticide resistance in eggs from different insect species has been demonstrated when resistance was already quantified in the adult or larval stages [26–30]. Eggs collected from insect strains with resistance in the adult stage have been shown to have similar resistance. These studies determined that insecticide resistance had developed in eggs but did not determine the type or mechanisms of resistance.

Bed bugs, *Cimex lectularius*, have been extensively studied as adults with regards to insecticide resistance. Molecular technology has revealed that adult bed bugs have three types of resistance to pyrethroid insecticides: enhanced enzyme detoxification, KDR resistance, and target-site insensitivity. Recent research has revealed that bed bug eggs are also highly resistant to pyrethroids (Figure 4). Eggs collected from a strain that is considered pyrethroid-susceptible (Harlan) were much easier to kill with the insecticide deltamethrin, compared to eggs collected from strains (Richmond and Epic Center) where pyrethroid resistance was previously determined in adults.

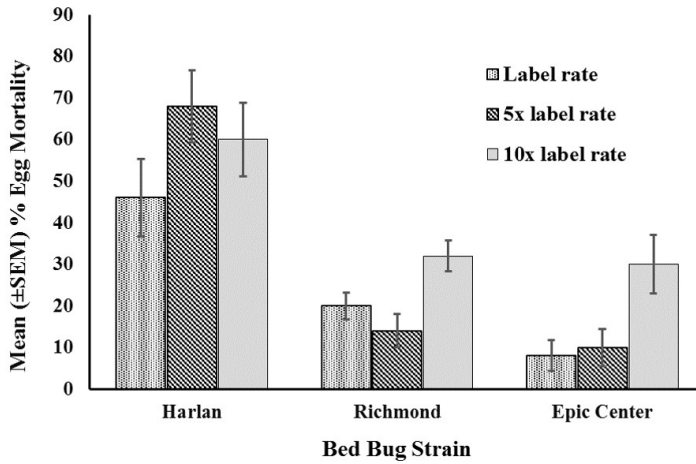


Figure 4. Mean percent mortality of eggs from a susceptible strain of bed bug eggs (Harlan) and two pyrethroid-resistant strains (Richmond and Epic Center) that were treated with the insecticide deltamethrin (0.05%). Bed bug eggs from the resistant strains had much lower mortality values compared to the susceptible strain eggs at each tested rate, showing that resistant eggs were much more difficult to kill. This study determined that resistance had already developed in the egg stage of bed bugs (Taken from Campbell et al. 2015 [30]).

Head lice, *Pediculus humanus capitis* (Phthiraptera: Pediculidae), have been shown to be highly resistant to pyrethroid insecticides [26]. Eggs, nymphs, and adults were evaluated from three different resistant head louse populations. Eggs were found to be highly resistant to permethrin in populations that had already demonstrated a high resistance to pyrethroid insecticides in adults and nymphs. This study suggests that there are similar resistance mechanisms within head louse eggs and adults from the same population.

Resistance patterns between eggs and first instars of Reduviid bugs, *T. infestans*, have been evaluated [31]. Insecticide resistance varied between eggs from different populations of *T. infestans* that were collected throughout Argentina and Bolivia. Eggs from a resistant strain that were aged several days were found to be as resistant to deltamethrin as the first instars (Toloza et al. 2008). Also, eggs from the resistant strain were found to be resistant to lambda-cyhalothrin but susceptible to fipronil and fenitrothion. First instar nymphs from a resistant strain had similar patterns of resistance as the eggs.

Eggs have also been found to be more tolerant than adult stages of the lesser grain borer, *R. dominica*, to the fumigant phosphine. Not only were eggs more tolerant to phosphine, eggs that were collected from adults that were previously screened for resistance were harder to kill with phosphine compared to eggs collected from strains that were previously determined to be susceptible to phosphine [27]. Screening for resistance in the adult stage against the fumigant phosphine was a reliable indicator for determining resistance in the egg stage of the lesser grain borer, with 9 of the 10 strains of adults that were phosphine resistant also exhibiting resistance in the egg stage. Eggs of *Liposcelis bostrychophila*, a stored product pest, were found

to have a delay in embryonic development when fumigated with phosphine. This delay in embryonic development seems to be a method of resistance, causing control failures with phosphine treatments because the eggs were able to survive treatment this creates a problem for grain storage facilities because they may later find a reinfestation of the pest after the eggs hatch.

There is a lot left to learn about insect eggs and their resistance to different control methods. The lack of knowledge on egg biology, physiology, and control compared to other life stages is unfortunate when you consider how important this life stage is in relation to potential management strategies. Most studies that have evaluated the efficacy of insect control methods have mostly neglected the egg stage and have focused on adult or immature stages.

When the egg stage is ignored during the implementation of treatments, those eggs are left to hatch and possibly cause a reinfestation. Therefore, more studies on the efficacy of control treatments against eggs are needed, especially in cases in which the eggs are reasonably accessible and treatable. Insect eggs should not be ignored in pest management programs just because they are small, or do not bite or feed. Rather, control efforts targeting insect eggs are advantageous because the pests would be eliminated before it has a chance to cause any damage.

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